Brain, Behavior, and Immunity xxx (2011) xxx–xxx

Contents lists available at ScienceDirect

Brain, Behavior, and Immunity

journal homepage: www.elsevier.com/locate/ybrbi

Early childhood socioeconomic status is associated with circulating interleukin-6 among mid-life adults

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ARTICLE INFO

Article history:
Received 23 February 2011
Received in revised form 16 May 2011
Accepted 31 May 2011
Available online xxxx

Keywords:
Childhood socioeconomic status
Inflammation
Interleukin-6
Early biological programming
Immune phenotype
SC childhood interview

ABSTRACT

It is proposed that socioeconomic conditions in early childhood effect immune programming, with poorer conditions resulting in adult phenotypes that are prone to inflammation. Recent evidence supports this possibility, showing an inverse association of childhood SES with adult markers of systemic inflammation. In this study, we further investigate this association, extending prior studies to include an examination of multiple indices of SES across distinct periods of childhood. Subjects were 112 men and women, 40–60 years of age (88.6% Caucasian). Childhood SES was indexed by a composite of three indicators of parental wealth (parental home and vehicle ownership, and number of bedrooms per child in the family home) averaged across 2 year periods of childhood between 1 and 18 years old. Higher adult serum concentrations of interleukin (IL)-6 were associated with lower SES in early childhood (years 1–2) ($\beta = -.05$, $p < .05$), associations that were independent of adult age, personal income, educational attainment, gender, race, body mass index, and physical activity. These associations support recent suggestions that the early environment may program immune phenotypes that contribute to disease risk.

1. Introduction

Growing evidence suggests that poorer socioeconomic conditions during childhood confer increased risk for a range of chronic inflammatory diseases and premature mortality among adults; effects that are independent of socioeconomic circumstances during adulthood (Cohen et al., 2010; Galobardes et al., 2004, 2006, 2008; Gliksman et al., 1995). In this regard, it has been proposed that social inequalities impact psychobiologic programming (Barker, 1998; Hertzman, 1999), resulting in adult phenotypes that are prone to central activation of brain regions involved in processing emotions, such as the amygdala (Gianaros et al., 2008), and increased peripheral activation of the sympatho-adrenal–medullary and hypothalamic–pituitary–adrenal (HPA) systems (Chen and Matthews, 2001; Chen et al., 2004, 2010a,b). Possibly as a result of increased activation of the HPA axis and heightened systemic levels of cortisol, lower childhood SES has also been associated with decreased responsiveness of adult immune cells to glucocorticoid signaling that down-regulates inflammation (Miller et al., 2009; Miller and Chen, 2007), and an associated increase in circulating markers of inflammation, such as C-reactive protein (CRP) and interleukin (IL)-6 (Danese et al., 2007; Phillips et al., 2009; Pollitt et al., 2007; Taylor et al., 2006a,b), although some report more modest effects that are attenuated when controlled for adult SES (Gimeno et al., 2008; Pollitt et al., 2007). Recent evidence also suggests that lifestyle factors, such as smoking, diet and physical activity, may also contribute to associations of childhood SES with adult inflammation (Danese et al., 2007; Pollitt et al., 2007). Further understanding of factors related to higher systemic levels of inflammatory markers in adulthood is warranted given their association with increased risk for a range of chronic diseases, including cardiovascular and metabolic disease, cancer, osteoarthritis, and dementia (Chung et al., 2009).

Animal evidence also supports an association of early adversity with programming of physiologic systems in adulthood (Coe and Lubach, 2003; Shanks and Lightman, 2001; Stevens et al., 2009; Zhang et al., 2006). For example, maternal separation, maternal stress, and pre- and post-natal exposure to corticotrophin-releasing hormone (CRH) have been associated with epigenetic changes to DNA that result in altered neuroplasticity of the amygdala and hippocampus, programming of hippocampal glucocorticoid receptors, and increases in neuroendocrine responses to stress (Gunnar and Quevedo, 2007; Liu et al., 1997; Weaver et al., 2004; Zhang and

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0889-1591/$ - see front matter © 2011 Elsevier Inc. All rights reserved.
doi:10.1016/j.bbi.2011.05.016