Biological Psychology 115 (2016) 43–49

Contents lists available at ScienceDirect

Biological Psychology

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

Social support sources matter: Increased cellular aging among adults with unsupportive spouses

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ARTICLE INFO

Article history:
Received 29 June 2015
Received in revised form 11 January 2016
Accepted 12 January 2016
Available online 15 January 2016

Keywords:
Social support
Telomere length
Married persons

ABSTRACT

Social support is associated with better health but it is unknown whether the health advantages of social support depend on the support source. Using a probability sample of older U.S. adults ($n = 1430$) we compared leukocyte telomere length, a biomarker of cellular aging, between married adults whose support sources either did or did not include their spouse. Despite having social support from other sources, participants who lacked spousal support had shorter telomeres relative to those with spousal support. The size of this telomere difference was comparable to differences between men and women and was independent of sociodemographic variables, coronary heart disease risk, diagnosed chronic disease and other social relationship resources such as the number of support sources, the number of friends, or the availability of financial support. Our findings suggest that relative to other sources of social support, spousal support may be especially important for cellular aging, a general biological mechanism that is implicated in age-related chronic disease risk.

1. Introduction

Social support, which encompasses both received and perceived availability of social resources (Cohen, 2004), is associated with better mental (Barger, Messerli-Burgy, & Barth, 2014; Finch, Okun, Pool, & Ruehlman, 1999) and physical health (Croezen et al., 2010; Holt-Lunstad, Smith, & Layton, 2010). Despite this evidence a key question persists; are the health benefits of social support dependent upon who provides it? Some models of social support are silent with regard to this issue (Berkman, Glass, Brissette, & Seeman, 2000; Cohen, 2004) whereas others explicitly view supportive relationships as exchangeable, i.e., that problems in one social relationship milieu can be offset by support from other relationships (Cantor, 1979; Holt-Lunstad et al., 2010; Lepore, 1992). A possible exception to these general models of support is marital relationships, where spousal support is the strongest determinant of well-being relative to other support sources (Buber & Engelhardt, 2008; Chen & Feeley, 2014; Dean, Kolody, & Wood, 1990; Li, Ji, & Chen, 2014; Okabayashi, Liang, Krause, Akiyama, & Sugisawa, 2004). In particular, low spousal support, as compared to moderate or high support, is most strongly associated with poor well-being even in the context of support from other sources (Dean et al., 1990). Thus, a lack of spousal support is particularly detrimental and is unlikely to be overcome by support from other sources (Baumeister & Leary, 1995; Coyne & DeLongis, 1986).

Although important for well-being, the extent to which spousal support is singular for physical health has received less attention. Spousal support is a primary determinant of the health benefits of marital relationships (Burman & Margolin, 1992) and poor marital quality is associated with worse physical health through several biologically plausible pathways, including activation of the hypothalamic–adrenal–pituitary axis (HPA), autonomic nervous system, or inflammatory processes, especially in response to conflict (see Robles, Slatcher, Trombello, & McGinn, 2014 for a review). This suggests that spousal support, or a lack thereof, should be an important determinant of biological processes related to poor physical health among married people. Thus, the aim of the present study was to examine associations between spousal support and leukocyte telomere length, a marker of cell function and senescence (Aviv, Kark, & Susser, 2015) that declines with age (Houben, Giltay, Rius-Ottenheim, Hageman, & Kromhout, 2011) and is associated with increased chronic disease risk and premature mortality (Haycock et al., 2014; Needham, Rehkopf et al., 2015; Willeit et al., 2010).

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http://dx.doi.org/10.1016/j.biopsycho.2016.01.003
0301-0511/

Table 1
Demographic, health and social characteristics of married NHANES participants.

	Without spousal support (n = 218)		With spousal support (n = 1212)		P value
Age (years)	M = 70.3	[69.1, 71.5]	M = 69.2	[68.8, 69.6]	0.079
Sex (% female)	67	[60, 74]	39	[36, 41]	<0.001
Race/ethnicity (%)					
Mexican American	4	[2, 6]	3	[2, 4]	
Other Hispanic	3	[1, 9]	3	[2, 6]	
Non-Hispanic White	85	[78, 90]	86	[82, 89]	0.551
Non-Hispanic Black	4	[2, 7]	5	[3, 6]	
Other race	4	[1, 12]	4	[2, 6]	
Education (%)					
<9th grade	10	[7, 15]	11	[9, 14]	
9–11 no diploma	15	[9, 22]	15	[12, 18]	
High school diploma	30	[22, 40]	28	[24, 32]	0.582
Some college	28	[18, 40]	21	[19, 24]	
College graduate or higher	18	[12, 25]	25	[21, 29]	
Chronic diseases (%)					
None	71	[62, 79]	69	[66, 72]	
One	17	[12, 23]	21	[19, 24]	0.712
Two or more	12	[7, 20]	10	[8, 13]	
Framingham risk (%)	M = 14.0	[12.8, 15.1]	M = 15.6	[15.0, 16.2]	0.012
Telomere length ratio	M = 0.87	[0.82, 0.93]	M = 0.91	[0.89, 0.94]	0.049
Number of friends (%)					
0	1	[0, 4]	2	[1, 3]	
1–2	13	[8, 21]	11	[9, 14]	
3–4	17	[10, 26]	17	[14, 19]	0.125
5–6	31	[22, 43]	26	[22, 30]	
7–11	17	[11, 24]	21	[18, 25]	
12 or more	21	[14, 29]	24	[21, 27]	
Financial support available (% yes)	81	[71, 88]	84	[81, 87]	0.422
Number of support sources					
0	0	–	0	–	
1	67	[57, 76]	43	[39, 47]	
2	20	[14, 27]	23	[19, 27]	
3	12	[7, 19]	21	[18, 25]	<.001
4	1	[0, 6]	8	[6, 10]	
5	0	[0, 2]	4	[3, 5]	
6	0	–	1	[0, 2]	
7	0	–	0	[0, 1]	
8	0	–	0	[0, 0]	
9	0	–	0	[0, 0]	

Note: NHANES; U.S. National Health and Nutrition Examination Survey 1999–2002. 95% confidence intervals are in brackets. All values are weighted and incorporate the NHANES complex survey design. Framingham risk reflects the percentage risk of a having a coronary heart disease event over the next ten years. Chronic diseases included prior diagnosis of cancer, heart disease or stroke.

Telomeres are protein–nucleotide sequences that cap the ends of chromosomes and help to promote chromosomal stability (for a review see D'Mello et al., 2014). With each successive replication of the cell, telomeres shorten and when a critical threshold is met the result is cellular senescence. This mechanism serves several critical genomic purposes, including the prevention of chromosomal fusions and unregulated cellular activity (Chan & Blackburn, 2003). However, senescent cells also show increased secretion of pro-inflammatory cytokines which, in turn, may accelerate the progression of chronic diseases with immune system involvement (Blackburn, 2005).

Shorter leukocyte telomere length is associated with increased risk of age-related chronic diseases and increased overall risk of earlier mortality (Haycock et al., 2014; Needham, Rehkopf et al., 2015; Willeit et al., 2010). Shorter leukocyte telomeres have been independently associated with depression in cross-sectional investigations (Puterman et al., 2013; Simon et al., 2006) and tend to decline with chronic stress over as short a time-span as 1 year (Puterman, Lin, Krauss, Blackburn, & Epel, 2014). Moreover, leukocyte telomere length is inversely associated with coronary heart disease (CHD) risk (Haycock et al., 2014), cancer incidence and mortality (Willeit et al., 2010) and with type-2 diabetes, myocardial infarction, and stroke in case-control studies (D'Mello et al., 2014). Thus leukocyte telomere length reflects a useful biomarker of cellular aging and may serve as a general biological mechanism linking spousal support with age-related chronic disease risk.

The key question for social relationship theory is whether or not spousal support is associated with biologically-plausible pathways that are implicated in age-related chronic disease risk. If not, this would confirm general support models (Berkman et al., 2000; Cohen, 2004; Lepore, 1992). If so, this would challenge general support models and show that social support is contingent upon the support source (Baumeister & Leary, 1995; Coyne & DeLongis, 1986). Beyond associations between shorter leukocyte telomere length and marital status (Mainous et al., 2011), there is some cross-sectional evidence showing an association between low social support and shorter leukocyte telomere length (Carroll, Diez Roux, Fitzpatrick, & Seeman, 2013). In particular, a greater number ambivalent social ties (Uchino et al., 2013) and the level of perceived social control exerted by one's social network are associated with shorter leukocyte telomere length (Uchino et al., 2015). We know of no prior studies that have directly tested associations between spousal support and leukocyte telomere length.

Our research question was whether spousal support was associated with telomere length. We examined several working hypotheses. First, we hypothesized that individuals who did not nominate their spouse as a source of social support would have shorter leukocyte telomeres than those who nominated a spouse.