Twin Research and Human Genetics
Volume 18 ▌ Number 5 ▌ pp. 533–544 ▌ © The Author(s) 2015 ▌ doi:10.1017/thg.2015.62

Genetic and Environmental Overlap Between Childhood Maltreatment and Adult Physical Health

Susan C. South,[1] Markus H. Schafer,[2] and Kenneth F. Ferraro[3]
[1]Department of Psychological Sciences, Purdue University, West Lafayette, IN, USA
[2]Department of Sociology, University of Toronto, Toronto, ON, Canada
[3]Department of Sociology and Center on Aging and the Life Course, Purdue University, West Lafayette, IN, USA

Past research demonstrates a phenotypic relationship between childhood maltreatment and adult health problems. Explanations of this association usually point to either: (a) a direct causal link, whereby exposure to early stress disrupts biological functioning during sensitive periods of development; or (b) an indirect effect operating through socioeconomic attainment, poor health behaviors, or some other pathway leading from childhood to adulthood. The current study examined whether the association between childhood maltreatment and adult health reflects genetic or environmental mediation. Using a large sample of adult American twins, we separately estimated univariate biometric models of child maltreatment and adult physical health, followed by a bivariate biometric model to estimate genetic and environmental correlations between the two variables. We found that a summary count of chronic health conditions shared non-trivial genetic overlap with childhood maltreatment. Our results have implications for understanding the relationship between maltreatment and health as one of active interplay rather than a simple cause and effect model that views maltreatment as an exogenous shock.

■ **Keywords:** childhood abuse, physical health, gene-environment correlation, twins

Previous research reveals that childhood is a sensitive period of development, and that major insults during this time, such as malnutrition, family violence, and chronic poverty, may have lasting effects on physical health, even into later life (Hayward & Gorman, 2004; O'Rand & Hamil-Luker, 2005). In particular, child maltreatment is associated with many adult health problems, including chronic illness, functional limitations, and low self-rated health (Greenfield, 2010; Greenfield & Marks, 2009a; Springer et al., 2003). Extant evidence suggests that various forms of abuse exert a direct, long-term influence on adult health (Felitti et al., 1998; Greenfield, 2010); additionally, the influence of abuse on many specific health conditions is mediated through pathways such as smoking (Springer, 2009), diet (Greenfield & Marks, 2009b), and lowered sense of personal control (Irving & Ferraro, 2006).

Though the descriptive associations between childhood adversities and adult health are compelling, the reasons for these relationships remain a matter of considerable investigation and debate (Bengtsson & Boström, 2009; Yi et al., 2007). For instance, some studies imply direct, long-term effects of early misfortune — sometimes conceptualized through; "biological embedding"; mechanisms (Shonkoff et al., 2009) — while other scholars emphasize the need to identify the many possible intermediary pathways connecting early experiences to adult health (e.g., O'Rand & Hamil-Luker, 2005; Springer, 2009; Springer et al., 2003).

One unexamined possibility is that childhood maltreatment is related to adult physical health because there are genes that directly influence both adult health and vulnerability to experiencing child maltreatment. That is, there may be evidence of gene-environment correlation (rGE) or an empirical association between 'genetic differences and differential environmental exposure' (Johnson, 2007, p. 424). Genetic factors can influence what environments people are exposed to, making those environments, in turn, heritable (Jaffee & Price, 2007). Genetic endowment for exposure to abuse could reflect a link between abuse perpetration and victimization, such that families pass down the proclivity towards aggressive behavior. If irascibility and violence have a genetic contribution, exposure to child abuse would be, in part, the outworking of an inauspicious intergenerational cycle (Barnes et al., 2013; DiLalla & Gottesman, 1991; Hines & Saudino, 2002).

RECEIVED 25 November 2014; ACCEPTED 27 July 2015.

ADDRESS FOR CORRESPONDENCE: Susan C. South, 703 Third Street, West Lafayette, IN 47907, USA. E-mail: ssouth@purdue.edu

There are three types of rGE (Jaffee & Price, 2007; Plomin et al., 1977) that have different implications for maltreatment and health. First, genes can be correlated with people's environments *passively*, when the same heritable vulnerability influences both a parent's tendency to parent in a certain way and children's likelihood to behave in a certain way (Schulz-Heik et al., 2010); for instance, parents' genetically influenced traits (e.g., parental impulsivity) both (1) create environments that the child experiences (e.g., abuse), and (2) are then genetically passed on to the child (e.g., child impulsivity, which may impact adult health). *Active* rGE occurs when people seek or create environments that correspond with their genetic propensities. If heritable factors (e.g., social-cognitive skills) give a developing child more or less foresight in de-escalating or escaping experiences of abuse and better or worse ability to achieve healthy behaviors, this would indicate an *active form* of genetic influence. Finally, rGE might be *evocative*, when a child's genetic profile evokes a response from their environment. A child may elicit harsh treatment from abuse-prone others as a result of a stress-inducing illness, disease, or disability. Active and evocative forms of rGE are both non-passive forms of rGE, reflecting a correlation between genetic characteristics of a person and their environmental exposures, and are indistinguishable in a standard twin study.

It is important to note that none of these possibilities imply that a child impels (nor deserves) maltreatment — such a claim would justifiably raise ethical concerns and be tantamount to 'blaming the victim'. Recognizing that genetic influences may be correlated with a phenotype of abusive experience, however, makes no normative claims and stands as an empirical question. Taking this perspective, although maltreatment is conventionally thought of as a wholly exogenous shock, it is in essence an interactional process (Belsky, 1980; Kadushin et al., 1981; Lloyd, 2013), albeit one of power and domination. In a recent review, Lloyd (2013, p. 473) suggests that it is better to conceptualize abuse as 'cycles of aversive interaction' that are 'embedded in family patterns of coercion and control' rather than as a set of limited or singular experiences or as a label affixed to a given parent. These conclusions are informed by research observing actual interactional dynamics; for instance, some parents respond aggressively to undesired child behavior, which often evokes defiance or hostility from the child, which subsequently produces an escalation in punishment (Borrego et al., 2004; Parke, 1974; Patterson, 1979). There is even burgeoning support from behavior genetics, as one study found evidence for a non-passive rGE between child maltreatment and child conduct problems, suggesting that maltreatment results from parents responding to genetically influenced conduct problems (Schulz-Heik et al., 2010).

rGEs cannot be tested directly using univariate twin modeling, but they can be inferred by estimating the genetic correlation (rA) between two phenotypes, or observed variables. Univariate twin modeling in a structural equation modeling framework uses genetically informative family data to decompose the variance in a construct, like adult physical health, into the amount of variance due to additive genetic influences (A), common or shared environmental influences (C), or unique, non-shared environmental influences (E). When examining two or more phenotypes in a bivariate model, it is also possible to estimate whether there is overlap between the genetic influences on one variable (e.g., child maltreatment) and the genetic influences on the other variable (e.g., health). This overlap is estimated as a rA that ranges from -1 to +1, with evidence of genetic overlap suggesting the presence of non-passive (i.e., active or evocative) rGE.

It is also possible to examine environmental mediation between maltreatment and health. In the same way that the magnitude of genetic overlap between two variables can be calculated, correlations can be computed to determine the amount of shared (rC) and non-shared environmental (E) influences common to two phenotypes. Evidence of overlapping shared environmental influences (rC) would indicate either passive rGE or shared environmental influences that contribute to both maltreatment and health. Non-shared environmental overlap (rE) would reflect systemic non-shared environmental influences that contribute to both variables, or common sources of error (e.g., Schulz-Heik et al., 2010).

Estimating genetic overlap requires some level of heritability for both child maltreatment and adult health conditions. Many aspects of 'physical health' have non-zero heritability (Komaroff, 1999). Genetic effects are most obvious in the case of single-gene disorders, such as sickle-cell anemia and cystic fibrosis. Beyond these straightforwardly chromosomal pathologies, health outcomes such as self-rated health, body mass index, and comorbid chronic conditions (Johnson & Krueger, 2005a, 2005b; Romeis et al., 2000; Stunkard et al., 1990) also demonstrate considerable estimated heritability — often between 30–70% in studies. Although many health conditions are commonly accepted as having some genetic basis, the heritability of childhood maltreatment may initially appear less apparent. However, studies have consistently documented that many putatively environmental or stressful events have significant heritability estimates (Kendler & Baker, 2007; Rowe, 1981). Even exposure to seemingly exogenous stressors, such as financial strain, perceived neighborhood safety, and discrimination demonstrate evidence for moderate heritability, above 0.20 (Schnittker, 2010). Researchers examined the genetic and environmental influences on both high risk (a summary index that included childhood abuse or neglect) and low risk (e.g., combat exposure or natural disaster) trauma and found that both were substantially heritable (60% for high risk vs. 47% for low risk; Sartor et al., 2012). If genes have some bearing on whether children's dispositions are whiny, agitating, or recalcitrant — the type of characteristics that shape the sequence of ongoing child–adult interactions that

https://doi.org/10.1017/thg.2015.62 Published online by Cambridge University Press

may culminate in abusive outbursts (Collins, 2008, p. 140) — then it is reasonable to expect some heritability of abusive experience.

To our knowledge, there has been only one study to date to examine the genetic and environmental influences on childhood maltreatment as retrospectively self-reported by adult participants. Using the twin sample from the National Longitudinal Study of Adolescent Health (ADD Health), the authors found that a composite maltreatment score was primarily attributable to unique environmental effects ($E = 0.66$), with some evidence of the shared environment ($C = 0.17$) and twin-specific environment ($T = 0.12$) but only modest evidence of genetic influences ($A = 0.06$; Schulz-Heik et al., 2009). There was evidence of greater genetic influences when examining physical maltreatment ($A = 0.28$) and neglect ($A = 0.24$) separately, but the rAs among the different forms of maltreatment were negligible (e.g., rA for physical maltreatment and neglect = 0.01). The authors concluded that their findings suggested small genetically mediated child effects on physical maltreatment and neglect, but that most of the effects on child maltreatment can be explained by idiosyncratic family context.

In the current study, our goal was to extend this previous work by examining the associations between childhood maltreatment and adult health using bivariate biometric modeling in a sample of adult twins. We began by first estimating univariate heritability and environmental influences separately for childhood maltreatment and adult health, operationalized here as a summary count of chronic health conditions (see Johnson & Krueger, 2005a, 2005b). Next, we examined possible mechanisms for the associations between childhood maltreatment and adult physical health. Parents endow their children with both traits and experiences that shape their life chances, and so our goal was to explore whether health problems and exposure to social adversity spring from a common genetic or environmental source. We used bivariate decomposition in order to estimate the overlapping genetic and environmental influences on maltreatment and health. A non-zero rA would indicate that genetic influences on the experience of maltreatment are shared with genetic influences on adult health (suggestive of a non-passive rA and genetic mediation), thereby challenging the assumption that maltreatment causes adult health problems. The presence of common shared environmental influences would indicate the presence of passive rGE or true shared environmental mediation. Overlapping non-shared environmental influences would indicate environmental mediation (or error), and would be the strongest evidence that maltreatment in childhood leads to adult health problems. By examining these questions, our goal was to illustrate how the major assumption embedded in studies of child maltreatment and health — that abusive experiences are a purely exogenous shock with lifelong consequences — may reach too far and ignore other possible etiological mechanisms.

Materials and Method

Sample

For the current study we utilized data from the MacArthur Foundation Survey of Midlife Development in the United States (MIDUS), a nationwide population study geared toward examining the interplay between physical health, psychological wellbeing, and the social environment. The MIDUS study sample consists of a nationally representative sample of individuals between the ages of 25–74 years in the non-institutionalized civilian population of the continental United States. A twin subsample of the main MIDUS sample was collected by screening members of approximately 50,000 households with a telephone survey to determine whether an immediate relative was a twin (Kessler et al., 2004). A total of 14.8% of the respondents who were contacted identified a twin relative; of those, 60% agreed to be contacted for study recruitment. Eligible twin pairs were invited to participate by recruiters from the Institute for Social Research at the University of Michigan. The final response rate for complete twin interviews (i.e., both members of the twin pair were contacted by an interviewer, agreed to participate, and completed a short zygosity screening questionnaire) was approximately 26%.[1]

A total of 1,914 individuals (957 twin pairs) participated in MIDUS data collection beyond the twin screener; we excluded individuals with missing or indeterminate zygosity information, as well as opposite-sex twin pairs, resulting in a sample of 1,386 individuals from 714 same-sex twin pairs. This sample included 672 complete twin pairs (164 monozygotic (MZ) male pairs, 186 MZ female pairs, 124 dizygotic (DZ) male pairs, and 198 DZ female pairs) as well as 42 singletons who were missing data on their co-twin.

Average age of the sample was 44.6 years ($SD = 12.16$, range 25–74), 57% of the sample was female, 7% of the sample was non-White, and 26% of the sample had at least a bachelor's degree. The twins in our sample are largely comparable to the excluded twins, though the latter were slightly less likely to be non-White (4% vs. 7%), slightly more likely to have a college degree (30% vs. 26%), and slightly older (mean = 45.56 vs. 44.63). Compared to the main (non-twin) random digit dialup MIDUS sample, the twin sample respondents used in our study were more likely to be White, slightly less educated, and slightly younger.

Measures

Zygosity. Twin zygosity was determined through the use of a self-report screener that assessed physical similarity (e.g., similarity of eye and hair color) and the degree to which others were confused about their identity during childhood. These types of questions generally are about 90% accurate at correctly classifying zygosity (Lykken et al., 1990) when validated against blood typing.[2]

Adult physical health. Twin respondents participated in the full MIDUS survey (Kendler et al., 2000), including

https://doi.org/10.1017/thg.2015.62 Published online by Cambridge University Press

two mailed self-administered questionnaire booklets and a computer-assisted telephone interview that lasted approximately 30 min. Health was assessed through a variety of measures, but our study focused on a summary count of chronic conditions. Previous research suggests that self-reports of chronic disease are highly valid for well-known conditions with straightforward diagnostic criteria, though measurement error is higher for diseases with non-established diagnostic criteria (Colditz et al., 1986; Haapanen et al., 1997). The score of chronic conditions was created by summing the number of the following 29 conditions the respondent had 'experienced' or 'been treated for' during the past year: asthma, bronchitis, or emphysema; other lung problems; arthritis, rheumatism, or other bone or joint disease; sciatica, lumbago, or recurring backache; persistent skin trouble; thyroid disease; hay fever; recurring stomach trouble, indigestion, or diarrhea; urinary or bladder problems; constipation; gall bladder trouble; persistent foot trouble; trouble with varicose veins; AIDS or HIV infection; autoimmune disorder; trouble with gums or mouth; trouble with teeth; high blood pressure; emotional disorder; alcohol or drug use problems; migraine headaches; chronic sleeping problems; diabetes or high blood sugar; neurological disorders; stroke; ulcer; hernia or rupture; and piles or hemorrhoids. Prior twin research with the MDUS data has also used this summary chronic condition measure (Johnson & Krueger, 2005a, 2005b). To handle the right-skew of the variable (skew = 1.86), a log10 transformation was applied before biometric modeling.

Childhood maltreatment. Maltreatment was operationalized with the Conflict Tactics Scale (CTS; Straus, 1979), a list of events occurring during childhood that encompasses both physical and emotional forms of abuse. Respondents were asked to report how often their mother, father, brother, sister, or anyone else victimized them in the following ways: (a) emotional abuse — insulted or swore; sulked or refused to talk; stomped out of the room; did or said something out of spite; threatened to hit; smashed or kicked something in anger; and (b) physical abuse — pushed, grabbed, or shoved; slapped; threw something; kicked, bit, or hit with a fist; hit or tried to hit; beat up; choked; burned or scalded. Reports concerning these types of specific events are regarded as more valid than general recollections about childhood experiences (Hardt & Rutter, 2004). Retrospective reporting, however, may lead to some underestimate of total maltreatment prevalence (Carlin et al., 1994). Consistent with previous research, we identified *frequent* experiences as a particularly salient indicator of childhood maltreatment, dichotomizing 'often' and 'sometimes' reports versus reports that the actions occurred 'rarely' or 'never' (see Irving & Ferraro, 2006, Schafer & Ferraro, 2013; Springer, 2009). Previous research indicates that though CTS measures have acceptable validity, their internal consistency reliability is modest because they measure relatively rare events (Straus et al., 1998). Thus, we use dichotomized indicators to enhance internal consistency (Straus et al., 1998). To capture a cumulative toll of abusive exposure during childhood, we created a count of how many of the five potential perpetrators frequently abused the respondent emotionally (from 0 to 5) and physically (from 0 to 5).[3]

Data Analysis

The current study used biometric modeling with twin data to examine genetic and environmental contributions of variance to childhood maltreatment and adult health. As with all statistical modeling with the twin design, our analyses rested on certain assumptions: the assumption that twins can be generalized to the general population; that there is no assortative mating between parents of twins, which would result in DZ twins having genetic similarity greater than 50%; and the equal environments assumption, that the shared environments of MZ twins and DZ twins contribute equally to their resemblance (see Blokland et al., 2013, for more details).

To obtain initial estimates of genetic and environmental influences on each phenotype, within twin pair correlations (i.e., the correlation between twin 1 and twin 2 on a health outcome variable) were estimated and compared for MZ (identical) and DZ (fraternal) twin pairs. Genetic influences are indicated when the MZ correlation is greater than the DZ correlation. If the MZ correlation is less than twice the DZ correlation, this suggests the presence of shared environmental influences. If the MZ correlation is less than 1.0, then we can assume that there are significant non-shared environmental influences on the phenotype (Plomin et al., 2008).

Although twin correlations provide an initial estimate of the genetic and environmental influences on a trait, they do not allow for formal statistical comparison of alternative models. Thus, biometric model fitting (Neale & Cardon, 1992) was used to estimate the genetic and environmental influences on maltreatment and chronic conditions, as well as the genetic and environmental covariances between the two variables. Classic univariate twin modeling compares the covariances between identical twins, who share 100% of their genes, and fraternal twins, who share, on average, 50% of their segregating genes (those on which humans tend to differ), to decompose the variance in a trait (phenotype) into the amount due to additive genetic effects (A), shared environmental influences (C), or those factors that twins growing up in the same household share in common that act to make them more similar, and non-shared environmental influences (E), or those experiences that are unique to each member of a twin pair growing up in the same household. This univariate ACE model assumes the correlation between the A latent factors is fixed to 1.0 for MZ twins and 0.5 for DZ twins and that the correlation between the C latent factors is 1.0 for both types of twins. A full ACE model was

https://doi.org/10.1017/thg.2015.62 Published online by Cambridge University Press

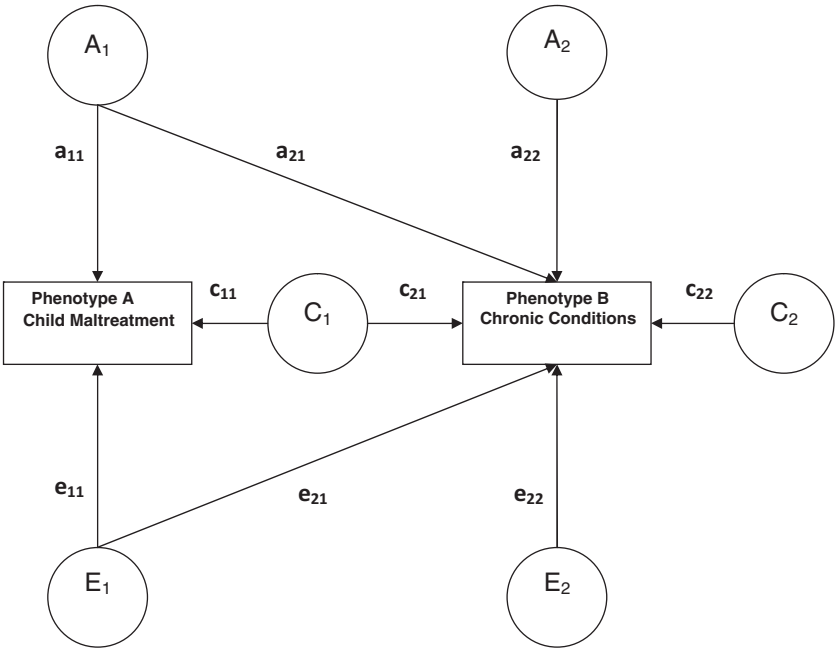

FIGURE 1

A bivariate (Cholesky) decomposition for two phenotypes, child maltreatment and chronic conditions, shown for only one member of the twin pair. Factors A_1 (additive genetic), C_1 (shared environmental), and E_1 (nonshared environmental) account for all of the variation in childhood maltreatment and any of the variation in chronic conditions that is in common with maltreatment. Factors A_2, C_2, and E_2 account for any residual additive genetic, shared environmental and non-shared environmental variation in the risk for chronic conditions.

compared to two reduced models: an AE model in which variance is attributed to additive genetics and non-shared environment, and a CE model in which no genetic variance is estimated.

This univariate ACE model can be extended to a bivariate (Cholesky) decomposition, which estimates the ACE variance components unique to each phenotype as well as the extent to which common genetic and environmental factors impact both phenotypes. As shown in Figure 1, variance in the dependent, downstream variable (i.e., chronic conditions) is partitioned into variance that overlaps with the independent variable (i.e., maltreatment) and variance that is unique to the dependent variable. The latent factors A_1, C_1, and E_1 contribute to the maltreatment and health variables, while A_2, C_2, and E_2 contribute to the health variable only. The unique factors (A_2, C_2, and E_2) represent any unique genetic or environmental effects on health that are independent of maltreatment, while the other paths represent genetic and environmental covariance between childhood maltreatment and health. Each path coefficient (e.g., a11, c11, e11) is a partial regression coefficient that indexes the strength of the latent factor on the phenotype. Standardized path coefficients can be squared and summed to obtain the total phenotypic variance; heritability (h^2) and commensurate estimates for shared and non-shared environment are proportions of total variance due to genetics, shared environment, and unique environment, respectively.

The bivariate model also provides estimates of the genetic and environmental correlations between the two variables. rA indexes the degree of overlap in the genetic influences on the upstream phenotype and the genetic influences on the downstream phenotype, and like any other correlation it ranges from -1 to +1. Similar types of correlations (i.e., overlap) are estimated for shared and non-shared environmental influences.

We first fit a full Cholesky decomposition with all possible parameters. The covariance parameters (a_{21}, c_{21}, and e_{21}) were dropped individually to examine the change in model fit. Biometric models were fit to the raw data in Mplus (Muthén & Muthén, 1998–2012) using full-information maximum-likelihood (ML). Following standard procedures to correct for potential biases in model fitting, the raw childhood maltreatment variable and transformed health outcome variable were regressed on age, age^2, age × gender, and age^2 × gender (McGue & Bouchard, 1984), and the residuals from the regressions were entered into the biometric models. Model fit was evaluated using chi-square difference tests for nested models, the Akaike Information Criterion (AIC; Akaike, 1987), and the root mean square error of approximation (RMSEA). The AIC statistic is an information theoretic fit statistic that enables one to select the model that best fits the data with the fewest number of parameters. Lower AIC values suggest better fitting models (Markon & Krueger, 2004). Hu and

https://doi.org/10.1017/thg.2015.62 Published online by Cambridge University Press

TABLE 1

Univariate ACE Models for Childhood Maltreatment and Chronic Conditions

	χ^2	*df*	$\Delta\chi^2$	Δdf	*p*	AIC	RMSEA	h^2	c^2	e^2
Childhood maltreatment										
ACE	7.05	6				3,826	0.022	0.28 (0.04, 0.52)	0.17 (-0.03, 0.37)	0.55 (0.47, 0.63)
AE	9.78	7	2.73	1	0.10	3,827	0.033	0.47 (0.40, 0.55)	–	0.53 (0.46, 0.60)
CE	12.20	7	5.15	1	0.02	3,829	0.046	–	0.38 (0.32, 0.44)	0.62 (0.56, 0.68)
Chronic conditions										
ACE	7.57	6				3,586	0.027	0.43 (0.35, 0.52)	0.00 (0.00, 0.00)	-0.57 (0.49, 0.65)
AE	7.57	7	0.00	1	1.00	3,584	0.015	0.43 (0.35, 0.52)	–	0.57 (0.49, 0.65)
CE	19.72	7	12.15	1	0.00	3,596	0.072	–	0.32 (0.25, 0.39)	0.68 (0.61, 0.75)

Note: $N = 1{,}344$. ACE refers to a model that includes additive genetics (A), common environment (C), and unique environment (E). AE includes additive genetic and non-shared environment only, and CE includes common and non-shared environment only. χ^2 = chi-square, *df* = degrees of freedom, AIC = Akaike's information criterion, RMSEA = root mean square error of approximation. h^2, c^2, and e^2 are the proportions of variance (and 95% CI) for genetic, common environmental, and non-shared environmental influences respectively.

TABLE 2

Goodness of Fit Results From Bivariate Twin Models for Child Maltreatment and Chronic Conditions

		Change in model fit					
Model		χ^2	*df*	$\Delta\chi^2$	Δ *df*	*p*	AIC
1	Full model	23.41	17				7,389
2	Drop a_{21}	24.90	18	1.49	1	.2225	7,388
3	**Drop c_{21}**	**23.42**	**18**	**0.01**	**1**	**.9058**	**7,387**
4	Drop e_{21}	25.81	18	2.40	1	.1217	7,389
5	Drop a_{21} and c_{21}	33.35	19	9.94	2	.0069	7,395

Note: $N = 1{,}344$. χ^2 = chi-square, *df* = degrees of freedom, AIC = Akaike's information criterion. Values of $p > .05$ indicate significantly better fit than the comparison model. Best fitting model is shown in bold type.

Bentler (1999) recommend that good fitting models have RMSEA $\leq$ 0.06.

Results

Descriptive Statistics

On average, participants reported just over two chronic conditions ($M = 2.19$, $SD = 2.36$). The cumulative exposure maltreatment score ranged from 0 to 8, with a mean of 2.07 ($SD = 2.09$). Maltreatment was significantly correlated with number of chronic conditions ($r = 0.15$, $p < .0001$). The MZ twin correlation for the age and gender regressed maltreatment variable was 0.44 and the DZ correlation was 0.32; this suggested, as has been found previously for putatively environmental variables, that childhood maltreatment is genetically influenced. The MZ twin correlation for the transformed and age- and gender-regressed chronic health conditions variable was 0.43, while the DZ correlation was 0.19, again suggesting the presence of genetic influences on a summary count of chronic conditions.

Univariate Biometric Modeling

We first fit univariate models to the twin data to examine the proportion of variance attributed to genetic and environmental factors for chronic health and child maltreatment (see Table 1). Results for the three components of variance are presented as proportions, thus the total variance will sum to 1.0 for each variable.

For child maltreatment, removing genetic influences led to a decrease in model fit according to all indices. When shared environment was removed, there was a non-significant increase in chi-square and small increases in AIC and RMSEA. Given that the full ACE model provided the lowest chi-square, AIC, and RMSEA values, it was retained as the best-fitting model. For chronic health, removing genetic influences led to a significant decrement in model fit according to all indices, but when the shared environmental parameter was removed, chi-square did not change and AIC And RMSEA were both lower. The parameter estimates from the full ACE model for chronic conditions demonstrated moderate genetic ($h^2 = 0.43$) and unique environmental ($e^2 = 0.57$) influence. Confirming what the twin correlations suggested, childhood maltreatment demonstrated genetic influences ($h^2 = 0.28$ in the full ACE model). Given the relatively small sample size and reduced power to differentiate between the A and C effects, a full ACE bivariate decomposition was estimated for childhood maltreatment and chronic health (see below).

Results of Bivariate ACE Model

Table 2 presents the results of model fit for the bivariate ACE Cholesky decomposition. We began with a full ACE bivariate model, and fit nested models in which the individual covariance parameters were dropped one at a time. It was possible to remove the A, C, and E covariance parameters

https://doi.org/10.1017/thg.2015.62 Published online by Cambridge University Press

TABLE 3

Proportions of Variance and Overlap from Bivariate Decomposition for Childhood Maltreatment and Chronic Conditions

	Total[a]			From maltreatment[b]			Unique to health[c]			Overlap		
	h^2	c^2	e^2	h^2	c^2	e^2	h^2	c^2	e^2	rA	rC	rE
Chronic conditions	0.43	0.00	0.57	0.10	–	0.01	0.90	–	0.99	0.32 (0.09, 0.56)	–	0.08 (-0.02, 0.18)

Note: $N = 1{,}344$. Estimates are from the best-fitting model in Table 2. 95% confidence intervals are provided for the genetic and environmental correlations. [a]The total proportions of variance in chronic conditions. [b]Amount of the total due to the shared overlap with child maltreatment. [c]Amount of the total that was unique to chronic conditions.

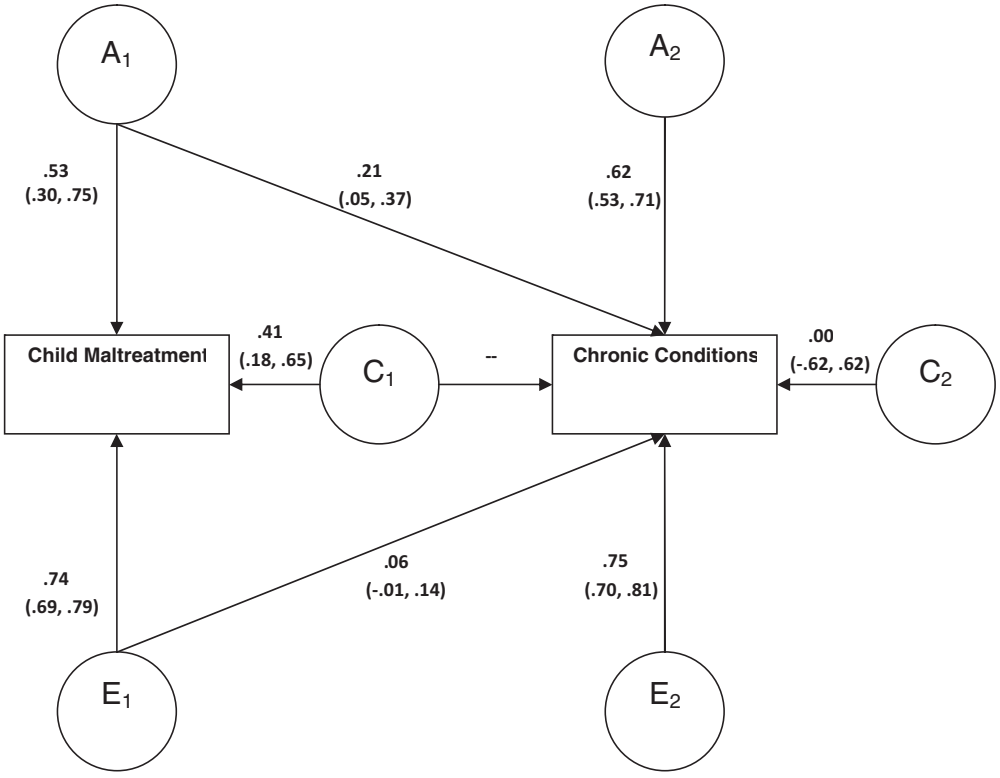

FIGURE 2

Parameter estimates from the best-fitting bivariate (Cholesky) decomposition for child maltreatment and chronic conditions, shown for only one member of the twin pair.

individually without a significant drop in model fit, although a model that removed A and C was significantly worse than the full model. The model in which the C covariance parameter was dropped was chosen as the best-fitting model based on lowest AIC value and non-significant change in chi-square. The parameter estimates and 95% confidence intervals for the best-fitting model are given in Figure 2.

Table 3 presents the proportion of genetic and environmental variance in chronic conditions that is due to common influences with maltreatment and unique influences. As shown, genetic influences accounted for 43% of the total variation in chronic conditions, roughly equal to the heritability estimate from the univariate modeling. The estimate of shared environment was 0% and the standardized estimate for unique environment was 57%. In the best-fitting bivariate model, 10% of the total heritability of chronic conditions was shared with childhood maltreatment. Also shown in Table 3 are three parameters, rA, rC, and rE, which represent the correlation of genetic influences, shared environmental influences, and unshared environmental influences, respectively, between maltreatment and chronic conditions. For any given correlation, values approaching 1.00 indicate a greater overlap between the underlying influences on childhood maltreatment and that of chronic conditions. There was a moderate rA between childhood maltreatment and chronic conditions (rA = 0.32) and a small unique environmental correlation (rE = 0.08). The shared environmental correlation was not computed, as the best-fitting model dropped the shared environmental covariance.

Discussion

Childhood maltreatment is associated with a host of adult health problems, and current research is focused on assessing the breadth of this problem (Greenfield, 2010) and

https://doi.org/10.1017/thg.2015.62 Published online by Cambridge University Press

sorting out causal pathways that link early life experience with adult conditions (Springer, 2009). In the current study, we examined an unexplored area of inquiry: What is the magnitude of the genetic or environmental mediation between child maltreatment and adult disease? Previous research has demonstrated that social stressors such as financial strain, discrimination, and romantic dissolution, as well as childhood maltreatment and high-risk trauma, have at least moderate degree of heritability (Boardman et al., 2011; Sartor et al., 2012; Schnittker, 2010; Schulz-Heik et al., 2010). The current study continues in this vein, examining the genetic and environmental influences on a measure of childhood maltreatment. We then extended the previous work by examining whether there were overlapping genetic and environmental influences between maltreatment and chronic health conditions in adulthood.

With regard to the genetic and environmental influences on health, the findings from the current analyses are in line with the previous work in this area. We found that the full univariate ACE model included non-zero heritability, suggesting genetic effects on a summary count of chronic health conditions. Prior investigators have reached similar conclusions concerning health outcomes (e.g., Poulsen et al., 1999, for diabetes; Snieder et al., 2003, for hypertension) and for chronic conditions, also using the MIDUS sample, see Johnson and Krueger (2005a, 2005b).

In the full univariate ACE model of child maltreatment, we estimated that nearly one third of the variation (28%) in exposure to child maltreatment was attributable to genetic influence. In a more parsimonious model that included parameters for genetics and non-shared environment only, the estimated heritability was even greater, 47%. Therefore, we feel confident in concluding *at least a quarter of the variation in childhood maltreatment owes to genetic influence.* This range is admittedly wide, but suggests that there is a non-ignorable genetic component of the phenotype. Our findings also compare favorably to the previous work in this area. Another study examining retrospective self-reports of maltreatment also reported a heritability estimate of 28% for physical maltreatment (from a homogeneity model collapsing across males and females), a one-item measure of being slapped, hit, or kicked (Schulz-Heik et al., 2009). Thus, there is great consistency in heritability of maltreatment across these two studies, despite our sample being older and the fact that our measure of maltreatment included both physical abuse and emotional abuse by multiple perpetrators, and not just parents or primary caregivers.

Next, we turned to examining genetic and environmental mediation of the relationship between maltreatment and chronic health conditions. From the best-fitting bivariate model, there was evidence of a small to moderate rA between child maltreatment and chronic conditions. This is suggestive of non-passive rGE, or true genetic mediation between maltreatment and health outcomes. Finding overlap between genetic influences on childhood maltreatment and adult health suggests that *the probability of acquiring many diseases has a genetic basis shared with the broad genetic propensity to experience high levels of physical and emotional abuse.* Our findings are in line with work on trauma more broadly, which has found that high-risk trauma (including childhood neglect and physical abuse) has very high rAs with adult mental health (i.e., major depression and post-traumatic stress disorder; Sartor et al., 2012). Of course, the accumulation of different physical health conditions (our chronic conditions variables, assessed broadly in the MIDUS, ranging from asthma to severe varicose veins) is shaped by a broad range of factors, including health behavior, exposure to risks, access to care, and so on, but some aspect of the genetic code also appears to influence individual-level scores on this disparate array of problems. MIDUS twins who were genetically predisposed to experiencing chronic conditions were also genetically influenced to experiencing maltreatment; one possibility is that another, genetically influenced trait or characteristic, such as lack of social skills or high levels of disinhibition/low levels of conscientiousness, place individuals at risk for both outcomes.

Our results, at least for this measure of health outcomes, suggest that the causal efficacy of child maltreatment — increasingly conceptualized as 'a life-course social determinant of health' (Greenfield, 2010) — is complicated somewhat because some portion of that correlation reflects genetic confounding. Causal inference in the maltreatment-health relationship often treats childhood misfortune as an exogenous shock, which affects stress responses (Morton et al., 2012) or increases the probability of deleterious health behaviors (Greenfield & Marks, 2009a; Springer, 2009; Springer et al., 2003). While there is certainly much truth to these explanations, our findings suggest that genetic explanations also deserve attention. If parents who pass along genes for health problems are the same parents who are more likely to abuse their children, then there is some built-in confoundedness in the link between child maltreatment and adult health unaccounted for by multiple pathway models (Springer, 2009) or current formulations of the stress process framework (Turner & Butler, 2003). Studies that ignore genetic possibilities risk overstating the causal nature of childhood experiences. More importantly, however, the recognition of complex gene-environment interplay makes several demands of future research: studies should give careful attention to gene-environment interactions (e.g., how childhood maltreatment moderates broad genetic propensities) not only for distal health outcomes, but also for the intervening variables that link childhood maltreatment to adult disease (e.g., alcohol or drug use, social network acquisition).

There are many limitations of this study that lend caution to our interpretation of findings. First, a core assumption of behavioral genetics modeling is that the environments of MZ and DZ twins do not differ. If MZ twins were treated

https://doi.org/10.1017/thg.2015.62 Published online by Cambridge University Press

more similarly by household members than DZ twins due to physical appearance or personality, this would violate the equal environments assumption and could result in an overestimation of genetic effects (see Horwitz et al., 2003). Violation of the equal environments might, however, result in the underestimation of genetic effects. As children, MZ twins might be more likely than their DZ counterparts to distinguish themselves behaviorally and to seek out distinct environmental experiences (see Kessler et al., 2004, pp. 135–136). This scenario could occur if one twin took on the role of instigator or troublemaker, while another was more conflict-avoidant in the family microsystem. There is some evidence for this type of behavioral differentiation within families (Kessler et al., 2004). Second, it is not possible to tease apart the type of non-passive, rGE (i.e., evocative vs. active) at play in the link between maltreatment and adult health using a twin study (Neiderhiser et al., 2004); future research might wish to utilize different methods to identify, for instance, the role of evocative versus passive rGE (Marceau et al., 2013). Third, it is possible that there were gene × environment interaction effects that we did not account for, which could bias the genetic effects; future research should utilize biometric moderation models to examine whether genetic and environmental influences on health differ as a function of level of child maltreatment (e.g., Purcell, 2002).

Finally, this study relied upon retrospective questions from childhood, and so we are not able to assert that MIDUS respondents reported the events as they actually happened. There is always the possibility of bias when relying upon retrospective designs, but the MIDUS investigators were careful to avoid using loaded words such as 'abuse' or 'adversity' in the survey. Rather, the questionnaire used questions from the well-validated Conflict Tactics Scale (Straus, 1979). This approach was intended to minimize biases in recollection and reporting. Another potential issue is that people with particular personality characteristics, such as neuroticism or pessimism, or a history of depression, may be more inclined to recall unfavorable childhood experiences as well as over-report health problems. To the extent that personality traits are heritable, the apparent genetic commonality between childhood victimization and adult health could boil down to this factor alone. Supplementary analyses adjusted for neuroticism as a personality trait, but the major conclusions of the study were not altered.[4]

In summary, findings from the current study suggest that there is a role for genes in both avoiding disease in adulthood and having previously avoided abuse during childhood. Avoiding disease is a central aspect of successful aging (Rowe & Kahn, 1997). Those subjected to early trauma may be less likely to achieve this desirable adult status because of direct long-term health effects or through myriad intervening causal pathways; but, as this study indicates, such victims also must often contend with less advantageous genetic endowments.

Acknowledgments

Support for this research was provided by a grant from the National Institute on Aging (R01 AG033541). Data were made available by the Inter-university Consortium for Political and Social Research, Ann Arbor, MI. Neither the collector of the original data nor the Consortium bears any responsibility for the analyses or interpretations presented here.

Endnotes

1 The MIDUS, as a national population-based sample drawn from random-digit dialing, has a somewhat lower response rate compared to twin registries such as the Australian Twin Register (69%, Nelson et al., 2002), the UK Adult Twin Registry (49%, Burri et al., 2011), or the University of Washington Twin Registry (estimated at 21–38%, Afari et al., 2006).

2 When twins were initially recruited into the MIDUS study, they were asked a series of questions to determine whether they were MZ/DZ. Scores were given based on the answers, and scores were used to classify twins as MZ or DZ. This was not confirmed with DNA in MIDUS; however, eight self-report questions that were assessed in MIDUS were also used in the Virginia Twin Registry, which did confirm zygosity with DNA. Logistic regression was conducted to predict zygosity based on molecular genetic classification of being MZ/DZ. 'Strong classification' (i.e., predicting the likelihood of being MZ was <10 or >90%) was obtained for 86% of the pairs. There were pairs that were unable to be classified using the questionnaire method, and they were excluded from the current study (as they have been from numerous other studies using the MIDUS twin data set).

3 In supplementary analyses we used alternative coding schemes for the abuse measures. We estimated models with chronic conditions as the outcome variable, and the following maltreatment variables: (1) a score that only included reports about perpetrating mothers and fathers, which was created by summing dichotomized indicators (*sometimes/often* vs. *rarely/never*) for physical abuse and emotional abuse (range: 0 = no abuse by either parent, to 4 = both forms of abuse by both parents); (2) separate dummy variables (yes/no) for maternal abuse and paternal abuse; and (3) a summary score that was the average (1–4 scale, 1 = *never*, 4 = *often*) across the five possible perpetrators for each of 15 items that included emotional, physical, and severe physical abuse. Substantive conclusions were generally unchanged from the main results presented in the text. In all cases, individual ACE covariance parameters could be removed from the full model without a significant decrement in fit, although removing A and C covariance parameters at the same time resulted in significantly worse fit for two of the four models ($p = .009$ for summed score across perpetrating mothers and fathers, $p = .002$ for average across all perpetrators) and close to significantly worse for the other two models (dad abuse only, $p = .05$ and mom abuse only, $p = .06$). For all possible coding schemes, results from the full model including all covariance parameters indicated a non-zero rA (range = 0.25–0.70) with small non-shared environmental correlations (range rE = 0.03–0.15). Full results are available from the first author.

https://doi.org/10.1017/thg.2015.62 Published online by Cambridge University Press

4 We re-estimated our bivariate analyses with maltreatment and the chronic conditions health variable, but regressing out the personality trait of neuroticism from the chronic conditions variable before it was entered into the biometric model. Fit statistics indicated that any of the ACE covariance parameters could individually be removed from the full model (chi-square = 21.02, $df = 17$, AIC = 7,386) without a significant decrease in fit (e.g., when dropping A covariance, chi-square = 21.43, $df = 18$, Δchi-square = 0.41, $\Delta df = 1$, $p = .52$, AIC = 7,384); but, parameter estimates controlling for neuroticism (for chronic conditions, $A = 0.41$, $C = 0$, $E = 0.59$, rA = 0.18, rC = 0.99, rE = 0.07) were similar to parameter estimates not controlling for neuroticism ($A = 0.43$, $C = 0$, $E = 0.57$, rA = 0.30, rC = 1.00, rE = 0.08).

References

Afari, N., Noonan, C., Goldberg, J., Edwards, K., Gadepalli, K., Osterman, B., Evanoff, C., & Buchwald, D. (2006). University of Washington Twin Registry: Construction and characteristics of a community-based twin registry. *Twin Research and Human Genetics: The Official Journal of the International Society for Twin Studies*, *9*, 1023–1029.

Akaike, H. (1987). Factor analysis and AIC. *Psychometrika*, *52*, 317–332.

Barnes, J. C., TenEyck, M., Boutwell, B. B., & Beaver, K. M. (2013). Indicators of domestic/intimate partner violence are structured by genetic and nonshared environmental influences. *Journal of Psychiatric Research*, *47*, 371–376.

Belsky, J. (1980). Child maltreatment: An ecological integration. *American Psychologist*, *35*, 320–335.

Bengtsson, T., & Boström, G. (2009). Do conditions in early life affect old-age mortality directly and indirectly? Evidence from 19th-century rural Sweden. *Social Science and Medicine*, *68*, 1583–1589.

Blokland, G. A., Mosing, M. A., Verweij, K. H., & Medland, S. E. (2013). Twin studies and behavior genetics. In T. D. Little (Ed.), *The Oxford handbook of quantitative methods in psychology. Vol. 2: Statistical analysis* (pp. 198–218). New York: Oxford University Press.

Boardman, J. D., Alexander, K. B., & Stallings, M. C. (2011). Stressful life events and depression among adolescent twin pairs. *Biodemography and Social Biology*, *57*, 53–66.

Borrego, J. J., Timmer, S. G., Urquiza, A. J., & Follette, W. C. (2004). Physically abusive mothers' responses following episodes of child noncompliance and compliance. *Journal of Consulting and Clinical Psychology*, *72*, 897–903.

Burri, A., Cherkas, L., Spector, T., & Rahman, Q. (2011). Genetic and environmental influences on female sexual orientation, childhood gender typicality and adult gender identity. *PLoS ONE*, *6*, e21982.

Carlin, A. S., Kemper, K., Ward, N. G., Sowell, H., Gustafson, B., & Stevens, N. (1994). The effect of differences in objective and subjecive definitions of childhood physical abuse on estimates of its incidence and relationship to psychopathology. *Child Abuse & Neglect*, *18*, 393–399.

Colditz, G. A., Martin, P., Stampfer, M. J., Willett, W. C., Sampson, L., Rosner, B., ... Speizer, F. E. (1986). Validation of questionnaire information on risk factors and disease outcomes in a prospective cohort study of women. *American Journal of Epidemiology*, *123*, 894–900.

Collins, R. (2008). *Violence: A micro-sociological theory*. Princeton, NJ: Princeton University Press.

DiLalla, L. F., & Gottesman, I. I. (1991). Biological and genetic contributors to violence — Widom's untold tale. *Psychological Bulletin*, *109*, 125–129.

Felitti, V. J., Anda, R. F., Nordenberg, D., Williamson, D. F., Spitz, A. M., Edwards, V., ... Marks, J. S. (1998). Relationship of childhood abuse and household dysfunction to many of the leading causes of death in adults: The Adverse Childhood Experiences (ACE) Study. *American Journal of Preventive Medicine*, *14*, 245–258.

Greenfield, E. A. (2010). Child abuse as a life-course social determinant of adult health. *Maturitas*, *66*, 51–55.

Greenfield, E. A, & Marks, N. F. (2009a). Profiles of physical and psychological violence in childhood as a risk factor for poorer adult health: Evidence from the 1995–2005 National Survey of Midlife in the United States. *Journal of Aging and Health*, *21*, 943–966.

Greenfield, E. A, & Marks, N. F. (2009b). Violence from parents in childhood and obesity in adulthood: Using food in response to stress as a mediator of risk. *Social Science and Medicine*, *68*, 791–798.

Haapanen, N., Miilunpalo, S., Pasanen, M., Oja, P., & Vuori, I. (1997). Agreement between questionnaire data and medical records of chronic diseases in middle-aged and elderly Finnish men and women. *American Journal of Epidemiology*, *145*, 762–769.

Hardt, J., & Rutter, M. (2004). Validity of adult retrospective reports of adverse childhood experiences: Review of the evidence. *The Journal of Child Psychology and Psychiatry*, *45*, 260–273.

Hayward, M. D. & Gorman, B. K. (2004). The long arm of childhood: The influence of early-life social conditions on men's mortality. *Demography*, *41*, 87–107.

Hines, D. A., & Saudino, K. J. (2002). Intergenerational transmission of intimate partner violence: A behavioral genetic perspective. *Trauma, Violence, and Abuse*, *3*, 210–225.

Horwitz, A. V., Videon, T. M., Schmitz, M. F., & Davis, D. (2003). Rethinking twins and environments: Possible social sources for assumed genetic influences in twin research. *Journal of Health and Social Behavior*, *44*, 111–129.

Hu, L., & Bentler, P. M. (1999). Cutoff criteria for fit indexes in covariance structure analysis: Conventional criteria versus new alternatives. *Structural Equation Modeling*, *6*, 1–55.

Irving, S. M., & Ferraro, K. F. (2006). Reports of abusive experiences during childhood and adult health ratings. *Journal of Aging and Health*, *18*, 458–485.

Jaffee, S. R., & Price, T. S. (2007). Gene-environment correlations: A review of the evidence and implications for prevention of mental illness. *Molecular Psychiatry*, *12*, 432–442.

Johnson, W. (2007). Genetic and environmental influences on behavior: Capturing all the interplay. *Psychological Review*, *114*, 423–440.

https://doi.org/10.1017/thg.2015.62 Published online by Cambridge University Press

Johnson, W., & Krueger, R. F. (2005a). Higher perceived life control decreases genetic variance in physical health: Evidence from a national twin study. *Journal of Personality and Social Psychology*, *88*, 165–173.

Johnson, W., & Krueger, R. F. (2005b). Predictors of physical health: Toward an integrated model of genetic and environmental antecedents. *Journal of Gerontology: Social Sciences*, *60B*, 42–52.

Kadushin, A., Martin, J. A., & McGloin, J. (1981). *Child abuse: An interactional event*. New York: Columbia University Press.

Kendler, K., & Baker, J. H. (2007). Genetic influences on measures of the environment: A systematic review. *Psychological Medicine*, *37*, 615–626.

Kendler, K. S., Thornton, L. M., Gilman, S. E., & Kessler, R. C. (2000). Sexual orientation in a national sample of twin and sibling pairs. *American Journal of Psychiatry*, *157*, 1843–1846.

Kessler, R. C., Gilman, S. E., Thornton, L. M., Kendler, K. S. (2004). Health, wellbeing, and social responsibility in the MIDUS twin and sibling subsamples. In O. G. Brim, C. D. Ryff, & R. C. Kessler (Eds.), *How healthy are we? A national study of wellbeing at midlife* (pp. 124–152). Chicago: University of Chicago Press.

Komaroff, A. L. (Ed.). (1999). *Harvard medical school family medical guide*. New York: Simon & Schuster.

Lloyd, S. A. (2013). Family violence. In G. W. Peterson & K. R. Bush. (Eds.), *Handbook of marriage and the family* (pp. 449–485). New York: Springer.

Lykken, D. T., Bouchard, T. J., McGue, M., & Tellegen, A. (1990). The Minnesota Twin Family Registry: Some initial findings. *Acta Genetica Medica Gemellologiae*, *39*, 35–70.

Marceau, K., Horwitz, B. N., Narusyte, J., Ganiban, J. M., Spotts, E. L., Reiss, D., … Neiderhiser, J. M. (2013). Gene–environment correlation underlying the association between parental negativity and adolescent externalizing problems. *Child Development*, *84*, 2031–2046.

Markon, K., & Krueger, R. F. (2004). An empirical comparison of information-theoretic selection criteria for multivariate behavior genetic models. *Behavior Genetics*, *34*, 593–610.

McGue, M., & Bouchard, T. J. (1984). Quality of twin data for the effects of age and sex. *Behavior Genetics*, *14*, 325–343.

Morton, P. M., Schafer, M. H., & Ferraro, K. F. (2012). Does childhood misfortune increase cancer risk in adulthood. *Journal of Aging and Health*, *24*, 948–984.

Muthén, L. K., & Muthén, B. O. (1998–2012). *Mplus user's guide* (7th ed.). Los Angeles, CA: Muthén & Muthén.

Neale, M. C., & Cardon, L. R. (1992). *Methodology for genetic studies of twins and families*. Dordrecht, The Netherlands: Kluwer.

Neiderhiser, J. M., Reiss, D., Pedersen, N. L., Lichtenstein, P., Spotts, E. L., Hansson, K., … Elthammer, O. (2004). Genetic and environmental influences on mothering of adolescents: A comparison of two samples. *Developmental Psychology*, *40*, 335–351.

Nelson, E. C., Heath, A. C., Madden, P. A., Cooper, M. L., Dinwiddie, S. H., Bucholz, K. K., … Martin, N. G. (2002). Association between self-reported childhood sexual abuse and adverse psychosocial outcomes: Results from a twin study. *Archives of General Psychiatry*, *59*, 139–145.

O'Rand, A. M., & Hamil-Luker, J. (2005). Processes of cumulative adversity: Childhood disadvantage and increased risk of heart attack across the life course. *Journal of Gerontology: Social Sciences*, *60B*, 117–124.

Parke, R. D. (1974). Rules, roles and resistance to deviation in children: Explorations in punishment, discipline and self-control. In A. Pick (Ed.), *Minnesota symposium on child psychology. Vol. 8*: (pp. 111–143). Minneapolis: University of Minneapolis Press.

Patterson, G. (1979). A performance theory of coercive family interaction. In R. B. Cairns (Ed.), *The analysis of social interactions: Methods, issues and illustrations* (pp. 119–163). Hillsdale, NJ: Erlbaum.

Plomin, R., DeFries, J. C., & Loehlin, J. C. (1977). Genotype-environment interaction and correlation in the analysis of human behavior. *Psychological Bulletin*, *84*, 309–322.

Plomin, R., DeFries, J. C., McClearn, G. E., & McGuffin, P. (2008). *Behavioral genetics* (5th ed.). New York: Worth Publishers.

Poulsen, P., Kyvik, K. O., Vaag, A., & Beck-Nielsen, H. (1999). Heritability of Type II (non-insulin-dependent) mellitus and abnormal glucose tolerance — A population based twin study. *Diabetologia*, *42*, 139–145.

Purcell, S. (2002). Variance components models for gene-environment interaction in twin analysis. *Twin Research*, *5*, 554–571.

Romeis, J. C., Scherrer, J. F., Xian, H., Eisen, S. A., Bucholz, K., Heath, A. C., … True, W. R. (2000). Heritability of self-rated health. *Health Services Research*, *35*, 995–1010.

Rowe, D. C. (1981). Environmental and genetic influences on dimensions of perceived parenting: A twin study. *Developmental Psychology*, *17*, 203–208.

Rowe, J. W., & Kahn, R. L. (1997). Successful aging. *The Gerontologist*, *37*, 433–440.

Sartor, C. E., Grant, J. D., Lynskey, M. T., McCutcheon, V. V., Waldron, M., Statham, D. J., … Nelson, E. C. (2012). Common heritable contributions to low-risk trauma, high-risk trauma, posttraumatic stress disorder, and major depression. *Archives of General Psychiatry*, *69*, 293–299.

Schafer, M. H., & Ferraro, K. F. (2013). Childhood misfortune and adult health: Enduring and cascadic effects on somatic and psychological symptoms. *Journal of Aging and Health*, *25*, 3–28.

Schnittker, J. (2010). Gene-environment correlations in the stress-depression relationship. *Journal of Health and Social Behavior*, *51*, 229–243.

Schulz-Heik, R. J., Rhee, S. H., Silvern, L., Haberstick, B. C., Hopfer, C., Lessem, J. M., … Hewitt, J. K. (2010). The association between conduct problems and maltreatment: Testing genetic and environmental mediation. *Behavior Genetics*, *40*, 338–348.

Schulz-Heik, R. J., Rhee, S. H., Silvern, L., Lessem, J. M., Haberstick, B. C., Hopfer, C., … Hewitt, J. K. (2009).

https://doi.org/10.1017/thg.2015.62 Published online by Cambridge University Press

Investigation of genetically mediated child effects on maltreatment. *Behavior Genetics*, *39*, 265–276.

Shonkoff, J. P., Boyce, W. T., & McEwen, B. S. (2009). Neuroscience, molecular biology, and the childhood roots of health disparities. *Journal of the American Medical Association*, *301*, 2252–2259.

Snieder, H., Harshfield, G. A., & Treiber, F. A. (2003). Heritability of blood pressure and hemodynamics in African- and European-American youth. *Hypertension*, *41*, 1196–1201.

Springer, K. W. (2009). Childhood physical abuse and midlife physical health: Testing a multi-pathway life course model. *Social Science and Medicine*, *69*, 138–146.

Springer, K. W., Sheridan, J., & Kuo, D., & Carnes, M. (2003). The long-term health outcomes of childhood abuse. *Journal of General Internal Medicine*, *18*, 864–870.

Straus, M. A. (1979). Measuring intra family conflict and violence: The conflict tactics scale. *Journal of Marriage and the Family*, *41*, 75–88.

Straus, M. A., Hamby, S. L., Finkelhor, D., Moore, D. W., & Runyan, D. (1998). Identification of child maltreatment with the parent-child conflict tactics scales: Development and psychometric data for a national sample of American parents. *Child Abuse & Neglect*, *22*, 249–270.

Stunkard, A. J., Harris, J. R., Pedersen, N. L., & McClearn, G. E. (1990). The body-mass index of twins who have been reared apart. *New England Journal of Medicine*, *322*, 1483–1487.

Turner, H. A., & Butler, M. J. (2003). Direct and indirect effects of childhood adversity on depressive symptoms in young adults. *Journal of Youth and Adolescence*, *32*, 89–103.

Yi, Z., Gu, D., & Land, K. C. (2007). The association of childhood socioeconomic conditions with healthy longevity at the oldest-old ages in China. *Demography*, *44*, 497–518.

https://doi.org/10.1017/thg.2015.62 Published online by Cambridge University Press