

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, we revisit a large, longitudinally studied cohort of British women. In an earlier study, 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, we show that the same index of adversity predicts accelerated deterioration of health across the potentially reproductive period, and increased levels of the inflammatory biomarker *c*-reactive protein at age 44. These associations are robust to controlling for adult socioeconomic position, and do not appear to be a consequence of accelerated reproductive strategy, smoking, or BMI. We argue that childhood psychosocial adversity may lead to the lasting embedding of somatic damage that accelerates age-related physical decline. This provides a compelling adaptive rationale for the accelerated reproductive schedules observed in women who experience childhood psychosocial adversity.

Keywords: psychosocial adversity, psychosocial acceleration, weathering hypothesis, biological embedding, *c*-reactive protein, health, life history, humans

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 (henceforth, CPA) 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, & Dickens, 2011; Pesonen et al., 2008; Tither & Ellis, 2008). This phenomenon – termed psychosocial acceleration (Ellis, 2004) – has been given an adaptive evolutionary interpretation (Belsky et al., 1991; Chisholm, 1993; Ellis, Figueredo, & Schlomer, 2009). In general, as the extrinsic risk of becoming unable to reproduce (for example, 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 life-history strategy becomes ‘faster’ (Charnov, 1991; Cichon, 1997; Promislow & Harvey, 1990). Thus, for psychosocial acceleration to be an adaptive strategy, CPA 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, 2010, 2011; Placek & Quinlan, 2012). However, the adaptive interpretation of psychosocial acceleration critically depends on CPA being associated with subsequent morbidity or mortality risk in adulthood at the individual level. This assumption has never been directly tested in the adaptationist literature on psychosocial acceleration.

Exactly *why* CPA would be associated with increased morbidity or mortality risk has been the subject of debate in the recent literature (see Belsky, 2014; Nettle, Frankenhuys, & Rickard, 2013; Rickard, Frankenhuys, & Nettle, 2014). Rickard, Frankenhuys and Nettle (2014) have argued for the importance of somatic damage caused by CPA that accelerates processes of age-related health deterioration. This idea builds on a number of earlier sources, and is inspired in particular by the ‘weathering hypothesis’ (Geronimus, 1992; A. T. Geronimus, 1996; A.T. Geronimus, 1996; 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*, 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.

To test the assumptions of the weathering-based account, it would not be sufficient to demonstrate an association between CPA and accelerated health deterioration in adulthood. Accelerated health deterioration could be a consequence of the individual following a fast life-history strategy entrained by early-life adversity (Ellis & Del Giudice, 2013). A fast life-history strategy entails reproductive effort early in life, and possibly other risky behaviours, and thus may lead to a reduction in safety and self-maintenance through adulthood. The weathering-based account requires there to be accelerated health deterioration that is not a consequence of the individual's life-history strategy, but which occurs regardless of what strategy they follow. It is this *extrinsic* health deterioration as a consequence of CPA on which the weathering-based adaptive explanation for CPA depends.

There are a large number of epidemiological papers showing that various types of CPA 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). However, this literature has not been integrated with the adaptationist literature on psychosocial acceleration. Specifically, it has not yet been demonstrated for any cohort that the same psychosocial factors that are linked to accelerated reproductive strategy are also linked to accelerated health deterioration with age. In this paper, we return to a large, longitudinally-studied cohort of British women (the NCDS cohort) who were the subject of an earlier study (Nettle et al., 2011). In that study, 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, we investigate how the same childhood psychosocial adversity index predicts health across the potentially reproductive years in these women.

We consider two outcome measures. The first is self-reported health. This was measured at ages 23, 33 and 42 with the single item 'How is your health in general?'. Self-reported health is a widely used measure in epidemiological studies, and the single-item measure 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, is blood levels 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 CPA 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 are 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 we present c-reactive protein data in this paper as an analysis which is complementary to theirs.

Under the assumptions of the weathering hypothesis, we predict that higher scores on the adversity index will relate to poorer self-rated health, and in particular a greater deterioration of self-rated health over the three measurement ages, and to higher levels of c-reactive protein at age 42. We also predict that these associations will be robust to controlling for the adult environment and proxies of the reproductive and behavioural strategies that the individual has followed.

To characterise the adult environment, we use a composite measure of socioeconomic position over ages 23-42. 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. To investigate whether effects of childhood adversity on health are the consequence of life-history decisions rather than their cause, we control for age at first pregnancy, smoking, and body mass index (BMI). The first of these is a direct measure of reproductive strategy. The second and third may capture some of the behavioural variation in self-care that is relevant to health and may stem from following a fast life-history strategy.

Methods

Ethics statement

This paper reports only secondary analysis of existing anonymised datasets. Full written consent for participation and data use was provided by all participants and their families at the times of original data gathering. 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'.

The National Child Development Study

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 March 1958 (Power & Elliott, 2006). We consider only the female cohort members (potential n=8959) here. The current analyses involve 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 CPA (Nettle et al., 2011, Supporting Information).

Measures

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

Health outcomes. Self-reported health (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, we have 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, section 3). C-reactive protein levels (CRP) 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 ($\ln\text{CRP}$). Unlike Lacey et al. (2013), we did not exclude values greater than 10mg/L (under 4% of cases). These are likely to reflect viral infection or other acute inflammation. However, we reason that such health problems represent part of the phenomenon under study and as such should not be excluded.

Adult socio-economic position. Our index of adult socioeconomic position (SEP) 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 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).

Age at first pregnancy. Age at first pregnancy in years (AFP) 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.

Smoking and body BMI. Smoking (SMOKE) was measured by participant report at age 42, as a categorical variable of never smoked / former or occasional smoker / current smoker. BMI (weight in kgs divided by height in metres squared) was taken by measurement of height and weight at age 23.

Statistical methods

All analyses were performed in R (R Core Development Team, 2013) using packages stats, nlme, psych and ggplot2. The HEALTH data involves repeated measures for the same participant, and thus we used a linear mixed model with a random effect of participant, using maximum likelihood estimation (package nlme). This allows all available data to be used from participants who completed the

measure at some time points but not others. For InCRP, we used a simple linear model (package stats). The analysis strategy in each case was to begin with a simple model (model 1) with just CPA (plus age and the interaction between CPA and age in the case of HEALTH) as a predictor. Then in a sequence of increasingly adjusted models, we added SEP (model 2), AFP (model 3), then SMOKE and BMI (model 4) as predictors. At each stage only significant predictors ($p < 0.05$) from the step before were retained in the model. Effective sample sizes became smaller with increasing model complexity due to missing and invalid values.

Results

Self-reported health.

Table 1 summarises the models predicting HEALTH from age, CPA and the other covariates. In the unadjusted model 1, there was a significant effect of age, with average health becoming worse at the later ages. There was no main effect of CPA on adult health, but there was a significant interaction between CPA and age. Figure 1 plots mean health by age for the different levels of CPA. As the figure shows, women with higher CPA scores tended to have poorer HEALTH at all ages, but the discrepancy was greater at the later ages. In particular, the two-adversity group (1112 women) and three-adversity group (345 women) had similar health to the one-adversity group at 23, but substantially poorer health at ages 33, a pattern which persisted at 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).

Models 2-4 sequentially add in further covariates. Model 2 added adult SEP, which itself had a significant positive effect on health. Adult SEP was significantly but only very weakly associated with CPA ($r_{4710} = -0.05$, $p < 0.05$), and its addition to the model did not substantially attenuate the strength of the interaction between age and CPA. Earlier age at first pregnancy was significantly associated with poorer health, but its addition (model 3) only slightly attenuated the interaction between age and CPA. The other covariates had significant effects in expected directions (smoking associated with poorer health; higher BMI with poorer health). However, even in the fully adjusted model 4, the interaction of age with CPA remained a significant predictor of health.

C-reactive protein

InCRP was significantly negatively correlated with HEALTH at all age three points, most strongly with HEALTH at 42, which was the closest rating in time to the blood sample from which InCRP is derived ($r_{3721} = -0.20$, $p < 0.05$).

Table 2 summarises the models predicting InCRP. In model 1, there was a significant effect of CPA on InCRP, with greater CPA scores associated with higher InCRP (see figure 2). SEP was a significant inverse predictor of InCRP. Including SEP attenuated the effect of CPA by approximately one third. The only other significant predictor of InCRP was BMI, with larger BMIs associated with higher InCRP. When both SEP and BMI were retained in the model (model 5), the effect of CPA on InCRP was attenuated by almost a half compared to model 1, but nonetheless remained significant.

Table 1. Parameter estimates (standard errors) from models predicting self-reported health across adulthood. The 'n' row refers to number of valid observations rather than number of participants (each participant contributes up to 3 observations at different time points). The '% of model 1 effect' row captures as a percentage the degree to which the magnitude of the Age*CPA parameter is attenuated by the addition of covariates.

Model	1	2	3	4
Age	-0.0098* (0.00093)	-0.0098* (0.00093)	-0.010* (0.00099)	-0.010* (0.0010)
CPA	0.0010 (0.024)	0.025 (0.023)	0.017 (0.026)	0.028 (0.027)
Age*CPA	-0.0019* (0.00069)	-0.0019* (0.00069)	-0.0016* (0.00074)	-0.0017* (0.00078)
SEP		0.13* (0.0072)	0.11* (0.0077)	0.075* (0.0084)
AFP			0.0095* (0.0016)	0.0075* (0.0018)
SMOKE Former/occ.				-0.054* (0.020)
SMOKE Regular				-0.21* (0.022)
BMI				-0.016* (0.0026)
n	14997	14997	13424	11335
% of model 1 effect	100	99	84	86

* p<0.05

Table 2. Parameter estimates (standard errors) from models predicting lnCRP. The 'n' row refers to number of participants in the model. The '% of model 1 effect' row captures as a percentage the degree to which the magnitude of the CPA parameter is attenuated by the addition of covariates.

Model	1	2	3	4	5
CPA	0.12* (0.026)	0.082* (0.026)	0.064* (0.027)	0.062* (0.027)	0.064* (0.027)
SEP		-0.21* (0.021)	-0.20* (0.023)	-0.13* (0.022)	-0.14* (0.021)
AFP			-0.0060 (0.0048)		
SMOKE Former/occ.				-0.012 (0.053)	
SMOKE Regular				-0.062 (0.06)	
BMI				0.12* (0.0074)	0.12* (0.0074)
n	3260	3260	2954	2775	2860
% of model 1 effect	100	68	53	51	53

* p<0.05

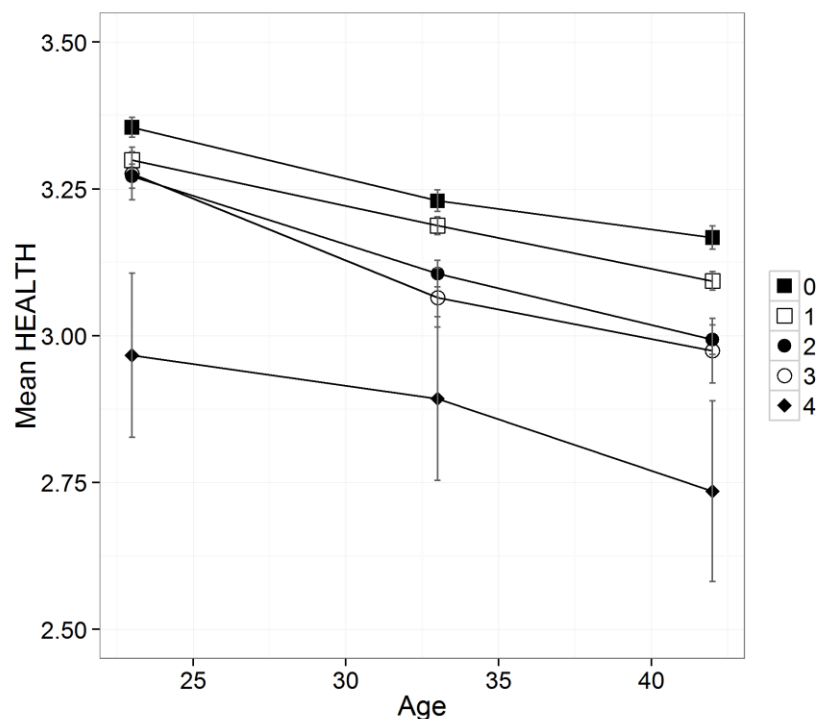


Figure 1. Mean self-rated health at ages 23, 33 and 42 for women with different scores on the childhood psychosocial adversity index. Error bars represent one standard error.

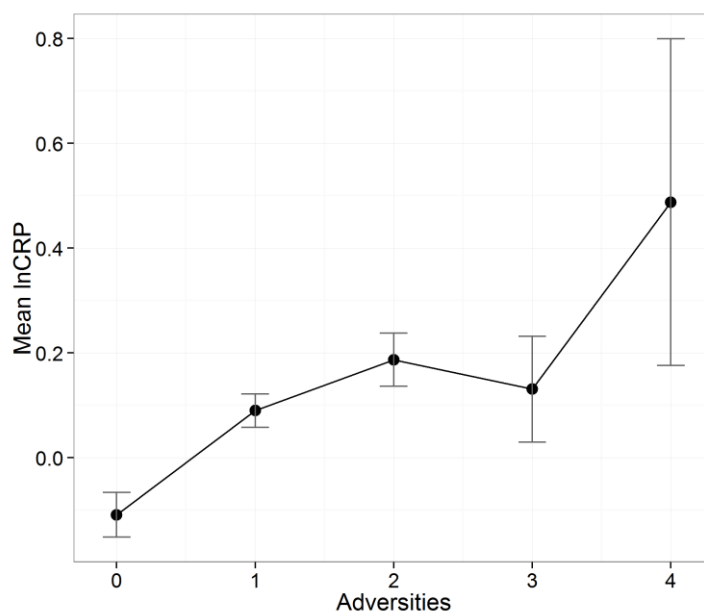


Figure 2. Means of the logarithm of blood c-reactive protein levels at age 44 for women with different scores on the childhood psychosocial adversity index. Error bars represent one standard error.

Discussion

We found evidence that CPA, as measured by the simple index created in our previous study, was associated with poorer health by age 42. For self-rated health, the association was specifically with the change in health across adulthood, rather than the mean. 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, there was a significant and dose-dependent relationship with the CPA index, more adversity being associated with higher c-reactive protein levels.

The associations did not appear to be due to women who had experienced greater CPA living in poorer adult environments (as proxied by adult socioeconomic position), since the correlation between CPA and adult socioeconomic position in the sample was extremely low, and the addition of adult SEP to the statistical models did not substantially attenuate the effects. It also seems unlikely that the associations were entirely a consequence of following a fast life-history, since controlling for age at first pregnancy, BMI and smoking did not abolish them, although it did slightly reduce their size. In the case of self-rated health, there was a significant effect of age at first pregnancy (earlier first pregnancy associated with poorer health). This could be interpreted in terms of the somatic costs of early reproductive effort. There were also effects of BMI on both self-rated health and c-reactive protein, and of smoking on self-rated health only. Nonetheless, even adjusting for these effects, the effects of CPA remained significant, with over 80% (self-rated health) or over 50% (c-reactive protein) of their original strength.

The results are consistent with large number of previous findings that there appear to be long-term health costs associated with experiencing CPA, above and beyond the effects of known health predictors such as smoking and socioeconomic position that may covary with CPA (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). We 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 index of CPA we used here. Our results also concur with previous studies in finding BMI and SEP to be important predictors of c-reactive protein levels (Kivimäki et al., 2005; Visser, Bouter, McQuillan, Wener, & Harris, 1999), and BMI, SEP and smoking to be predictors of self-rated health (Christian, Iams, Porter, & Leblebicioglu, 2013).

Our results directly address the critical assumption underlying the weathering-based explanation for psychosocial acceleration. That explanation depends on people who experience CPA developing into adults whose health will deteriorate relatively rapidly with age, regardless of what they do. This provides an adaptive rationale for their early reproduction. In our 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 the weathering-based explanation for psychosocial acceleration have been presented elsewhere (A. T. Geronimus, 1996; A.T. Geronimus, 1996; Geronimus, Bound, & Waidmann, 1999), but these findings are particularly suggestive, since the same CPA index that is known to predict age at first pregnancy in this cohort was here found to predict subsequent health, even controlling for proxies of life-history strategy.

The current study has a number of limitations. The index of CPA used is an extremely crude one, and doubtless fails to capture all the relevant dimensions and constituents of CPA. 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 we found weathering effects with such a crude and limited index makes it plausible that the true associations between CPA and adult health are stronger than those we found. Our 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 CPA is thus suggestive, and further research is required to drill down into which health problems in particular are made more probable in individuals who experience CPA. A further limitation is that we have no information about mortality over the time period studied. Women who die become missing from the NCDS data, and we are thus unable to test for associations between CPA and premature death. However, female mortality before the age of 42 is extremely rare in this population, and so it is unlikely that there would be sufficient statistical power to detect differential mortality by CPA. Since poor self-rated health and high c-reactive protein are statistically associated with subsequent mortality, we might infer that high-CPA women will go on to die younger than low-CPA women on average. The current study also gives no information about the physiological mechanisms by which CPA becomes biologically embedded in the soma. Candidate mechanisms include oxidative damage and telomere loss (Geronimus et al., 2010; Kananen et al., 2010; Rickard et al., 2014; Shalev et al., 2013; Tyrka et al., 2010), but these have not as yet been measured in the NCDS cohort.

These limitations noted, the current results do suggest that differential weathering may be a consequence of experiencing CPA, and that this is a promising and general adaptive explanation for why women who experience CPA tend to accelerate their reproductive schedules. Indeed, considering psychosocial acceleration in the light of the weathering hypothesis leads to the possibility that the reproductive schedules of high-CPA women may not be specifically accelerated at all. It may be aging itself – the decline in biological performance over time – that is accelerated. The reproduction of high-CPA women might be occurring at the same biological age as that of their low-CPA peers, but they reach milestones of biological aging sooner due to the stressful experiences they undergo.

As well as early reproduction, CPA has been associated with many non-reproductive outcomes, such as increased psychopathology (Heim & Nemeroff, 2001), shorter time perspective (Chisholm, 1999), and less cooperative social attitudes (McCullough, Pedersen, Schroder, Tabak, & Carver, 2013). It would be interesting to investigate to what extent differential health weathering might also provide an adaptive rationale for these changes. For example, a consistent finding is increased anxiety in people who experienced CPA (Heim & Nemeroff, 2001). Anxiety is also increased in people who are physically infirm (Scott et al., 2007). Bateson, Brilot and Nettle (2011) argue that is adaptive to up-regulate threat detection mechanisms when physical ability to deal with undetected threats is poor. Thus, poorer somatic state consequent on CPA might provide an adaptive rationale for greater anxiety. Poor somatic weathering might possibly be at the centre of the suite of psychological and behavioural phenomena that have been found to follow from experiencing psychosocial adversity as a child.

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