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Social isolation in childhood and adult inflammation: Evidence from the National Child Development Study

Rebecca E. Lacey[a,*], Meena Kumari[a,b], Mel Bartley[a]

[a] Department of Epidemiology and Public Health, University College London, London WC1E 6BT, United Kingdom
[b] Institute of Social & Economic Research, University of Essex, Colchester CO4 3SQ, United Kingdom

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Summary
Background: Social isolation is known to be associated with poorer health amongst adults, including coronary heart disease. It is hypothesized that this association may be mediated by inflammation. There has been little prospective research on the long-term impact of social isolation in childhood on adult health or the pathways which might be involved. The aim of this study was to investigate whether social isolation in childhood is associated with increased adult inflammation and the mechanisms involved across the life course.
Methods: This study used multiply-imputed data on 7462 participants of the National Child Development Study in Great Britain. The association between child social isolation (7—11 yrs) and levels of C-reactive protein (CRP) in middle age (44 yrs) was examined. We additionally investigated the role of adult social isolation, psychological distress, health behaviors and socioeconomic factors as potential mediators using path analysis and concurrent measurements made across the life course.
Results: Socially isolated children had higher levels of C-reactive protein in mid-life (standardized coefficient = 0.05, $p \leq 0.001$). In addition, children who were socially isolated tended to have lower subsequent educational attainment, be in a less advantaged social class in adulthood, were more likely to be psychologically distressed across adulthood and were more likely to be obese and to smoke. All of these factors partially explained the association between childhood social isolation and CRP. However, this association remained statistically significant after considering all mediators simultaneously.

* Corresponding author at: Department of Epidemiology and Public Health, University College London, 1-19 Torrington Place, London WC1E 6BT, United Kingdom. Tel.: +44 0207 679 1676; fax: +44 0203 108 3354.
E-mail address: Rebecca.lacey@ucl.ac.uk (R.E. Lacey).

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Conclusions: Social isolation in childhood is associated with higher levels of C-reactive protein in mid-life. This is explained in part through complex mechanisms acting across the life course. Identification and interventions targeted toward socially isolated children may help reduce long-term adult health risk.

1. Introduction

Chronic low-grade inflammation is known to be associated with adverse health outcomes, such as type II diabetes (Bassuk et al., 2004), depression (Danner et al., 2003), and coronary heart disease (Danesh et al., 2004). Many studies have shown associations between childhood adversities and raised C-reactive protein (CRP) levels — a reliable marker of low-grade inflammation (Pepys and Hirschfield, 2003). Childhood adversities known to be associated with increased adult inflammation include socioeconomic disadvantage (Phillips et al., 2009; Carroll et al., 2011; Miller and Cole, 2012), abuse and neglect (Danese et al., 2007), parental divorce (Lacey et al., 2013), as well as combined adverse childhood events scores (Slopen et al., 2010, 2013).

One childhood adversity which is not frequently investigated in relation to adult health is social isolation. Humans have a fundamental need to be socially connected to and supported by others (Baumeister and Leary, 1995). Social isolation in adulthood is known to be detrimental to health being associated with, for example, coronary heart disease (Kawachi et al., 1996). Chronic inflammation is thought to be one pathway linking psychosocial diversity to heart disease (Uchino, 2006). Few studies have addressed longer-term processes between childhood social isolation and adult health. Chronic isolation has been linked to school drop-out, problem drinking, and depression (Asher and Paquette, 2003) — risk factors for poorer adult health. However, little research has explored whether childhood social isolation is associated with objective markers of poorer adult health, such as inflammation.

Two studies using the Dunedin birth cohort have begun to investigate this association. Caspi and colleagues (2006) found that childhood social isolation, as indicated by items of the Rutter behavior scale, was associated with cardiovascular risk factors at age 26 (overweight, hypertension, raised glycated hemoglobin concentration, low maximum oxygen consumption and elevated total cholesterol). Danese et al. (2009) found that children with high levels of social isolation had a 60% increased risk of a CRP value >3 mg/L at age 32, compared to children who experienced a very low level of isolation.

A number of pathways are hypothesized to be important between child isolation and adult health. Firstly, poor social relations in childhood may result in lower educational attainment and this has many consequences including adverse trajectories of occupational and social position (Brown and Taylor, 2008). Secondly, child social isolation may increase psychological distress in adult life (Katz et al., 2011; Takizawa et al., 2014). Danese et al. (2009) also found that severe child social isolation was associated with increased depressive symptoms in adulthood. Thirdly, childhood social isolation may increase the risk of adult social isolation through the development of social and emotional mal-adaptation (Coplan et al., 2012). Caspi and colleagues (2006) found that child social isolation was strongly associated with adolescent and adult isolation, and that adult social isolation was in turn strongly associated with cardiovascular risk factors at the age of 26. Finally, social isolation in childhood may lower self-esteem and increase the risk of uptake and maintenance of adverse health behaviors, such as smoking (Niemela et al., 2011), problem alcohol consumption (Zimmerman et al., 1997) and overeating (Ackard et al., 2003). All of these factors in turn have been associated with poor health in adulthood. For instance, an association between adult socioeconomic position and inflammation has been shown numerous times, e.g. Ramsay et al. (2008), as have associations between adult social isolation (Shankar et al., 2011), psychological distress (Taylor et al., 2006), smoking (Koenig et al., 1999), alcohol misuse (Albert et al., 2003), and BMI (Festa et al., 2001), with inflammation.

Unlike previous studies we propose that mechanisms acting between child social isolation and inflammation are linked in complex ways. We therefore consider pathways in combination with each other. We extend previous work by utilizing a British birth cohort with follow-up into middle-age (more than a decade greater than previously examined), by accounting for missing data and by explicitly modeling the mechanisms involved. We conceptualize social isolation in this study as social withdrawal or social rejection, and this is reflected in our measure of child social isolation. Our hypothesis is that children who are socially isolated have higher CRP levels in midlife, and that this is accounted for by complex pathways across the life course acting through adult social isolation, health behaviors, material disadvantage and psychological distress. Fig. 1 shows the conceptual model tested in this study.

2. Methods

2.1. Sample

This study used data from the National Child Development Study (NCDS) which aimed to recruit all babies born in Great Britain during one week of 1958, achieving a sample of 17,414 (98.2%) (Power and Elliott, 2005). Participants were surveyed at the following ages: 7, 11, 16, 23, 33, 42, 44, 46, and 50 yrs. Information was collected from multiple sources on educational, social, medical, economic and aspects of participants' lives. Informed consent was sought from respondents for each survey and ethical approval was obtained from the South East and London multicenter research ethics committees (Shepherd, 2012). The age 44 survey took the form of a biomedical assessment, during which blood samples were taken on a sub-sample