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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.