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Journal

Health Psychology, 33(5)

ISSN

0278-6133

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Publication Date

2014-05-01

DOI

10.1037/a0032752

Peer reviewed

HHS Public Access

Author manuscript

Health Psychol. Author manuscript; available in PMC 2016 April 15.

Published in final edited form as:

Health Psychol. 2014 May ; 33(5): 410–418. doi:10.1037/a0032752.

Early Adversity and Health Outcomes in Young Adulthood: The Role of Ongoing Stress

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Abstract

Objective—The current study examined the prospective effects of exposure to stressful conditions in early childhood on physical health in young adulthood, and explored continuing exposure to stressors, as well as depression, in adolescence as possible mechanisms of this relationship.

Design—A prospective longitudinal design was used to examine 705 mother-child pairs from a community-based sample, followed from offspring birth through age 20.

Main Outcome Measures—Mothers provided contemporaneous assessments of early adverse conditions from offspring birth through age 5. Offspring responses to the UCLA Life Stress Interview, Structured Clinical Interview for DSM Disorders (SCID), Physical Functioning subscale of the SF-36 Health Survey, and questions about the presence of chronic disease were used to assess youth stress at age 15, depression from ages 15 to 20, and physical health at age 20.

Results—Early adversity conferred risk for elevated levels of social and non-social stress at youth age 15, as well as depression between ages 15 and 20. Social and non-social stress in turn had effects on physical health at age 20, directly and indirectly via depression.

Conclusions—Findings suggest that early adverse conditions have lasting implications for physical health, and that continued exposure to increased levels of both social and non-social stress in adolescence, as well as the presence of depression, might be important mechanisms by which early adversity impacts later physical health.

Keywords

Early adversity; stress; depression; health; community sample

Early adverse experiences, such as chaotic family environments, low socioeconomic status, and experiences of abuse, have been associated with a variety of poor health outcomes in adolescence and adulthood, including chronic illness and higher death rates from disease

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(Felitti et al., 1998; Davis, Luecken, & Zautra, 2005; Power, Hypponen, & Smith, 2005; Springer, Sheridan, Kuo, & Carnes, 2007). Similarly, markers for risk of disease and death, such as inflammation and increased blood pressure, have been linked to adverse environments in childhood (Danese, Pariante, Caspi, Taylor, & Poulton, 2007; Slopen, Koenen, & Kubzansky, 2012; Loucks, Almeida, Taylor, & Matthews, 2011). Despite this striking evidence for the long-term health consequences of early stressful experiences, the mechanisms by which early adversity prospectively shapes health remain largely unknown.

One potential explanation for this relationship involves the contributions of early adversity to continuing stress exposure. That is, children who are exposed to early adverse experiences might continue to be exposed to more stressful experiences, such as reduced support in social relationships or financial difficulties, throughout adolescence and young adulthood. This continuity of stress is hypothesized to occur because experiences of early adversity tend to be rooted in relatively stable structural contexts, such as financial instability or family conflict, which increase the likelihood of later exposure to stress (Pearlin, 1989). In addition, early adversity has lasting effects on children's cognitive and interpersonal styles (Bifulco, Moran, Ball, & Lillie, 2002; Hankin, 2005), as well as on their acquisition of coping behaviors (Turner & Lloyd, 1995). These negative effects of early adversity are likely to influence the types of environments that children select into later in life, as well as increase the probability that individuals will perceive stress and react in ways that escalate stressful circumstances, such as interpersonal conflict (e.g., Campbell, Simpson, Boldry, & Kashy, 2005).

In support of this hypothesis, early adversity has been shown to create a vulnerability to increased chronic and episodic stress later in life (Hammen, Hazel, Brennan, & Najman, 2012; Hazel, Hammen, Brennan, & Najman, 2008; Uhrlass & Gibb, 2007). This continuity of stress has been examined mostly in relation to mental health outcomes, and in these studies, ongoing stress exposure appears to partially explain the effects of childhood adversity on later mental health, particularly depressive symptoms (Hazel et al., 2008; Kessler & Magee, 1994; Turner & Butler, 2003; Uhrlass & Gibb, 2007). However, there is reason to think that ongoing stress exposure might also affect physical health outcomes. There is evidence linking both social Cohen et al., 1997; Troxel, Matthews, Gallo, & Kuller, 2005) and non-social stress (Deinzer, Kleineidam, Stiller-Winkler, Idel, & Bachg, 2000; Paik, Toh, Lee, Kim, & Lee, 2000) with indicators of physical health. Both types of stress are thought to impact physical health by triggering inflammation, a biological process that has been implicated in a variety of negative health outcomes (Avitsur, Powell, Padgett, & Sheridan, 2009; Miller, Rohleder, & Cole, 2009). Stress has also been shown to negatively influence individuals' health behavior, including smoking, drinking, sexual behavior, and exercise and eating patterns (Conway, Vickers, Ward, & Rahe, 1981; Steptoe et al., 1996).

Depression might also serve as an important factor in the effects of early adversity and ongoing stress on physical health. There is a robust relationship between stress and later depression (Hammen, 2005), and depression has been associated with indicators of poor health, such as inflammation, as well as increased morbidity from physical health conditions (Kiecolt-Glaser & Glaser, 2002). Thus, although both social and non-social stress might have direct effects on health, it is also likely that they affect physical health indirectly via

increases in depressive symptomatology. There is some that evidence that early adversities such as socioeconomic adversity, might lead to both poor mental health and poor physical health, which have reciprocal influences on one another (Wickrama, Conger, & Abraham, 2005). Nevertheless, no study to-date has examined social and non-social stress as specific mechanisms of the relationship between early adversity and poor physical health, taking into account the indirect influences of stress on health via depression.

Existing research on multiple adversities in early childhood indicates that adversities tend to be highly clustered, and a composite of early adversities might therefore best capture the interrelatedness of early childhood adversities, and their cumulative impact on future stress and health (Dong et al., 2004; Evans & Kim, 2007; Finkelhor, Ormrod, & Turner, 2007; Green et al., 2010). However, previous studies have also relied almost exclusively upon retrospective reports of early adversities (e.g., Kessler & Magee, 1994; Uhrlass & Gibb, 2007), which can be inaccurate and suffer from high rates of false negatives (Hardt & Rutter, 2004).

Moreover, in studies of stress and physical health, the construct and measurement of stress have been highly diverse, ranging from the psychological construct of perceived stress (e.g., Troxel et al., 2005), to animal models (Avitsur et al., 2009), to stress interviews that assess environmental characteristics of the context in which stressors occur (Miller et al., 2009). It is therefore unclear whether findings reflect the effects of subjective or objective experiences of stress on health. In addition, recent research has suggested that social and non-social stressors have different effects on emotional and biological systems in ways that might have distinct implications for physical health. For example, social evaluative stressors, a particular type of social stressor, have been shown to have a stronger effect on the HPA axis and inflammation than stressors without social evaluation (Dickerson et al., 2009; Dickerson & Kemeny, 2004). Moreover, stress in social relationships has been found to predict depression more strongly than non-interpersonal stressors (Hammen, 2005). Despite this fact, few studies have examined the effects of social and non-social stress on health simultaneously, but separately.

The present project seeks to address these gaps in the existing literature by examining the effects of a composite measure of early adversity on physical health, and testing ongoing social stress, non-social stress, and depression as specific mechanisms of this relationship. To explore these pathways, the current project utilizes a longitudinal dataset with contemporaneous assessment of early childhood adversities at multiple timepoints during the first 5 years of the child's life, contextual assessment of levels of social and non-social stress during adolescence, and several measures of physical health in young adulthood. The use of multiple timepoints (in early childhood, adolescence, and young adulthood) allows analyses to more precisely determine the temporal relationships among variables of interest. We hypothesize that children's experiences of adversity by age 5 predict poorer physical health outcomes (self-rated health, interviewer-rated health, and presence of chronic disease) in young adulthood. We also test whether early adversity by age 5 predicts higher levels of chronic and acute stress across social domains and non-social domains, as well as the presence of depression, during adolescence. We hypothesize that both stress domains in turn

have direct effects on physical health, as well as indirect effects on physical health via youth depression.

Method

Participants

From a birth cohort study of children's development through age 5 including more than 7,000 children (Mater-University of Queensland Study of Pregnancy (MUSP); Keeping et al., 1989), 815 mother-child pairs were selected for a follow-up study of children at risk for depressive and other disorders at youth age 15, based on mothers' multiple reports of depressive symptoms on the Delusions-Symptoms States Inventory (DSSI; Bedford & Foulds, 1978) from pregnancy through child age 5. Families were selected to represent a range of symptom presence, chronicity, and severity of maternal depression, later verified by diagnostic interviews (see Hammen & Brennan, 2001 for details). From the original sample, 991 families were targeted for inclusion in the follow-up, and 815 consented and were included. The adolescent sample at age 15 was 50.4% male and 49.4% female. Families were largely lower and lower-middle income and predominantly Caucasian (91.4%; 3.6% Asian; 5% other or not reported).

At youth age 20, the mother-child pairs that participated at age 15 were again contacted for follow-up, and 705 (363 females) completed age 20 procedures. Mother-child pairs that participated in the age 20 follow-up did not differ from those who did not participate in terms of youth history of depression by age 15 ($\chi^2(1, n = 705) = 1.33$, $p = .25$) or mothers' marital status at youth age 15 ($\chi^2(2, n = 791) = .79$, $p = .48$). The youths who did not participate in age 20 procedures had lower family income at youth age 15 ($t(782) = 2.39$, $p < .05$) and were more likely to be male ($\chi^2(1, n = 815) = 8.71$, $p < .005$).

Procedure

Mothers completed measures at 4 time points during the child's early life (at their first prenatal visit—typically in the first trimester of pregnancy, 3-4 days after the child's birth, 6 months after birth, age 5), and at youth ages 15 and 20. Children completed measures at ages 15 and 20. These precise timepoints were selected largely on practical grounds (e.g., funding), but were intended to examine outcomes emerging from maternal and child health and environmental conditions across adolescence and early adulthood. At time points prior to and including age 5, questionnaires administered to mothers asked about maternal symptomatology, mothers' stress and social experiences, children's health and behaviors, and family demographic information. Interviews and questionnaires administered to mothers and youth at youth ages 15 and 20 asked about mother and child psychopathology, youth chronic and acute stress exposure, youth depressive symptomatology, and youth physical health. Postgraduate students were trained to appropriately conduct and reliably score interviews for the assessments at youth ages 15 and 20. Participants all gave informed consent (assent) and the institutional review/ethics panels of the University of Queensland, Emory University, and the University of California, Los Angeles approved the research protocol for the ages 15 and 20 follow-ups.

Measures

Early adversity—Five indicators were used to index exposure to adversities during the first five years of the child's life, based on information provided in the mother questionnaires at the 4 time points in early childhood. Parental separation was scored as present or absent based on mothers' reports of separating from her partner at any time during the first five years after the child's birth. Family income was assessed using an average of maternal ratings of the family's annual income (on a 7-point scale) at 3 of the early childhood assessments: pregnancy, 6 months after birth, and 5 years after birth. Relationship discord was assessed using the mean of mothers' reports of relationship satisfaction on the 8-item satisfaction scale of the Dyadic Adjustment Scale (DAS; Spanier, 1976); alphas, .85 to .97) at all 4 early childhood assessments. Maternal stress was measured using a checklist of 9 interpersonal, health, or occupational problems that occurred over the past 6 months, which was administered to mothers once during pregnancy and once at the child's birth. Finally, maternal depressive symptoms were assessed using the mean of mothers' reports on the DSSI at all 4 early childhood assessments. Importantly, these contemporaneous reports of maternal depressive symptoms corresponded significantly with retrospective diagnoses for maternal depressive disorders as measured by the Structured Clinical Interview for DSM-IV for lifetime disorders (SCID; First, Spitzer, Gibbon, & Williams, 1995) administered to mothers at youth age 15 ($\chi^2 = 42.59$, $p < .001$).

Pearson and point-biserial correlations among individual early adversities and health outcomes are presented in Table 1. A count of the number of adversities for each child was used as a cumulative measure of early childhood adversity, due to the fact that adversities have been found to cluster, and cumulative risk might be an important predictor of physical health. In order to create this composite, the continuous measures of early adversity were coded as either present or absent, using the 33rd percentile as the cut-off point. This cut-off point has been used in previous studies and was chosen to balance the need for a sufficient sample size for meaningful analyses with the selection of a reasonably adverse level of severity for each variable (Hazel et al., 2008). Due to the fact that few participants had all 5 adversities, participants with 4 or more adversities were combined to create a composite with a more normal distribution (range 0 to 4). An alternative total early adversity severity score was also calculated for each individual in order to capture the full variability of continuous variables. To create this severity score, continuous variables were standardized, and then values for all 5 individual adversities were summed.

Stress—Measures of youth stress were derived from semi-structured interviews with adolescents at youth age 15, using the UCLA Life Stress Interview (Hammen, 1991). The life stress interview is a face-to-face semi-structured interview that uses standard questions to probe adolescents' experiences with chronic and acute stress over the past 6 months. Chronic stress assessment involved probing each of several domains of functioning, and the interviewer assigned an objective severity rating for the level of chronic stress in each domain. Each domain was rated on a 5-point scale, using behaviorally specific anchor points (with 1 indicating exceptionally good conditions and 5 indicating extreme adversity). The four domains with social content (romantic relationships, relationship with a best friend, family relationships, and social life) were summed. Similarly, the two domains with non-

social content (academic performance and school behavior) were summed. Intraclass correlations for domains in the current sample were social life, .63; close friendship, .76; relationship with family members, .84; romantic relationship, .55; academic performance, .94; school behavior, .88.

Acute stress (life events) was assessed with a contextual approach, with the interviewer eliciting specific information about the nature and circumstances of each acute social or non-social stressor reported by adolescents as occurring in the past 12 months. Interviewers wrote narratives of each event that were presented to a team of raters who were blind to the adolescent's depression status and subjective reactions to the event. Individual acute stressors were judged by the team as having primarily social or non-social content, and were rated by the team for severity, taking into account the context in which the stressor occurred in order to judge the objective level of stress that the event would cause to the average individual. Stress severity was rated on a 5 point scale, with 1 indicating no stress and 5 indicating extremely severe stress. Interrater reliabilities based on independent ratings for 89 cases of both social and non-social stress yielded intraclass correlations of .92 for severity ratings. Severity levels were summed across all events with social content and across all events with non-social content.

Chronic and acute stress totals were combined to create composites of social and non-social stress in adolescence. Composite measures of acute and chronic stress are hypothesized to be more accurate indices of overall stress burden, since acute stressors often arise out of chronically stressful contexts (Pearlin, 1989). Consistent with previous literature, the chronic and acute social stress variables were standardized and summed to create a measure of total social stress burden (Hazel et al., 2008; Turner, Wheaton, & Lloyd, 1995). Correlations between acute and chronic stress were .17 ($p < .001$) for non-social stress and .10 ($p < .05$) for social stress.

Youth depression—The presence or absence of youth depression between ages 15 and 20 was assessed at age 20 using the SCID, which covered the past five years since the age 15 assessment. Independent judges' ratings of taped interviews yielded a significant Kappa for depression over the past 5 years (0.89). For the current project, onset of youth depression was coded as present between ages 15 and 20 if they met criteria for major depression, dysthymia, or depression not otherwise specified.

Physical health—Physical health outcomes in young adulthood were measured in three ways. First, the Physical Functioning subscale of the SF-36 Health Survey (Ware, Snow, & Kosinski, 2000), a well-validated self-report measure of health-related quality of life, was administered at the youth age 20 follow-up. The Physical Functioning subscale contains 10 items that evaluate the extent to which a person is limited in the performance of physical activities by their health (Ware, Jr. & Sherbourne, 1992). This subscale has been shown to be one of the best measures of pure physical health out of the SF-36 subscales and is able to predict severity of chronic medical conditions (McHorney, Ware, & Raczek, 1993). The alpha coefficient for these 10 items in the current sample was .92, indicating high internal consistency.

Second, general health functioning over the past 6 months was assessed at age 20 using the Health of Self chronic stress domain of UCLA Life Stress Interview. Using both general questions and specific probes, interviewers determined each youth's general functioning in the health domain, using behaviorally specific anchor points. Behavioral anchors for these ratings included information relevant to markers of actual disease (e.g., if the participant was overweight, ratings of health were automatically rated .5 points worse). The quality of each youth's health was rated using a 5-point scale, with 1 indicating exceptionally good health and 5 indicating a severe, life-threatening health problem (interrater reliability: .77).

Finally, at age 20 youth completed a checklist to report whether had one or more of 16 chronic diseases, and chronic illness was scored as present/absent. The most commonly endorsed chronic illnesses were asthma (n = 73), migraines (n = 41), and eczema (n = 22).

Physical health outcomes were validated against other variables in the dataset to ensure their relevance for actual disease outcomes. Both SF-36 physical functioning and interviewer-ratings of physical health were significantly correlated with age 20 youth reports of somatic symptoms including twitching, feeling dizzy, feeling overtired, aches and pains, headaches, nausea, eye problems, rashes, stomachaches, heart pounding or racing, and numbness or tingling, on the Young Adult Self Report of Child Behavior Check List (all p's < .05). In addition, both physical health outcomes predicted actual healthcare utilization at age 20 (both p's < .001).

Data Analyses

In order to evaluate the effects of early adversity on later physical health in the current sample, linear regression analyses were used to examine the prospective effects of early adversity (up to age 5) on Physical Functioning scores and interviewer-rated health at age 20. Logistic regression analyses were carried out to examine the prospective effect of early adversity on the presence or absence of chronic disease at age 20. The presence/absence of youth childhood chronic illness and gender were covaried for in these analyses.

Using a structural equation modeling (SEM) framework, we tested the indirect effect of early adversity on physical health via social stress, non-social stress, and depression. SEM permits the simultaneous examination of multiple indirect pathways from early adversity to young adult heath outcomes. Overall model fit was evaluated using several standard fit indices, including the likelihood ratio chi-square test, the comparative fit index (CFI; Bentler, 1990), the root-mean-square error of approximation (RMSEA; Browne & Cudeck, 1993), and the weighted root mean-square residual (WRMR; Muthén & Muthén, 1998-2007). All analyses were carried out in Mplus v5 using the WSLMV estimator to accommodate categorical endogenous variables (Muthén & Muthén, 1998-2007).

Results

Descriptive statistics for all main study variables, as well as Pearson and point-biserial correlations among these variables, are presented in Table 2.

First, we tested the effect of early adversity on physical health in young adulthood, using each of the three health outcome variables. Results of the corresponding regression analyses are presented in Table 3. These analyses revealed that the experience of a greater number of adversities by age 5 predicted significantly worse scores on the Physical Functioning subscale of the SF-36 at age 20, covarying for youth gender and childhood chronic illness. In addition, the number of early adversities by age 5 was marginally significant in predicting worse interviewer-rated health over the past 6 months at age 20, covarying for youth gender and childhood chronic illness. However, the logistic regression analysis revealed that early adversity was not a significant predictor of the presence of chronic disease at age 20. When these analyses were conducted using the alternative method of a total early adversity severity score, the pattern and significance of results were identical: total adversity severity significantly predicted the Physical Functioning subscale of the SF-36 ($b = .69$, $SE = .24$, $p < .01$, $\beta = .11$) and interviewer-rated health ($b = .02$, $SE = .01$, $p < .05$, $\beta = .09$), but did not predict the presence of chronic disease ($b = .05$, SE = .04, Wald = 1.39, OR = 1.05, 95% CI [.97 1.13], $p = .24$).

Second, we evaluated a mediational model that examined the effects of the number of early adversities on social, non-social stress, and the presence of depression in adolescence, as well as the effects of both types of stress on depression and physical health in young adulthood (Figure 1). Social and non-social stress, as well as the two physical health outcomes, were allowed to correlate. Chronic disease was not included as an outcome, given that earlier analyses indicated that the direct association between early adversity and chronic disease was not statistically significant. Gender was covaried for in all stages of the analysis where it was found to be a significant predictor. Standardized beta values are shown in Figure 1.

Fit indices indicated that the model tested provided a good overall fit to the data $\chi 2$ (df = 4, $N = 815$) = 10.42, $p = .03$; CFI = .98; RMSEA = .04 (90% CI .01, .08); WRMR = .56. As hypothesized, early adversity had a significant indirect effect on both self-reported physical functioning ($\beta = .05$, $p \leq .001$) and interviewer-rated health ($\beta = .09$, $p < .001$). In addition, most hypothesized direct paths within the model were significant. Early adversity significantly predicted both the social and non-social stress composites, as well as the presence of depression between ages 15 and 20. Social stress in adolescence in turn also predicted the presence of youth depression, but non-social stress did not predict depression. Social stress in adolescence, non-social stress in adolescence, and the presence of youth depression between ages 15 and 20 all had significant direct effects on interviewer-rated health at age 20. In addition, non-social stress in adolescence and youth depression between ages 15 and 20 had significant direct effects on self-reported physical functioning at age 20, although social stress in adolescence did not reach significance as a predictor of self-reported physical functioning ($p = .11$).

When the model was re-run using the alternative method of an early adversity severity score, fit indicates again indicated that the model tested provided a good overall fit to the data $\chi 2$ (df = 4, $N = 704$) = 7.36, $p = .12$; CFI = .99; RMSEA = .04 (90% CI .00, .07); WRMR = .47. In addition, the fit indicated the same patterns of significance for both direct and indirect paths in the model, except that social stress in adolescence became a significant predictor of

self-reported physical functioning at age 20, while depression between ages 15 and 20 became marginally significant as a predictor of self-reported physical functioning.[1]

Discussion

The present study explored the effects of early childhood adversity on physical health in young adulthood, and examined several pathways involved in these effects: ongoing social stress, non-social stress, and depression in adolescence. Results suggested that early adverse experiences significantly compromise physical health, as measured by the physical functioning subscale of the SF-36 and interviewer ratings of physical health, in young adulthood, a developmental period typically associated with robust health and prior to the development of most chronic diseases. In addition, findings showed that continuity of both social and non-social stress, as well as depression, are important mechanisms of this relationship. Higher levels of social and non-social stress in adolescence, as well as the presence of clinically significant depression between ages 15 and 20, had effects on physical health, and early adversity had a significant indirect effect on both physical health outcomes via these mechanisms.

Results suggesting that early adversity predicts poorer physical functioning and interviewer-rated health in young adulthood are consistent with previous research on the effects of early adversity on later physical health (e.g., Felitti et al., 1998). However, previous studies have relied almost exclusively on retrospective reports of childhood adversity, which tend to have high rates of false negatives and significant measurement error (Hardt & Rutter, 2004). The use of retrospective reporting for early adversity is likely to be especially problematic in studies of adults, given that they must report on experiences from many years earlier. The present project improved upon these designs by using a longitudinal dataset to show that early adversities measured by contemporaneous maternal report during the first five years of the child's life, were predictive of multiple measures of poor physical health in young adulthood.

Past studies of early adversity have also tended to focus on the negative effects of particular adversities, such as physical abuse (e.g., Shaw & Krause, 2002) or socioeconomic status (e.g., Power, Hypponen, & Smith, 2005). However, existing evidence suggests that early adverse experiences often occur in clusters rather than in isolation, and a composite of adversity might therefore more accurately represent the cumulative effects of multiple early adverse contexts (e.g., Evans & Kim, 2007; Green et al., 2010). As a result, the present study built upon past findings by using a measure of cumulative adversity that encompasses a number of different adversities to which a child may be exposed, consistent with previous research on the effects of early adversity on adult mental health (Hazel et al., 2008; Green et al., 2010).

1Additional analyses incorporating the youth's retrospective report of physical and/or sexual abuse up to age 15 into the count of early adversities did not show any appreciable changes in the results. As a result, these analyses have been omitted here in favor of a potentially less biased early adversity variable containing only adversities that occurred before age 5 and were based on mother's contemporaneous reports of adversity.

Despite the robust effects of early adversity on self-report and interview measures of physical health, early adversity did not predict the presence of chronic disease by age 20. The fact that physical health was examined in young adulthood likely contributed to this finding, given that most chronic diseases do not develop by age 20. Markers of risk for future disease, such as measures of general health and indicators of inflammation, might therefore be better measures of physical health in young adulthood. In addition, it is possible that covarying for childhood history of chronic illness also played a role in this finding, since the few chronic illnesses that are present in young adulthood might already have developed by age 5 (e.g., asthma). More sensitive measures of fluctuations in the symptom severity of chronic illnesses might therefore be necessary to explore whether early adversity and social stress affect the course and eventual outcomes of childhood chronic illnesses in young adulthood.

Results also provide support for the hypothesis that early adversity predisposes children to experiences of ongoing stress in adolescence, both in social and in non-social domains. The magnitude of the effects of early adversity on social and non-social were essentially the same. These findings are consistent with theories of stress that emphasize that stressful experiences do not usually occur in isolation. Instead, early adversity often predicts later experiences of stressful life events, resulting for some individuals in continued exposures to stress that can compound the deleterious effects of early adversity (e.g., Hazel et al., 2008). This continuity in stress exposure is thought to occur for several different reasons. First, early adversity and stressful life events are often rooted in stable contexts, such as socioeconomic disadvantage or family dysfunction, that predispose individuals to experience greater stress at other timepoints as well (Pearlin, 1989). In addition, adversities experienced in early childhood likely interfere with the acquisition of social skills and cognitive schemas, which can in turn affect individuals' selection into certain social and academic environments (Bifulco et al., 2002; Turner & Lloyd, 1995).

Previous work has provided some evidence in support of these theories, showing that early adversity might cause higher levels of overall stress at later timepoints (e.g., Hazel et al., 2008), as well as interfere specifically with later social relationships (Malinosky-Rummell & Hansen, 1993; Shaw & Krause, 2002). However, these studies have relied largely upon retrospective self-reports of childhood abuse, as well as self-report measures of stressful events later in life. Moreover, none of these studies has examined the specific effects of early adversity on social and non-social stress simultaneously. The present study therefore expanded upon these previous findings by examining in a single model, contextual, interviewer-rated measures of chronic and acute stress across multiple domains of social and non-social functioning.

Finally, results also showed that there was a significant indirect effect of early adversity on both self-reported physical functioning and interviewer-rated health through social stress, non-social stress, and depression in adolescence. Both stress domains and depression were significantly predicted by early adversity, and each of these variables also had negative effects on physical health. However, social stress appeared to be having negative physical health effects at least partially through depression, while non-social stress did not predict later depression and instead only had direct effects on physical health. Overall, these path

model findings suggest that both social stress and non-social stress in adolescence are important mechanisms by which early adversity has lasting effects on physical health in young adulthood. This is consistent with an existing body of literature showing that stress has important implications for later physical health, and can lead to increased risk for serious illness and death (e.g., Cohen et al., 1997; Troxel et al., 2005). In addition, results suggest that depression also plays an important role in the effects of early adversity and social stress on health. The lack of a significant effect of non-social stress on depression is consistent with a body of evidence suggesting that social stress might be a more potent predictor of later depression than non-social stress (e.g., Hammen, 2005). Non-social stress might therefore have effects on physical health through pathways other than depression.

Despite the conceptual and methodological advantages of the current study, several limitations must be acknowledged. First, our measures did not include several common adversity variables, such as neglect, and analyses focused on cumulative risk, rather than examining the relative impacts of specific adversities. Future studies would benefit from prospective studies of the effects of specific adversities using more precise methods, to supplement our focus on cumulative risk. In addition, neither of the two physical health outcomes used in path model analyses is a directly observed physiological marker of disease risk. The physical functioning subscale of the SF-36 has been validated using clinically accepted markers of serious health conditions (Ware et al., 2000), and interviewers used behavioral anchors related to disease markers, such as weight, in order to make health ratings. Nevertheless, future work will need to examine similar models using biological markers of disease to more precisely determine the effects of early adversity and later stress on physical health conditions.

Present analyses were conducted with a community sample over-selected for the presence of maternal depression (and hence, youth depression). This method had the advantage of emphasizing the effects of stress and depression, but the disadvantage of lack of generalizability to normal populations. To address the depression issue in the current analyses, early maternal depression was included as a part of the early adversity composite, and youth depression was included in the hypothesized path model. Nevertheless, findings still are likely not indicative of what would be found in a truly random community sample. In addition, the current sample was limited in terms of socioeconomic diversity due to the population served by the Mater Hospital, and ethnic diversity due to the Australian general population of that era. As a result, findings should be replicated in more ethnically diverse populations, and in random community samples that are more representative of the general population in terms of socioeconomic status.

Future studies in this area might benefit from comparing different ways in which specific clusters of adversities affect physical health. There are likely differential effects on health for different combinations of contextual stressors and adversities (e.g., Green et al., 2010). In addition, it is possible that acute and chronic stress play different roles in the relationship between early adversity and physical health, and future studies might investigate more complex models that take this into account. Future research should examine the biological pathways involved in the effects of early adversity on social functioning and physical health. In particular, markers of inflammatory activity have been studied in relation to both early

adversity (Danese et al., 2007; Slopen et al., 2012) and social stress and behavior (Eisenberger, Inagaki, Mashal, & Irwin, 2010; Miller et al., 2009), and inflammation is associated with the development of various serious illnesses.

Finally, small effect sizes in the current findings indicate that the amount of variance accounted for by the hypothesized mechanisms is relatively small, and additional variables not included here are therefore contributing to the outcomes. Future studies should consider inclusion of potential biological mechanisms, as well as additional psychosocial and environmental factors, such as ongoing financial instability, poor access to healthcare, and risky health behaviors. As noted above, other refined measures of early adversity exposure and objective health outcomes might also yield stronger patterns of relationships. Future studies that address these increasingly complex models and identify robust predictors will be needed to translate long-term longitudinal models into clinically relevant targets. Moreover, although the present study represents an advance in our theoretical understanding of the long-term effects of early adversity, future studies are needed to further explore earlier indicators of these negative pathways, such as cognitive biases or behavioral tendencies that might lead to stressful social interactions in childhood and early adolescence. Such developmentally informed analyses might help to refine the questions of what interventions at what timepoints could reduce risk for poor social and physical functioning in adolescence and young adulthood.

Acknowledgements

This research was supported by the National Health and Medical Research Council, the Mater Misericordiae Mother's Hospital in Queensland, Australia, the NIMH grant R01 MH52239, and an NIMH health psychology predoctoral training grant MH15750 (Dr. Stanton & Dr. Dunkel-Schetter). We thank Professor Jake Najman of the University of Queensland and MUSP colleagues William Bor, M.D., and Gail Williams, Ph.D. Special thanks to project coordinators Robyne LeBrocque, Cheri Dalton Comber, and Sascha Hardwicke. Finally, we appreciate the helpful feedback of Christopher Conway and three anonymous reviewers on a previous version of this article.

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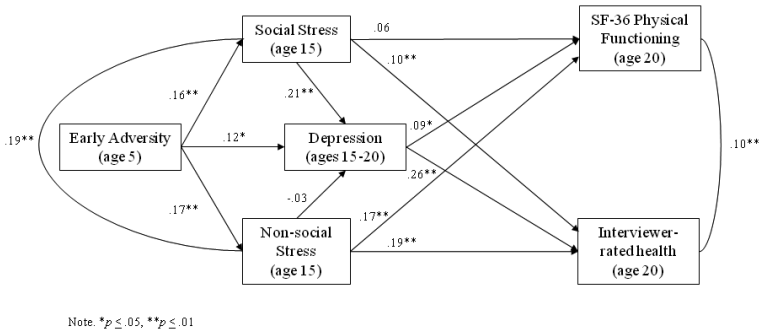

Figure 1.

The effects of early adversity on SF-36 physical functioning and interviewer-rated health, via social stress, non-social stress, and depression.

Table 1

Correlations among Individual Early Adversities and Health Outcomes

Study Variable	1	2	3	4	5	SF36 Physical Functioning	Interviewer-rated health
1. Parental separation	--					.06	.08 *
2. Maternal depressive symptoms	.20 **	--				.08 *	.09 *
3. Marital satisfaction	−.38 **	−.42 **	--			−.07 +	−.05
4. Maternal stressful life events	.27 **	.37 **	−.41 **	--		.07	.06
5. Family income	−.36 **	−.23 **	.29 **	−.33 **	--	−.09 *	−.08 *

Note.

* $p \leq .05$

** $p \leq .01$

+ $p < .10$

Table 2

Correlation Matrix of Early Adversity, Stress, and Physical Health Variables

Study Variable	M	SD	Range	1	2	3	4	5
1. Early adversity	1.64	1.36	0-4	--				
2. Social Stress at 15	0	1.47	−3.4-7.1	.16**	--			
3. Non-social Stress at 15	0	1.47	−2.7-9.0	.13**	.19**			
4. SF-36 Physical Functioning at 20	89.97	18.08	0-100	.14**	.13**	.19**	--	
5. Interviewer-rated physical health at 20	2.31	0.57	1-4.5	.09*	.19**	.21**	.17**	--
6. Presence of chronic disease at 20	0.23	0.42	0-1	0.06	.20**	.12**	0.05	.24**

Note.

*$p \leq .05$

**$p \leq .01$

Table 3

Regression Analyses Predicting Health from Early Adversity

	SF-36 Physical Functioning (N = 619)				Interviewer-rated Health (N = 697)				Presence of Chronic Disease (N = 464)				
	b	***SE***	***β***	**ΔR^2**	***b***	***SE***	***p***	**ΔR^2**	***B***	***SE***	**Wald**	**OR**	**95% CI**
Step 1				0.01				0.02					
Child illness	1.62	2.27	0.03		0.26**	0.07	0.15		0.83**	0.30	7.42	2.28	1.26-4.14
Gender	2.90*	1.44	0.08		0.03	0.04	0.03		1.1**	0.25	19.94	3.07	1.88-5.02
Step 2				0.02				0.02					
Child illness	.88	2.26	0.02		0.24**	0.07	0.14		0.79**	0.31	6.69	2.21	1.21-4.02
Gender	2.83*	1.43	0.08		0.03	0.04	0.03		1.13**	0.25	20.08	3.08	1.88-5.04
Early adversity	1.75**	0.53	0.13		0.03+	0.02	0.07		0.07	0.08	.74	1.07	.91-1.27

Note.

*$p \leq .05$

**$p \leq .01$

+$p \leq .06$