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Original Article

What the future held: childhood psychosocial adversity is associated with health deterioration through adulthood in a cohort of British women

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ABSTRACT

Childhood psychosocial adversity is associated with accelerated onset of reproductive effort in women. Adaptive explanations for this phenomenon are built on the assumption that greater childhood psychosocial adversity is statistically associated with having a shorter period of healthy adult life during which reproduction will be possible. However, this critical assumption is never actually tested using individual-level longitudinal data. In this study, I revisit a large, longitudinally-studied cohort of British women. In an earlier paper, we showed that a simple index of psychosocial adversity in the first seven years of life predicted age at first pregnancy in a dose-dependent manner. Here, I show that the same index of adversity also predicts accelerated deterioration of health across the potentially reproductive period, and increased levels of the inflammatory biomarker *c*-reactive protein at age 44–46. These associations are robust to controlling for adult socioeconomic position, and do not appear to be solely a consequence of accelerated reproductive schedule. I argue that childhood psychosocial adversity may cause latent somatic damage that will, in adulthood, accelerate age-related physical decline. This provides a compelling adaptive rationale for the accelerated reproductive schedules observed in women who experience childhood psychosocial adversity.

1. Introduction

> He says his body's too old for working; His body's too young to look like his.
>
> [Tracy Chapman, *Fast Car*]

A large corpus of work has shown that women who experience childhood psychosocial adversity tend to go on to exhibit relatively early menarche, sexual debut, and first pregnancy (e.g. Alvergne, Faurie, & Raymond, 2008; Belsky, Steinberg, & Draper, 1991; Belsky et al., 2007; Chisholm, Quinlivan, Petersen, & Coall, 2005; Ellis et al., 2003; Nettle, Coall, & Dickins, 2011; Pesonen et al., 2008; Tither & Ellis, 2008). This acceleration of reproductive schedule in response to childhood psychosocial adversity has been viewed as an evolved adaptive response, rather than as a consequence of system dysregulation (Belsky et al., 1991; Chisholm, 1993; Ellis, Figueredo, & Schlomer, 2009). In general, as the risk of becoming unable to reproduce due to morbidity or mortality over the adult period increases, the optimal age at which to initiate reproductive effort becomes younger; that is, the optimal reproductive strategy becomes 'faster' (Charnov, 1991; Cichon, 1997). Thus, for acceleration in response to childhood psychosocial adversity to be an adaptive strategy, childhood psychosocial adversity must somehow be statistically associated with some aspect of morbidity or mortality risk in adulthood (Chisholm, 1993; Ellis et al., 2009). A number of studies have demonstrated correlations between average life expectancy and average age at first reproduction at the population level (Bulled & Sosis, 2010; Low, Hazel, Parker, & Welch, 2008; Nettle, 2010a, 2011; Placek & Quinlan, 2012). However, adaptive explanations for reproductive acceleration in response to psychosocial adversity require more than this: they depend on individual exposure to childhood psychosocial adversity predicting subsequent individual morbidity or mortality.

There are a number of subtly different accounts of why childhood psychosocial adversity might be associated with increased morbidity or mortality risk in adulthood. Of particular relevance to the current study, Rickard, Frankenhuis, and Nettle (2014) argued that childhood psychosocial adversity may cause latent somatic damage that accelerates processes of age-related health deterioration later on in life. This idea builds on a number of earlier sources, and is inspired in particular by the 'weathering hypothesis' (Geronimus, 1992; Geronimus, Hicken, Keene, & Bound, 2006). The weathering hypothesis was originally developed to explain why the health gap between African-Americans and white Americans widens through mid-life. *Ex hypothesi*,

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adverse life situations force the child to divert energy into short-term survival needs as orchestrated by the physiological stress response. Repeated activation of the physiological stress imposes cumulative phenotypic costs in the long term, exactly because it reallocates effort away from self-repair and investment in the future value of bodily systems. Accelerated health deterioration with age is the manifestation of these accumulated costs in system-wide premature decline in performance. An individual whose health will deteriorate faster with age has a shorter expected window during which she can bear and parent children, and this alters the balance of costs and benefits in favour of a faster reproductive schedule (Geronimus, 1996a).

If accelerated health deterioration following childhood psychosocial adversity is to provide an adaptive explanation for the evolution of reproductive acceleration, then it is important to demonstrate that the accelerated health deterioration is not solely a consequence of the accelerated reproductive trajectory. If it were, the explanation would be circular (women accelerate their reproduction because they are going to die young, and die young because they expend their energy on early reproduction). To avoid this circularity, theoretical models in this area include a component of morbidity/mortality risk that is extrinsic, meaning that it is not modifiable by the strategies that the individual adopts (Cichon, 1997; Nettle, 2010b; Shokhirev & Johnson, 2014). It is this extrinsic component of mortality/morbidity risk that determines the optimal speed of reproductive trajectory; if extrinsic mortality/morbidity is high, there is nothing that the individual can do about it other than try to get some reproduction done whilst they can. However, by following an accelerated reproductive schedule, individuals may further increase their mortality/morbidity risk, since fast growth and reproductive effort are costly and take their toll on health and survival (Boonekamp, Salomons, Bouwhuis, Dijkstra, & Verhulst, 2014; Metcalfe & Monaghan, 2003; Reznick, 1985). Thus, any associations between childhood psychosocial adversity and adult health deterioration will represent some mixture of a direct primary effect of childhood adversity on the developing body, and a secondary indirect effect due to the reproductive and behavioural strategies that the individual has adopted in response to her adverse experiences (Ellis & Del Giudice, 2013). The adaptive account discussed by Rickard et al. (2014) requires that, over evolutionary time, the direct primary effect has been non-zero, since this effect constitutes the selection pressure to which reproductive acceleration is the adaptive response.

No study has yet investigated in the same cohort whether the psychosocial factors that predict accelerated reproductive strategy also predict accelerated health deterioration with age, and if so, to what extent this is a direct primary relationship rather than solely a consequence of reproductive acceleration. Failure to find a direct primary association between childhood psychosocial adversity and accelerated health deterioration in a modern population would not warrant a decisive rejection of the adaptive evolutionary hypothesis, since the hypothesis states that across ancestral populations, the experience of psychosocial adversity was, on average, associated with increased extrinsic mortality/morbidity risk. In modern populations, especially in the developed world, the availability of health care or other environmental factors might have mitigated any such relationships. However, there are a large number of epidemiological papers from developed populations showing that various types of childhood psychosocial adversity are associated with poorer health, reduced survival, or accelerated aging in adulthood (e.g. Dube, Felitti, Dong, Giles, & Anda, 2003; Felitti et al., 1998; Geronimus et al., 2006; Geronimus et al., 2010; Larson & Halfon, 2013). This suggests that relationships between childhood psychosocial adversity and adult health are evident even under conditions of affluence, and can be profitably explored in contemporary populations.

In this paper, I return to a large, longitudinally-studied cohort of British women who were the subjects of an earlier paper (Nettle et al., 2011). In that paper, we computed a simple index of psychosocial adversity in the first seven years of life, and showed that high scores on this index were associated with earlier first pregnancy once the cohort members had grown up. The association was robust and dose-dependent. In the current paper, I had two aims. First, I aimed to investigate whether the same childhood psychosocial adversity index predicts health deterioration across the potentially reproductive years. Second, I aimed to explore the extent to which health deterioration following childhood psychosocial adversity was a consequence of accelerated reproductive trajectories rather than a direct primary effect. I approached this aim through mediation analysis: to the extent that health deterioration is a consequence of accelerated reproduction, then the association between childhood psychosocial adversity and health deterioration will be mediated by markers of reproductive strategy such as age at first pregnancy.

To measure health deterioration, I considered two outcome measures. The first was self-rated health. This was measured at ages 23, 33 and 42 with the question 'How is your health in general?' Self-rated health is a widely used measure in epidemiological studies, and the single item is considered methodologically adequate (DeSalvo, Bloser, Reynolds, He, & Muntner, 2006). Despite being extremely quick and simple to collect, it shows a significant correspondence with more objective measures of health status (Christian et al., 2011), and prospectively predicts survival (Benyamini & Idler, 1999; DeSalvo et al., 2006; Idler & Benyamini, 1997). It is thought to relate most strongly to physical, rather than psychological, morbidity (Cabrero-García & Juliá-Sanchis, 2014). The second measure, available in 3836 of the women at age 44–46, was blood level of *c*-reactive protein. *C*-reactive protein is a widely-used non-specific blood marker of inflammation (Pepys & Hirschfield, 2003). Increased inflammatory activity has been proposed as a general marker of the somatic damage caused by social and environmental stressors, particularly in childhood (Miller, Chen, & Parker, 2011). *C*-reactive protein levels prospectively predict a number of adverse outcomes such as cardiovascular disease (Danesh et al., 2004) and diabetes (Pradhan, Manson, Rifai, Buring, & Ridker, 2001), as well as consequent mortality (Kuller, Tracy, Shaten, & Meilahn, 1996; Wang et al., 2006). They correlate with self-rated health in women (Tanno et al., 2012). *C*-reactive protein levels also tend to increase with age (Hutchinson et al., 2000), making them in effect a marker of age-related increase in morbidity and tissue damage. A number of previous studies have linked childhood psychosocial adversity with increased *c*-reactive protein levels in adolescence or adulthood (Danese et al., 2009; Danese et al., 2008; Slopen et al., 2010; Taylor, Lehman, Kiefe, & Seeman, 2006), though a null finding in a small sample has also been reported (Carpenter, Gawuga, Tyrka, & Price, 2012). In the full NCDS cohort, Lacey, Kumari, and McMunn (2013) have shown that *c*-reactive protein levels at age 44–46 were elevated in individuals whose parents separated during their childhoods, or who had low-quality relationships with their parents. However, Lacey et al.'s analysis does not employ the index of psychosocial adversity used here, and so I present *c*-reactive protein data in this paper as an analysis which is complementary to theirs.

To control for health-affecting variation in the adult environment, and thus isolate the specific impact of childhood experience, I used a composite measure of socioeconomic position over ages 23–42 as a control variable. Socioeconomic position in developed economies is the overwhelming single predictor of morbidity and mortality risks (Lantz et al., 1998; Marmot, Kogevinas, & Elston, 1987; Smith & Egger, 1993) and thus is the obvious candidate for a summary variable for the environmental sources of such risks that an adult is exposed to. I also examined the effects of controlling for smoking and body mass index. Adult smoking and body mass are both increased by childhood psychosocial adversity (Anda et al., 1999; Gunstad et al., 2006), and both bad for health. Thus, these variables could thus produce associations between childhood psychosocial adversity and later health that are the consequence of individuals' behavioural responses to adversity rather than direct primary effects.

2. Methods

2.1. The National Child Development Study (NCDS)

The NCDS, also known as the 1958 British birth cohort, is an ongoing longitudinal study of all people born in the UK between 3rd and 9th of March 1958 (Power & Elliott, 2006). I considered only the female cohort members (potential n = 8959) here. The current analyses involved linking by cohort member ID data that were collected perinatally in 1958; in an interview with a parent in 1965; in interviews with the cohort member in 1981, 1991 and 2000; and in the 2002–2004 biomedical survey, during which blood samples were taken from a subset of cohort members. There has been substantial sample attrition over this period, but this is not strongly patterned by level of childhood psychosocial adversity (Nettle et al., 2011). Interested researchers may apply to the UK Data Archive (www.data-archive.ac.uk) for access to the data. The biomedical survey data are available only under special licence, granted to DN under the project 'Early-life adversity, child development and adult health'.

2.2. Index of childhood psychosocial adversity

The index of childhood psychosocial adversity is as described in Nettle et al. (2011), and refers to experiences occurring within the first seven years of life (for brevity, I refer to this variable as 'childhood adversity' hereafter). The four component variables were separation from mother, paternal involvement, residential relocations, and duration of breast-feeding. In each case, the component variable was dichotomised, with 1 representing the more adverse situation and 0 the less. The childhood adversity variable is the sum of the four components, and thus varies from 0 (none of the adversities) to 4 (all of them; see Supporting Information, available on the journal's website at www.ehbonline.org Section 1 for descriptive statistics for all variables along with details of the names of the original NCDS variables from which they are derived).

2.3. Health outcomes

Self-rated health was asked using a single item at the interviews of 1981, 1991 and 2000 (ages 23, 33 and 42). Responses were on a four-point scale (poor/fair/good/excellent). Given the large sample size and ordered structure of the responses, I treated the scale here as continuous, scoring poor as 1 and excellent as 4. This appears justified by the homogeneity of variance and approximately normal distribution of the residuals from all models (see Supporting Information, available on the journal's website at www.ehbonline.org, Section 3). *C*-reactive protein levels were measured by nephelometry in citrated blood plasma using latex particles coated with CRP-monoclonal antibodies (Elliot, Johnson, & Shepherd, 2008). Resulting values were right-skewed and were logarithmically transformed. Unlike Lacey et al. (2013), I did not exclude values greater than 10 mg/L (under 4% of cases). These are likely to reflect viral infection or other acute inflammation. However, I reasoned that such health problems represent part of the phenomenon under study and as such should not be excluded.

2.4. Adult socio-economic position

My index of adult socioeconomic position is the first factor arising from a principal components analysis of social class at ages 23, 33 and 42, educational qualifications at ages 23 and 33, and income at age 42 (see Supporting Information, available on the journal's Website at www.ehbonline.org Section 2 for more detail and alternative measures). The factor accounts for 49.12% of the variation in its components, and all components load substantially on it (loadings 0.57–0.89). Adult socioeconomic position was significantly but only very weakly associated with childhood adversity ($r = -0.05$, $p < 0.05$).

2.5. Age at first pregnancy

Age at first pregnancy in years was taken from participant report at age 33 as described in Nettle et al. (2011). Women who had not been pregnant at this time (%) were assigned the value of 33 since they had not been early reproducers.

2.6. Smoking and body mass index (BMI)

Smoking was measured by participant report at age 42, as a categorical variable of never smoked/former or occasional smoker/current smoker. BMI was computed from height and weight measurements of cohort members at age 23.

2.7. Statistical methods

All analyses were performed in R (R Core Developent Team, 2013) using packages stats, nlme, psych and ggplot2. When analysing repeated self-rated health measurements across time, I used linear mixed models with random intercepts (accounting for different average levels of health across individuals) and random slopes (accounting for different trajectories of health over time across individuals) for cohort member. Parameter estimation was by maximum likelihood (R package nlme). This approach allows all available data to be used from participants who completed the measure at some time points but not others. When interrogating self-rated health at one particular age, and for the analysis of *c*-reactive protein, I used simple linear models. Adult socioeconomic position was controlled in all models; specifications of other models were as given in the Results section. Effective sample sizes became smaller with increasing model complexity due to missing and invalid values. A criterion of $p = 0.05$ was used for statistical significance throughout, though exact p-values are reported below.

3. Results

3.1. Childhood adversity and self-rated health

Fig. 1 plots mean health by age for the different levels of childhood adversity. As the figure shows, women with higher childhood adversity had, on average, poorer self-rated at all ages, but the discrepancy was greater at the older ages than at the younger ones. In particular, the two-adversity group (1112 women) and three-adversity group (345 women) had similar health to the one-adversity group at age 23, but substantially poorer self-rated health at age 33, a pattern which persisted at age 42. The large standard error in the four-adversity group is due to small sample sizes for this category compared to the other categories (30 women). To test for an association between childhood adversity and self-rated over time, I fitted a linear mixed model with age, adult socioeconomic position, childhood adversity and the interaction between age and childhood adversity as predictor variables. As Table 1 shows, there were significant effects of age (poorer health at older ages) and adult socioeconomic position (poorer health with lower socioeconomic position). The main effect of childhood adversity was not significant. However, there was a significant interaction between age and childhood adversity. To understand this interaction, I fitted separate simple linear models for self-rated health at each of the three ages with adult socioeconomic position and childhood adversity as predictors. At age 23, childhood adversity was marginally non-significantly associated with self-rated health ($B = -0.021$, s.e.(B) = 0.011, $t = 1.90$, $p = 0.06$); at age 33 it was significantly associated with self-rated health ($B = -0.034$, s.e.(B) = 0.012, $t = 2.925$, $p = 0.003$); and at age 42, the association

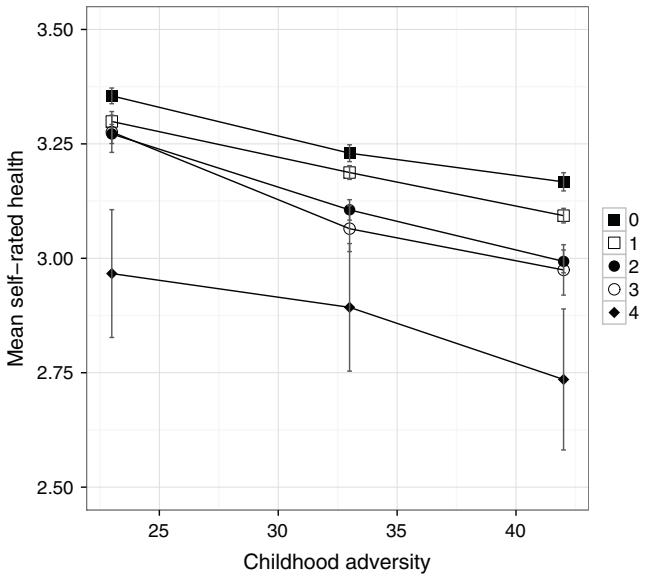

Fig. 1. Mean self-rated health at ages 23, 33 and 42 for women with different scores on the childhood psychosocial adversity index. Error bars represent one standard error.

was even stronger (B = −0.053, s.e.(B) = 0.013, t = 4.211, p < 0.001). The parameter estimates for childhood adversity thus became more negative with increasing age (−0.021, −0.034 and −0.053 respectively), bearing out the pattern apparent from Fig. 1 of childhood adversity predicting the deterioration of health across time more strongly than the level of health at age 23.

3.2. Age at first pregnancy as a mediator of the relationship between childhood adversity and self-rated health

To investigate whether the relationship between childhood adversity and deteriorating self-rated health was mediated by accelerated reproduction, I used path analysis to examine the relationships amongst childhood adversity, age at first pregnancy, and health at age 42 (Fig. 2; all relationships are controlled for adult SEP). In Fig. 2, the left-hand diagram shows the overall association between childhood adversity and self-rated health at 42, whilst the right-hand diagram shows the extent to which the effect was mediated by earlier age at first pregnancy. Although childhood adversity was significantly associated with earlier first pregnancy, and earlier first pregnancy was significantly associated with poorer health at age 42, the effect of childhood adversity and health at 42 controlling for age at first pregnancy was still significant. Indeed, the pathway from childhood adversity to health at 42 via earlier age at first pregnancy only accounted for 10.7% of the association between childhood adversity and health at 42, reducing the parameter estimate for the direct relationship from −0.053 to −0.047.

Table 1
Parameter estimates from a model predicting self-rated health across adulthood from age, adult socioeconomic position, childhood adversity, and the interaction between age and childhood adversity. There are 14997 observations from 6034 individuals. The model includes random intercepts and slopes for cohort member.

Variable	B	s.e.	t	p
Age	−0.010	0.001	9.931	<0.001
Adult socioeconomic position	0.130	0.007	18.306	<0.001
Childhood adversity	0.026	0.024	1.087	0.277
Age*Childhood adversity	−0.002	0.001	2.644	0.008

3.3. Smoking and BMI as mediators of the relationship between childhood adversity and self-rated health

To investigate whether smoking and BMI mediated the effect of childhood adversity on self-rated health, I added smoking status at age 42 and BMI at age 23 to a model predicting self-rated health at 42 from adult socioeconomic position, childhood adversity, and age at first pregnancy. Regular smoking significantly reduced self-rated health compared to never smoking, though the difference between former or occasional smoking and never smoked was not significant (former or occasional vs. never: B = −0.034, s.e.(B) = 0.028, t = 1.220, p = 0.223; regular vs. never: B = −0.265, s.e.(B) = 0.031, t = 8.561, p < 0.001). BMI was also negatively associated with self-rated health (B = −0.019, s.e.(B) = 0.004, t = 5.077, p < 0.001). However, the effects of childhood adversity (B = −0.034, s.e.(B) = 0.014, t = 2.436, p = 0.015), adult socioeconomic position (B = 0.083, s.e.(B) = 0.012, t = 7.063, p < 0.001), and age at first pregnancy (B = 0.009, s.e.(B) = 0.002, t = 3.778, p < 0.001) all remained significant. The effect of childhood adversity was attenuated by 35.2% in this adjusted model compared to the model including neither age at first pregnancy nor smoking.

3.4. C-reactive protein

C-reactive protein was significantly negatively correlated with self-rated health at all age three points, most strongly at age 42, which was the closest rating in time to the blood sample from which the *c*-reactive protein measurement was derived (r = −0.20, p < 0.05). To test for an association between childhood adversity and *c*-reactive protein, I ran a model predicting *c*-reactive protein level from adult socioeconomic position and childhood adversity. The effects of adult socioeconomic position (B = −0.206, s.e.(B) = 0.021, t = 10.026, p < 0.001) and of childhood adversity (B = 0.082, s.e.(B) = 0.026, t = 3.163, p = 0.002) were both significant. Fig. 3 shows how mean *c*-reactive protein levels vary with the level of childhood adversity. Age at first pregnancy was not significantly related to *c*-reactive protein level (B = −0.006, s.e.(B) = 0.005, t = 1.259, p = 0.208), and thus did play any role in mediating the relationship between childhood adversity and *c*-reactive protein (Fig. 4). I also added smoking and BMI to the model. The effects of smoking were not significant (former or occasional vs. never: B = −0.012, s.e.(B) = 0.053, t = 0.221, p = 0.825; regular vs. never: B = 0.062, s.e.(B) = 0.060, t = 1.029, p = 0.303), whilst the effect of BMI was highly significant and positive (B = 0.123, s.e.(B) = 0.007, t = 16.596, p < 0.001). Even with BMI in the model, the effect of childhood adversity on *c*-reactive protein remained significant (B = 0.064, s.e.(B) = 0.027, t = 2.418, p = 0.012), with the strength of the effect 65.9% of that in the model without BMI.

4. Discussion

I found evidence that childhood psychosocial adversity, as measured by the simple index created in our previous study, was associated with poorer health by the time the cohort members were in their forties. For self-rated health, the association was specifically with the change in health across adulthood, rather than the mean in young adulthood. The average self-rated health of women who had experienced a moderately high degree of adversity (scores of two or three) diverged markedly from the average self-rated health of those who with scores of zero or one after the age of 23 but before the age of 33. The group who had experienced very high adversity (score of four) appeared to have poor health at all ages, but their numbers were very small. For *c*-reactive protein at age 44–46, there was a significant and dose-dependent relationship with the childhood adversity index, more adversity being associated with higher *c*-reactive protein levels. The associations did not appear to be due to women who had

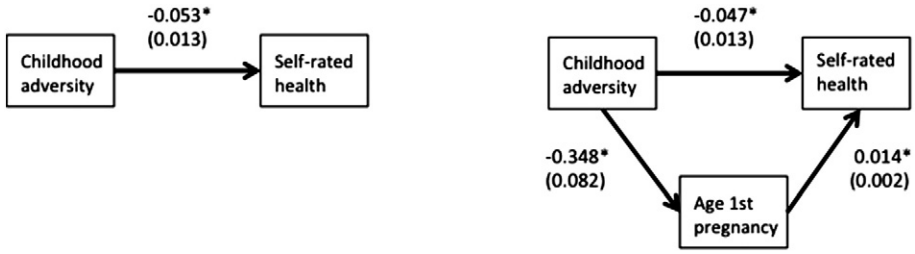

Fig. 2. Path diagrams for the association between childhood adversity and self-rated health at age 42. The left-hand diagram shows the overall association, whilst the right-hand diagram decomposes the association into the part mediated by age at first pregnancy and the part that is not. Numbers on arrows represent B coefficients (standard errors). *$p < 0.05$.

experienced greater childhood adversity living in poorer adult environments (as proxied by adult socioeconomic position), since the correlation between childhood adversity and adult socioeconomic position in the sample was extremely low, and the addition of adult socioeconomic position to the statistical models did not substantially attenuate the effects.

The results are consistent with large number of previous findings that there appear to be long-term health costs associated with experiencing childhood psychosocial adversity, above and beyond the effects of known health predictors such as smoking and socioeconomic position that may covary with childhood psychosocial adversity (Dube et al., 2003; Felitti et al., 1998; Larson & Halfon, 2013). For *c*-reactive protein in particular, a number of studies have found elevated adult levels in the blood of those who experienced adversity in childhood (Danese et al., 2009; Danese et al., 2008; Slopen et al., 2010; Taylor et al., 2006). I replicated the findings of Lacey et al. (2013) in the same cohort, albeit that they studied cohort members of both sexes and used parental separation as their main childhood measure, rather than the particular childhood adversity index I used here. The results also concur with previous studies in finding BMI and socioeconomic position to be important predictors of *c*-reactive protein levels (Kivimäki et al., 2005; Visser, Bouter, McQuillan, Wener, & Harris, 1999), and BMI, socioeconomic position and smoking to be important predictors of self-rated health (Christian, Iams, Porter, & Leblebicioglu, 2013).

Adaptive explanations for reproductive acceleration following childhood adversity require that there has been, over evolutionary time, an increment of adult mortality/morbidity risk in individuals who experience childhood adversity, regardless of whether they adopt a fast reproductive schedule or not. However, as discussed in the Introduction, there is also likely to be an increment in mortality/morbidity that is a consequence of following a fast reproductive schedule. I used mediation analysis to try to assess whether both of these increments were present in this cohort and estimate their relative magnitudes. I found that for self-rated health (though not *c*-reactive protein), starting to reproduce earlier was indeed costly: women who had a younger age at first pregnancy had poorer self-rated health at 42. Women who experienced more childhood adversity also had a younger age at first pregnancy. However, younger age at first pregnancy was not a substantial mediator of the childhood adversity–poorer health relationship, attenuating the coefficient by less than 11%. Nor were the relationships between childhood adversity and the health outcomes explained away by increased smoking or body mass amongst women who experienced childhood adversity. Thus, it seems reasonable to argue that the data support the assumption, critical to the adaptive evolutionary account, that childhood psychosocial adversity has a negative impact on future prospects for health and survival, regardless of how the individual subsequently chooses to live. This conclusion should be viewed as tentative, since the only measure of reproductive trajectory that I included was age at first pregnancy: a fuller study would adjust for number of children, inter-birth interval, and other aspects of reproductive career as well, and this might increase the estimate of how much of the childhood adversity-health relationship is a consequence of reproductive decisions.

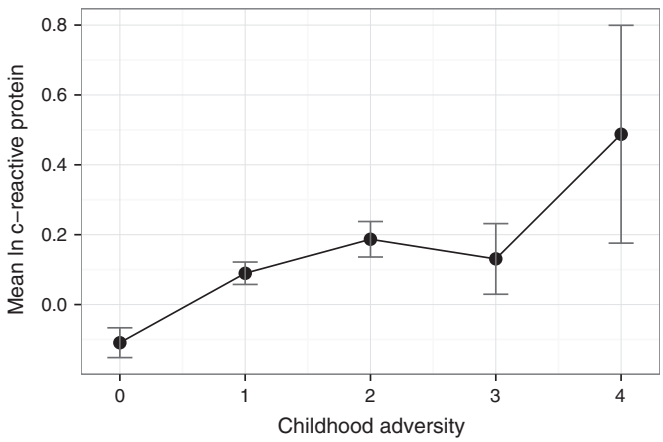

Fig. 3. Means of the logarithm of blood *c*-reactive protein levels at age 44–46 for women with different scores on the childhood psychosocial adversity index. Error bars represent one standard error.

The study had a number of other limitations. The index of childhood adversity is extremely crude, and doubtlessly fails to capture all the relevant dimensions and constituents. The main justifications for using it here are that it incorporates some of the main adversities found to be important in previous literature, and that in our earlier study, it was a good predictor of age at first pregnancy. The fact that I found associations with health using such a limited index makes it plausible that the true associations between childhood psychosocial adversity and adult health are stronger. The health outcome measures were also limited, and give no information about the sources of poor health. However, both self-rated health and *c*-reactive protein are widely used as general health markers, with substantial prospective validity for future survival (DeSalvo et al., 2006; Idler & Benyamini, 1997; Wang et al., 2006). Finding significant associations with childhood adversity is thus suggestive, and further research is required to drill down into which health problems in particular are involved. Another limitation is that I incorporated no information about mortality over the time period studied; women who died simply became missing in the dataset I used. However, Kelly-Irving et al. (2013) studied premature mortality in this cohort by linking to death registrations. They found that, although premature mortality is rare, adverse childhood experiences (a construct very similar to childhood adversities as used here) predicted excess mortality risk even controlling for socioeconomic position. Thus, it is likely that using mortality rather than poor health as the adult outcome would lead to the same conclusion: childhood psychosocial adversity has a negative impact. The current study also gives no

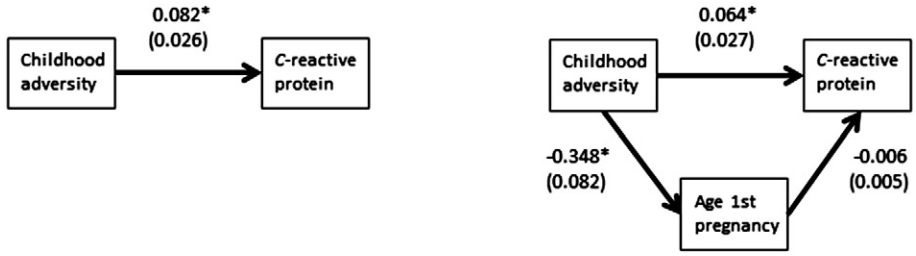

Fig. 4. Path diagrams for the association between childhood adversity and logged *c*-reactive protein levels at age 44–6. The left-hand diagram shows the overall association, whilst the right-hand diagram represents the extent to which the association is mediated by age at first pregnancy. Numbers on arrows represent B coefficients (standard errors). *$p < 0.05$.

information about the physiological mechanisms by which childhood psychosocial adversity becomes embedded in the soma. Candidate mechanisms include oxidative damage and telomere loss (Geronimus et al., 2010; Kananen et al., 2010; Shalev et al., 2013; Tyrka et al., 2010), but these have not yet been measured in the NCDS cohort.

A further limitation is the use of a developed-world population to test the assumptions of an evolutionary theory, assumptions that the theory strictly requires to hold across ancestral populations, not necessarily contemporary ones. In a sense, this limitation would be more acute had I found no evidence that childhood psychosocial adversity is associated with health deterioration in adulthood, since I could not exclude that there might have been such a nexus under ancestral conditions. As it was, the evidence fit with the assumptions of the theory, suggesting (though not of course demonstrating) that this was a nexus that existed in the past and continues to exist today. It is imperative to replicate this kind of study in human populations living under very different environmental conditions, but the evidence presented here is an important first step. If early-life adversity causing somatic damage that later leads to accelerated health deterioration is a rather general biological principle, as evidence from other taxa suggests that it may be (Boonekamp, Mulder, Salomons, Dijkstra, & Verhulst, 2014), then we might expect associations between childhood psychosocial adversity and adult health across all kinds of human populations, albeit that the sources of the adversity might be very different in different socio-ecologies.

The limitations of the study duly noted, the results support the view that childhood psychosocial adversity may lead to accelerated health deterioration across adulthood, that this is not solely a consequence of reproductive strategies, and hence that accelerated health deterioration could constitute a simple and general reason for women to accelerate their reproductive schedules following childhood psychosocial adversity. In the current results, an average woman who had experienced two childhood adversities had only slightly worse health at 23 than an average woman who had experienced one adversity. At 33, though, her health was worse than the one-adversity woman's would be at 42. Assuming, as seems not unreasonable, that organisms have evolved means of responding dynamically to their own somatic state (Rickard et al., 2014), it is not surprising that the women experiencing two or more adversities began their childbearing careers earlier than those who had experienced none or one. Evidence consistent with this explanation for reproductive acceleration have been presented elsewhere (Geronimus, 1996a, 1996b; Geronimus, Bound, & Waidmann, 1999), but these findings are particularly suggestive, since the same adversity index that is known to predict age at first pregnancy in this cohort was here found to predict subsequent health.

Viewing reproductive acceleration in the context of accelerated health deterioration leads to the insight that the reproductive schedules of high-adversity women may not, in one sense, be accelerated at all. It may be aging itself—the decline in biological performance over time—that is accelerated by childhood adversity. If aging is caused by the gradual accretion of somatic damage, then biological age is not the same thing as chronological age; an individual facing more stress will age faster (Geronimus et al., 2010). There is no reason that life-history milestones such as the age at maturation or first reproduction should be tied to chronological age. In fact, theoretical models predict that they should be triggered not by time but by changes in biological state (McNamara & Houston, 1996), such as reaching a threshold level of somatic damage. On this view, women who live through childhood psychosocial adversity might reach maturation and childbearing at exactly the same biological age as any other women; they just do it in fewer months, because they are aging at a higher rate.

Supplementary Material

Supplementary data to this article can be found online at http://dx.doi.org/10.1016/j.evolhumbehav.2014.07.002.

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