Psychoneuroendocrinology (2014) 43, 126—138

Available online at www.sciencedirect.com

ScienceDirect

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

Social relationships and their biological correlates: Coronary Artery Risk Development in Young Adults (CARDIA) study

Teresa E. Seeman [a,*], Tara L. Gruenewald [b], Sheldon Cohen [c], David R. Williams [d], Karen A. Matthews [e]

[a] David Geffen School of Medicine at UCLA, Los Angeles, CA, United States
[b] Davis School of Gerontology, University of Southern California, Los Angeles, CA, United States
[c] Department of Psychology, Carnegie Mellon University, Pittsburgh, PA, United States
[d] Department of Social and Behavioral Sciences, Harvard School of Public Health, Harvard University, Boston, MA, United States
[e] Department of Psychiatry, University of Pittsburgh, Pittsburgh, PA, United States

Received 1 October 2013; received in revised form 14 February 2014; accepted 15 February 2014

KEYWORDS
Social relationships;
Social support;
Social strains;
Allostatic load;
Biology

Abstract
Objective: Analyses test the hypothesis that aspects of social relationships (quantity of ties, social support and social strain) are associated with differences in levels of biological risk across multiple major physiological regulatory systems and consequently overall multi-systems risk (i.e., allostatic load [AL]).
Methods: Data are from the Coronary Artery Risk Development in Young Adults (CARDIA) study — a bi-ethnic, prospective, multi-center epidemiological study, initiated in 1985—1986 to track the development of cardiovascular risk in young adulthood (N = 5115). At the year 15 follow-up when participants were between 32 and 45 years of age, additional social and biological data were collected; biological data used to assess AL were collected at the Oakland, CA and Chicago, IL sites (N = 844).
Results: Social strains were most strongly and positively related to overall AL (Cohen's d = .79 for highest vs. lowest quartile), and to each of its component biological subsystems, independent of social ties and support as well as sociodemographics and health behaviors. Social ties and emotional support were also negatively related to AL (Cohen's d = .33 and d = .44 for lowest vs. highest quartiles of ties and support, respectively) though controls for social strains reduced these associations to non-significance. Social support and social strain were more strongly related to overall AL than to any of its component subscales while social ties were less strongly related to

* Corresponding author at: 10945 Le Conte Avenue, Suite 2339, Los Angeles, CA 90095. Tel.: +310 825 8253; fax: +310 794 2199.
E-mail address: tseeman@mednet.ucla.edu (T.E. Seeman).

http://dx.doi.org/10.1016/j.psyneuen.2014.02.008
0306-4530/

AL and to its component subscales. There was no evidence that effects differed by sex, age or ethnicity.
Conclusions: Findings focus attention on the particularly strong relationship between social strains and profiles of biological risk and support the cumulative impact of social factors on biological risks, showing larger effects for cumulative AL than for any of the individual biological systems.

1. Introduction

Humans are social animals that evolved in groups, tribes, clans, families. Thus, it is not surprising that our relationships with others appear to play significant roles in multiple aspects of our lives, not the least of which is our health (Brooks, 2008). Indeed, research interest in the role that social relationships may play in health and longevity is long-standing (House et al., 1988). Initial evidence for social relationship influences on health focused largely on links between social relationships — both number and quality — and major health outcomes, including major morbidity, functional status and mortality (House et al., 1988; Seeman, 1996). As such evidence has accumulated there has been a related increase in efforts to elucidate the pathways through which such relationships impact health, with particular interest in the ultimate biological pathways through which social influences are translated into health risks.

Links between social relationships and physiology are postulated to operate largely via social influences on our brain's cognitive-emotional interpretation of stimuli, and the resulting patterns of physiological activity in major biological regulatory systems, which, in turn, affect risks for most major health conditions (DeVries et al., 2003; Uchino, 2006). Functional magnetic imaging (fMRI) studies have begun to document the ways in which different social experiences are processed by the brain, showing effects on brain processes likely to influence biological systems (e.g., cardiovascular, metabolic, hypothalamic-pituitary-adrenal [HPA] axis, autonomic nervous system, inflammation). Eisenberger et al. (2007) found that greater reported social support was associated with diminished neurocognitive as well as neuroendocrine (cortisol) reactivity to social stressors, with diminished neural activity seen in regions (e.g., dorsal anterior cingulate cortex) previously shown to be associated with social separation distress (Eisenberger et al., 2003). Coan et al. (2006) reported that in married women who were shown cues indicating a probability of receiving an electric shock, the degree of attenuation of activation in neural structures supporting emotional and behavioral threat responses depended on whether the woman held her husband's hand (greatest attenuation), a stranger's hand, or no one's hand (least attenuation) with marital quality also related to the extent of attenuation associated with spousal hand-holding — better quality relationships associated with greater attenuation. These variations in neural activity as a function of social relationship characteristics are consistent with the hypothesis that there is further, downstream social patterning of the body's major physiological regulatory processes that are themselves regulated by these neural structures.

To date, efforts to understand potential biological pathways have focused on delineating relationships between aspects of social engagement, including numbers of relationships, types of relationships and the quality of social interactions (e.g., frequency and types of support); and various major physiological regulatory systems. Findings include evidence for positive relationships between greater social engagement and support and lower blood pressure (BP), better metabolic profiles and lower relative weight, lower levels of inflammation and of major stress hormones in adults (Seeman and McEwen, 1996; Uchino, 2006; Midei and Matthews, 2009). A growing literature also provides support for the hypothesis that these social influences are present throughout life, with evidence for differences in blood pressure (Evans, 2003); metabolic control (Helgeson et al., 2007); inflammation (Chen et al., 2011); and poorer stress hormone regulation (Evans, 2003) among young children (and even infants) according to the quality of their relationships with parents (Luecken and Lemery, 2004) and among adolescents (Helgeson et al., 2007). Though smaller in number, studies have also provided evidence that social interactions characterized by conflict or strain are associated with poorer biological profiles, including elevations in BP (Holt-Lunstad et al., 2003), metabolic functioning, inflammation (Brooks and Dunkel Schetter, 2011) and stress hormones such as cortisol, norepinephrine and epinephrine (see Seeman and McEwen, 1996; Brooks and Dunkel Schetter, 2011 for reviews).

Though such evidence points to relationships between the quantity and quality of social relationships and biological processes known to impact major health outcomes, the strength of these relationships is generally somewhat modest. The array of such relationships, however, — with evidence for associations across nearly all major regulatory systems — raises the possibility that from a cumulative risk standpoint, the health risks that may ensue from this array of individually modest physiological effects of social relationships could be significantly greater than would be suggested by findings for individual biological processes. Recent work on the concept of allostatic load — a multisystems view of physiological risks — has provided initial support for this hypothesis. In prior work, we and others have documented significant associations between greater social engagement (more social contacts) and support and greater allostatic load (Seeman et al., 2004; Evans et al., 2007). Indices of allostatic load in these cases represented cumulative, summated scores reflecting patterns of physiological dysregulation with