

# Social Relationships and Allostatic Load in the MIDUS Study

Kathryn P. Brooks  
University of California, Los Angeles

Tara Gruenewald  
University of Southern California

Arun Karlamangla, Peifung Hu, Brandon Koretz, and Teresa E. Seeman  
University of California

**Objective:** This study examines how the social environment is related to allostatic load (AL), a multisystem index of biological risk. **Method:** A national sample of adults ( $N = 949$ ) aged 34–84 rated their relationships with spouse, family, and friends at 2 time points 10 years apart. At the second time point, participants completed a biological protocol in which indices of autonomic, hypothalamic-pituitary-adrenal axis, cardiovascular, inflammatory, and metabolic function were obtained and used to create an AL summary score. Generalized estimating equations were used to examine the associations among 3 aspects of social relationships—social support, social negativity, and frequency of social contact—and AL. **Results:** Higher levels of spouse negativity, family negativity, friend contact, and network level contact were each associated with higher AL, and higher levels of spouse support were associated with lower AL, independent of age, sociodemographic factors, and health covariates. Tests for age interactions suggested that friend support and network support were each associated with higher AL among older adults, but at younger ages there appeared to be no association between friend support and AL and a negative association between network support and AL. For network negativity, there was a marginal interaction such that network negativity was associated with higher AL among younger adults but there was no association among older adults. **Conclusions:** These findings demonstrate that structural and functional aspects of the social environment are associated with AL, and extend previous work by demonstrating that these associations vary based on the type of relationship assessed and by age.

**Keywords:** allostatic load, social support, social negativity, relationships, aging

**Supplemental materials:** <http://dx.doi.org/10.1037/a0034528.supp>

The structure and function of social relations predicts rates of morbidity and mortality (see reviews by Cohen, 2004; Taylor, 2007; Uchino, 2004). Prospective studies demonstrate that individuals with more and/or higher quality social relationships have lower rates of disease-specific (Brummett et al., 2001; Pinquart & Duberstein, 2010) and all-cause mortality over time (Holt-Lunstad, Smith, & Layton, 2010).

One pathway by which relationships influence health involves stress-related physiological systems. Stressors elicit autonomic ner-

vous system (ANS) and hypothalamic-pituitary-adrenocortical (HPA) axis activity designed to achieve physiological homeostasis, and over time these activations can incur wear and tear on the systems involved and can have damaging effects on cardiovascular, immune, and metabolic parameters (McEwen, 1998). The physiological processes initiated to address psychosocial, behavioral, and environmental demands are referred to as allostatic processes, and allostatic load (AL) refers to the cumulative cost of these adaptations (McEwen, 1998). Operationalizations of allostatic load typically assess an array of biomarkers representing key systems which carry out allostatic processes, including neuroendocrine, cardiovascular, metabolic and immune biomarkers. A common strategy is to create dichotomous indicators of “allostatic load” on specific biomarkers defined as having a biomarker value in a “high risk” range based on clinically established criteria or sample-derived thresholds (e.g., top quartile). Information for individual biomarkers or biomarkers of specific systems are then combined to create a multisystem measure of risk (Seeman, Singer, Rowe, Horwitz, & McEwen, 1997). AL composites have been shown to predict functional decline over a 7-year period in older adults (Karlamangla, Singer, McEwen, Rowe, & Seeman, 2002), incidence of cardiovascular disease (Seeman, Singer, Rowe, Horwitz, & McEwen, 1997), the development of frailty (Gruenewald, Seeman, Karlamangla, & Sarkisian, 2009), and risk of mortality over 7 years (Seeman, McEwen, Rowe, & Singer, 2001), independent of sociodemographic characteristics and baseline health.

---

This article was published Online First January 20, 2014.

Kathryn P. Brooks, Department of Psychology, University of California, Los Angeles; Tara Gruenewald, Davis School of Gerontology, University of Southern California; Arun Karlamangla, Peifung Hu, Brandon Koretz, and Teresa E. Seeman, Department of Medicine/Geriatrics, Geffen School of Medicine, University of California.

This work was supported by grants from the National Institute on Aging (grant numbers K01-AG028582 to T.G., R01-AG032271 to T.S., R01-AG033067 to A.K., and P01-AG020166), the MacArthur Foundation Network on Midlife (which funded MIDUS I data collection), the University of California Los Angeles (UCLA GCRC Grant M01-RR000865 and CTSI Grant # UL1TR000124) and fellowship support from the NIMH (training Grant MH15750 to K.B.).

Correspondence concerning this article should be addressed to Kathryn P. Brooks, Department of Psychology, University of California, 1285 Franz Hall, Los Angeles, CA 90095. E-mail: [brooksk@ucla.edu](mailto:brooksk@ucla.edu)

Although positive aspects of relationships such as social support have been associated with dampened ANS and HPA responses to stress (Ditzen, Hoppmann, & Klumb, 2008; Smith, Ruiz, & Uchino, 2004), negative aspects such as conflict with a romantic partner have been associated with acute elevations in these same parameters (Kiecolt-Glaser et al., 1993). These stress-related alterations have deleterious downstream consequences for other allostatic systems, and there is evidence linking the quality of social relationships with cardiovascular (e.g., Smith & Ruiz, 2002; Baker et al., 2000), immune (for review, see Kiecolt-Glaser, Gouin, & Hantsoo, 2010), and metabolic parameters (Helgeson, Lopez, & Kamarck, 2009; Troxel, Matthews, Gallo, & Kuller, 2005).

A handful of cross-sectional studies suggest that the quantity and quality of social ties are related to AL, controlling for age. In a sample of older adults from the Wisconsin Longitudinal Study, individuals reporting more positive relations with a spouse and with parents in childhood had lower AL scores than individuals with less positive relations (Singer & Ryff, 1999). Among older adults from the MacArthur Study of Successful Aging, those reporting higher numbers of social ties were at lower risk of having high AL (Seeman, Singer, Ryff, Dienberg Love, & Levy-Storms, 2002). In the MacArthur sample, higher levels of demands/criticism from a spouse were related to higher AL scores, and higher levels of emotional support from the network as a whole were related to lower AL scores in men (Seeman et al., 2002). Finally, in a sample of older Taiwanese adults, those reporting consistently high numbers of social ties had lower AL than those reporting fewer numbers of ties (Seeman et al., 2004). In a related study of older Taiwanese adults, higher levels of perceived demands from others were associated with higher AL (Weinstein, Goldman, Hedley, Yu-Hsuan, & Seeman, 2003).

Taken together, these studies suggest that individuals reporting more social ties and more positive relationship experiences have lower AL. However, limited research to date has explicitly considered the extent to which these associations may vary based on the *type* of relationship. Close ties are generally more consequential for psychological functioning than peripheral ties, and marital quality is consistently a stronger predictor of health outcomes than the quality of relationships with other family members, friend, or acquaintances (Robles & Kiecolt-Glaser, 2003). There is some evidence that ties with family (spouse, children) are more influential for AL than peripheral ties (Seeman et al., 2002).

In addition to failing to test for moderation by relationship type, research to date has not given equal consideration to the positive and negative aspects of social relationships. The functional aspect of social relations most frequently studied in regards to health is *social support*, defined here as the perception or experience that one is loved and cared for by others, esteemed and valued, and part of a social network of mutual assistance and obligation (Wills, 1991). However, relationships can involve unpleasant aspects like conflict, insensitivity, and interference, which we refer to broadly as *social negativity* (Brooks & Dunkel Schetter, 2011). As defined here, social negativity involves behaviors from network ties that are directed at the recipient and are perceived as aversive or unwanted. The presence of social negativity is not equivalent to the absence of support, as positive and negative aspects of relationships consistently emerge as distinct factors (Okun & Lockwood, 2003). Previous research on relationships and AL has either treated

relationship quality as unidimensional or has used limited indicators of negativity, often a single item. In order to understand the associations between relationships and AL, it is necessary to account for support and negativity as distinct dimensions.

A final limitation of existing research on social relationships and AL is that it has been conducted exclusively among older adults. The effects of social relationships on AL may increase with age, due to the increased physical vulnerability that accompanies normal aging. In addition, older adults may care more deeply about the quality of their close relationships, as compared with younger adults (Carstensen, Isaacowitz, & Charles, 1999), which may render their relationships more impactful for psychological and physiological functioning. Alternatively, the associations among social relationship qualities and AL may be less discernible among older adults due to the presence of chronic disease and/or the accumulation of risk factors that accompany normal aging. Due to greater age-related physiological dysregulation, the structure and function of relationships may explain a relatively smaller percentage of the variance among older adults.

The aim of this study is to examine how structural and functional aspects of the social environment are related to AL in a large, community-based sample of adults whose ages range from young adulthood to older age. Based on evidence that more positive and/or less negative social functioning is related to lower AL, we expected that higher levels of support and lower levels of negativity would each be independently associated with lower AL, controlling for age and relevant sociodemographic factors. We expected that these effects would be stronger for spouse and family than for friends. Based on previous evidence that greater social integration is associated with lower AL, we expected that more frequent contact with friends and family would be related to lower AL, controlling for age and relevant sociodemographic factors. We also took advantage of the wide age range in the Midlife in the U.S. (MIDUS) study to explore whether the effects of these three social variables varied by age.

## Method

### Procedure

This study uses data from the study of Midlife in the U.S. (MIDUS), a longitudinal study of health and aging in the United States. The initial wave of the study (MIDUS 1) was conducted in 1994–1995, when a national sample of 3,487 individuals were surveyed via telephone using random digit dialing. All participants were noninstitutionalized, English-speaking adults aged 25–74 living in the U.S. The original cohort was resurveyed approximately 9 years later (range = 7.8–10.4 years); the longitudinal response rate at MIDUS 2, adjusted for mortality, was 75% (Radler & Ryff, 2010). Additional details about the sampling procedure are available elsewhere (Radler & Ryff, 2010).

The current analyses focus on the subset of individuals who participated in a biomarker substudy at MIDUS 2 ( $N = 949$ ). Participants were assigned to data collection sites based on their place of residence, and data were collected during a 24-hr stay at one of three General Clinical Research Centers (Washington, D.C., Los Angeles, CA, and Madison, WI) between July 2004 and May, 2009. The protocol included a physical exam, 12-hour overnight urine sample and fasting morning blood draw (for details of the

protocol, see Love, Seeman, Weinstein, & Ryff, 2010). Individuals participating in the biomarker protocol were comparable with the larger MIDUS 2 sample on demographic and health characteristics with the exception that participants in the biological protocol were less likely to smoke, more likely to have college degree, and less likely to have completed only high school or some college (Love, Seeman, Weinstein, & Ryff, 2010).

## Measures

**Social support and negativity.** At both MIDUS 1 and 2, support and negativity were measured from three sources—family (except spouse/partner), friends, and spouse/partner (if applicable)—using a self-administered questionnaire (Schuster, Kessler, & Aseltine, 1990). Similar items were used for each source, with the addition of two items in the spouse measures.

Social support was measured with items about the perceived availability of emotional support. For each source, respondents indicated how much the source “really cares about you” and “understands the way you feel about things,” how much they could “rely on [source] for help if you have a serious problem,” and how much they could “open up to [source] if you need to talk about your worries.” For spouse/partner, respondents also rated how often he or she “appreciates you” and how much “you can relax and be yourself around him or her.” Items were rated on 4-point scales (1 = *A lot*, 4 = *Not at all*), and the measures were internally consistent (Cronbach’s alpha = .84, .88, and .90 for family, friend, and spouse/partner scales, respectively).

Social negativity was measured from each source by asking respondents to indicate how often each source “makes too many demands on you,” “criticizes you,” “lets you down when you are counting on [him or her],” and “gets on your nerves.” For spouse/partner, respondents also rated how often he or she “argues with you” and “makes you feel tense.” Items were rated on 4-point scales (1 = *Often*, 4 = *Never*), and the measure was internally consistent (Cronbach’s alpha = .77, .78, and .87 for family, friend, and spouse/partner scales, respectively).

Scores were computed for each variable by calculating the mean of the values of the items in each scale, with items reverse-coded such that higher scores indicated higher standing on that scale. The scales were computed for cases that had valid values for at least 50% of the items on the particular scale.

On average, network level support and negativity showed high levels of stability from M1 to M2, with small mean differences between waves: support  $M(SD) = 0.04 (0.41)$ , negativity  $M(SD) = -0.07 (0.38)$ . Given this stability, for analyses reported here we used summary scores reflecting the average across the two time points as both a cumulative index of exposures and a potentially more reliable assessment. These summary indices also serve to avoid the statistical issues that arise when using baseline scores plus change terms (Glymour, Weuve, Berkman, Kawachi, & Robins, 2005).

**Social contact.** At both MIDUS 1 and 2, the frequency of contact with family and friends was assessed by asking respondents, “How often are you in contact with any of your [friends/members of your family]—including visits, phone calls, letters, or electronic mail messages?” Response options ranged from 1–8, where 1 = *Never or hardly ever* and 8 = *Several times a day*. Responses were then reverse-coded such that higher scores indi-

cated more frequent contact. In addition to assessing contact within each domain, we examined a summary network contact measure based on the sum of the two items. Summary indices were then created by averaging social contact across M1 and M2 scores

**Allostatic load.** Consistent with previous work, the measure of allostatic load was designed to summarize dysregulation across multiple physiological systems (Seeman et al., 2001; Gruenewald et al., 2012). Biomarkers were selected based on two major criteria. First and foremost, biological parameters were selected on theoretical grounds (i.e., based on their known role as components of major regulatory systems). Second, selected parameters reflect those for which information could be collected within the logistical and financial constraints of the MIDUS project itself. Selection of subscale components was confirmed by results of factor analyses (Buckwalter et al., 2011).<sup>1</sup> Measures of (a) *cardiovascular* functioning included resting systolic blood pressure, pulse pressure, and resting pulse rate; indicators of (b) *sympathetic nervous system* activity included overnight urinary measures of epinephrine and norepinephrine; measures of (c) *parasympathetic nervous system* activity included the following heart rate variability parameters: low frequency spectral power, high frequency spectral power, the standard deviation of R-R (heartbeat to heartbeat) intervals, and the root mean square of successive differences; indicators of (d) *hypothalamic pituitary adrenal axis* activity included an overnight urinary measure of the hormone cortisol and a serum measure of the hormone dehydroepiandrosterone sulfate; measures of (e) *inflammation* included plasma C-reactive protein, fibrinogen, and serum measures of interleukin-6 and the soluble adhesion molecules E-selectin and intracellular adhesion molecule-1; indicators of (f) *lipid/fat metabolism* included high density lipoprotein cholesterol, low density lipoprotein cholesterol, triglycerides, body mass index, and waist-hip ratio; and levels of glycosylated hemoglobin, fasting glucose, and the homeostasis model of insulin resistance served as measures of (g) *glucose metabolism*.

For each of these seven systems, a system risk score was computed by calculating the proportion of individual biomarker indicators for that system for which participant values fell into high-risk quartile ranges. High risk was defined as the upper or lower quartile of the biomarker distribution, depending on whether high or low values of the biomarker typically confer greater risk for poor health outcomes. System risk scores were continuous and could range from 0 to 1 (indicating 0%–100% of system biomarkers in high-risk range for a given participant) despite differences in the number of biomarkers across systems. System risk scores were only computed for individuals with values on at least half of the system biomarkers. Rates of missing data were very low—98% of participants had complete biomarker data for each system, with the exception of the parasympathetic system, for which 8% of respondents were missing data on the parasympathetic scale due to instrumentation failures and/or measurement difficulties. An allostatic load summary score was computed as the sum of the seven system risk scores (possible range from 0 to 7; see Gruenewald et al., 2012, for overview). Allostatic load scores were computed for

<sup>1</sup> Specifically, Buckwalter et al. (2011) used principal component factor analysis on a set of biomarkers and compared the predictive power of seven obliquely rotated factors to that of a composite AL marker. The set of factors predicted more of the variance in measures of mental and physical health, suggesting that AL is best analyzed as a multisystem construct.

participants with information on at least six of the seven systems, and 90.5% of participants had data for all seven systems.

**Covariates.** Selection of covariates for inclusion in the current analyses was based on prior evidence suggesting that they could be potential confounders (i.e., that they have been related to both social relationship quality and allostatic load).

Sociodemographic covariates included *age* (in years), *gender*, *race* (Non-Hispanic/Latino White vs. Non-White), and *education*, which was assessed based on a 5-category, degree-based measure ranging from *less than high school* to *graduate school or more* and was treated as an ordinal variable in analyses. These variables were all assessed at Time 2.

The remaining covariates were assessed at both M1 and M2 unless otherwise noted. When covariates were available at more than one time point, the measure from each time point was included as a separate predictor. Health behaviors included current *smoking status* (0 = *Never*, 1 = *Past*, 2 = *Current*) and mean hours per week of *physical exercise* (M1: mean hours per week of vigorous exercise, M2: weighted average of light, moderate, and vigorous exercise). Health status was assessed as self-reports of *major chronic conditions* (e.g., heart disease, stroke, hypertension, and diabetes) and *functional status*, measured as whether the individual had any impairments in basic activities of daily living. *Depressive symptoms* were assessed using the Center for Epidemiologic Studies Depression scale (CES-D; Radloff, 1977), and *anxious symptoms* were assessed using the Mood and Anxiety Symptom Questionnaire (MAS-Q; Clark & Watson, 1991). Depressive and anxious systems were assessed at M2 only, as comparable measures were not available at M1.

## Analytic Plan

We examined the associations among the social variables and AL using generalized estimating equations (SPSS, Version 15.0) in order to account for the fact that a subset of the sample were pairs of twins ( $n = 158$  twin pairs). Generalized estimating equations permit estimation of parameter coefficients and standard errors while accounting for clustering. Parameter estimates provided in the text and tables are unstandardized coefficients.

**Social relationships and AL.** We began by examining associations between each of the three social variables (support, negativity, and contact) from each of the three sources (family, friend, spouse/partner) and AL. Primary analyses examined these sources in separate models to minimize issues of multicollinearity and secondary analyses included all sources in a single model. As a complement to these source-specific analyses, we examined the associations between AL and network level social variables. For all analyses, baseline models initially controlled for age and sociodemographic variables, followed by stepwise addition of health status, health behaviors, and depressive and anxious symptoms. We tested whether the association between each social variable and AL varied as a function of age by adding an age interaction term, with age centered at the mean. We retained the age interaction term in subsequent models only if the age interaction term was significant in the baseline model.

Family and friend data were available for the entire sample at both M1 and M2. Analyses of spouse data were restricted to those with a spouse at both time points ( $n = 660$ ) because our analyses focused on social information from both measurement occasions;

75 participants were excluded from this sample because they had a spouse/partner at M1 only, and 76 were excluded because they had a spouse/partner at M2 only.

As noted above, we included three sets covariates in our final models that may be associated with both social functioning and physiology—health status, health behaviors, and depressive/anxious symptoms. The temporal ordering of the data did not permit rigorous tests of mediation, therefore, these variables were included as cofounders. However, examining the extent to which observed associations between social variables and AL were altered by the inclusion of these covariates would point to their potential mediational role. Therefore, when the addition of the full set of covariates resulted in a considerable reduction in the effect of a social variable, we examined the percentage reduction associated with each of the three sets of covariates and reported that information in the text.

**Supplemental analyses.** As some prior research has documented gender differences in the associations between social factors and AL (Seeman et al., 2002), we tested for moderation by gender. In order to ensure that our findings regarding AL were not being driven by one or two physiological systems, we examined the associations between social relationships and system-specific physiological risk scores. Finally, we conducted sensitivity analyses to examine whether individuals reporting high levels of change in the quality of their social relationship over time differed from those whose ratings remained relatively stable.

## Results

Table 1 provides demographic information and descriptive statistics. The sample was largely White and relatively highly educated, with over 50% reporting at least some college. Participants in this sample were 34 to 84 years of age at M2, with an average age of 55.07 ( $SD = 11.68$ ), and the majority were married at both time points (70%).

Individuals reporting higher support tended to be older, female, and White, and those reporting higher negativity tended to be younger, female, and non-White. Women reported more frequent social contact than men. At the network level, higher levels of support were associated with higher levels of contact ( $r = .35, p < .001$ ) and lower levels of negativity ( $r = -0.35, p < .001$ ); contact was not related to negativity (see Supplement for full correlation matrix, including source-specific social variables). Older age and lower levels of education were associated with higher AL, but AL did not vary by sex or race.

## Social Relationships and AL

Table 2 reports results of generalized estimating equations examining associations between each of the social domains and allostatic load. Within each domain, we report the source-specific results first, followed by the results for the summary network level indices.

**Support.** Higher spouse support was associated with lower AL, independent of age and other covariates. Family support was unrelated to AL and although the age interaction term was marginal in the baseline model, it was not significant in the final model. Friend support exhibited a surprising positive association with AL in the final model, though a significant age interaction

Table 1  
Descriptive Statistics (N = 949)

Variable	M (SD)
Age (M2)	55.07 (11.68)
Male (%)	46.1
Non-Hispanic/Latino White (%)	93.3
Education (%)	
< High school	3.2
High school diploma/G.E.D.	20.7
Some college/AA	28.6
BA/BS	23.5
Graduate school or more	23.9
Social experience (Average of M1/M2)	
Family support	3.51 (0.52)
Friend support	3.31 (0.56)
Spouse support*	3.64 (0.45)
Network level support (average of family, friends, and spouse)	3.44 (0.45)
Family negativity	2.06 (0.51)
Friend negativity	1.86 (0.40)
Spouse negativity*	2.17 (0.53)
Network level negativity (average of family, friends, and spouse)	2.03 (0.38)
Family contact	5.92 (1.22)
Friend contact	5.69 (1.39)
Network level contact (sum of family and friends)	11.61(2.05)
Health related covariates (M1 and M2)	
Smoking: M1 (% Never, Past, Current)	55.5, 30.7, 14.3
Smoking: M2 (% Never, Past Current)	56.6, 32.7, 10.8
Exercise: M1 (mean hrs/wk of vigorous exercise)	3.41 (1.56)
Exercise: M2 (mean weighted hrs/wk of light, moderate, and vigorous exercise)	3.38 (5.03)
Major chronic conditions: M1 (number of conditions)	0.46 (0.71)
Major chronic conditions: M2 (number of conditions)	1.02 (1.11)
Functional status: M1 (number of impairments)	0.16 (0.69)
Functional Status: M2 (number of impairments)	0.37 (0.98)
Depressive symptoms: M2	7.97 (4.34)
Anxious symptoms: M2	16.50 (4.34)
Allostatic load (M2)	
Allostatic load summary score	1.70 (1.03)

\* n = 660.

revealed that this association was largely seen at older ages. As illustrated in Figure 1, the pattern of values suggests that among older adults, higher levels of friend support were associated with higher AL, but there appeared to be no association for younger adults.

Network level support also exhibited a significant age interaction suggesting that greater support was associated with lower AL at younger ages but was associated with higher AL at older ages (see Figure 1). When models testing the association between support and AL were run in each age group separately (34–50, 50–65, 65+) higher support was associated with lower AL for those aged 34–50 (B = -0.28, p < .05) in the baseline model, but was rendered nonsignificant with the addition of health status (B = -0.16, p > .10). For the middle-aged and older groups, there was no association between support and AL, although the support coefficient was consistently positive in both the baseline (age 50–65, B = 0.07, p > .10; age 65+, B = 0.13, p > .10) and final models (age 50–65, B = 0.14, p > .10; age 65 + B = 0.19, p > .10).

**Negativity.** Higher family negativity was associated with higher AL in the baseline model, and the effect remained significant in the final model, although there was a 36% reduction in the effect. The addition of health status was associated with a 36% reduction in the effect, and health behaviors and depressive/anxious systems were associated with reductions of 5% and 9%, respectively. The effects of family negativity did not interact with age, and there were no associations or age interactions between friend negativity and AL.

Higher spouse negativity was associated with higher AL, and this effect did not vary by age. There was a 13% reduction in the effect when the full set of covariates were added to the baseline model, and tests of individual covariates revealed that the addition of health status and health behaviors were each associated with a 13% reduction.

Network level negativity was associated with marginally higher AL in the final model, and a marginal age interaction indicated that this relationship was strongest at younger ages. Tests of the model by age group indicated that among those aged 34–50, higher levels of negativity were associated with higher AL (B = 0.40, p < .01) but there was no association in the older groups (age 50–65, B = 0.15, p > .10; age 65+, B = -0.15, p > .10). These results are illustrated in Figure 1.

**Contact.** More frequent friend contact was associated with higher AL, independent of all covariates, but family contact was not related to AL. Averaged across both sources, higher network

Table 2  
Unstandardized Coefficients (and Standard Errors) for Social Variables From Generalized Estimating Equations Predicting AL

Variable	Baseline model <sup>a</sup>	Final model <sup>b</sup>
Family support	-0.03 (0.06)	0.02 (0.06)
Family support × age	0.01 (0.01) †	0.01 (0.01)
Friend support	0.04 (0.06)	0.12 (0.06)*
Friend support × age	0.01 (0.00)*	0.01 (0.00)*
Spouse support	-0.20 (0.08)*	-0.19 (0.08)*
Spouse support × age	0.01 (0.01)	—
Network level support	-0.05 (0.07)	0.03 (0.07)
Network level support × age	0.02 (0.01)**	0.02 (0.01)**
Family negativity	0.22 (0.07)**	0.14 (0.07)*
Family negativity × age	-0.01 (0.01) †	-0.01 (0.01)
Friend negativity	0.10 (0.08)	0.04 (0.08)
Friend negativity × age	0.00 (0.01)	—
Spouse negativity	0.16 (0.07)*	0.14 (0.07)*
Spouse negativity × age	-0.01 (0.01)	—
Network level negativity	0.26 (0.09)**	0.16 (0.09) †
Network level negativity × age	-0.01 (0.01)*	-0.01 (0.01) †
Family contact	0.04 (0.03)	0.03 (0.02)
Family contact × age	0.00 (0.00)	—
Friend contact	0.05 (0.02)*	0.05 (0.02)*
Friend contact × age	0.00 (0.00)	—
Network level contact	0.04 (0.01)*	0.04 (0.01)*
Network level contact × age	0.00 (0.00)	—

<sup>a</sup> Baseline model adjusted for age, sex, race, and education. <sup>b</sup> Final model adjusted for age, sex, race, education, major chronic conditions, functional status, smoking, physical activity, depressive symptoms, and anxious symptoms. Line breaks indicate a new model. Spouse analyses were conducted among only those participants who were married or partnered at both times points (n = 660).

† p < .10. \* p < .05. \*\* p < .01. \*\*\* p < .001.

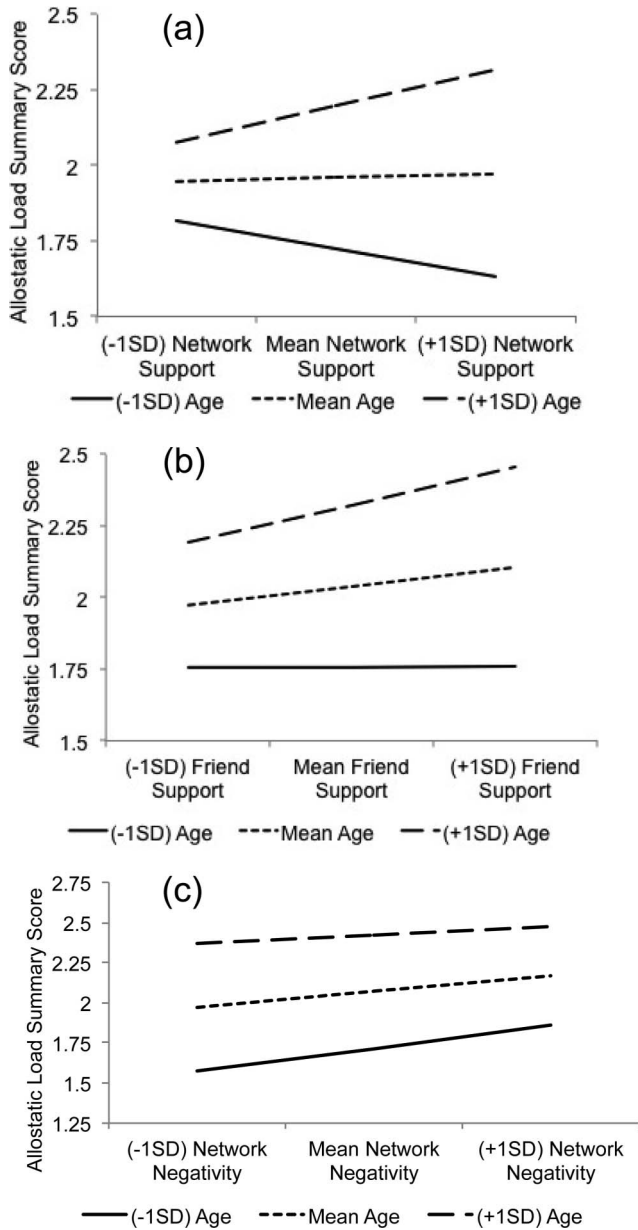


Figure 1. Predicted values of AL by (a) network level support and age, (b) friend support and age, and (c) network level negativity and age, controlling for sociodemographic covariates, health status, health behaviors, and depressive and anxious symptoms.

contact was associated with higher AL. There were no age × contact interactions.

As the positive associations between social contact and AL were unexpected, we ran a set of supplemental analyses to better understand the observed associations. First, we reasoned that the association between contact and AL might reflect the demands of caregiving, but controls for unpaid assistance and emotional support provided to others did not alter the original finding (results not shown). We explored whether high contact might exacerbate the effects of negativity, but none of the two- or three-way interactions

among the average social variables were significant (results not shown). Finally, we examined whether the frequency of social contact was confounded with other social structural variables which could have independent associations with AL, but controls for marital status, number of children, and number of friends did not alter the effect of contact (results not shown).

**Integrated models.** As reported in Table 3, when support, negativity and contact from friends and family were each entered as predictors in one integrated model, family negativity was associated with higher AL in the baseline model. This effect was reduced by 28% when the full set of covariates was added, but remained significant. Tests of individual sets of covariates indicated that health status was associated with the largest reduction in the effect (24%), with health behaviors and depressive/anxious symptoms associated with more modest reductions (7% and 10%, respectively). Friend contact was associated with marginally higher AL in both the baseline and final models.

When a similar model was run on the subset of continuously married/partnered participants, spouse support was associated with marginally lower AL in the baseline model. There was a 19% reduction in this effect when the full set of covariates was added and the effect was no longer significant. When tested individually, the percentage changes associated with the inclusion of health status, health behaviors, and depressive/anxious symptoms were 24%, 48%, and 10%. Similarly, family negativity was a marginal predictor of higher AL in the baseline model, but the effect was reduced by 30% in the final model and was no longer significant. The percentage reductions associated with health status, health behaviors, and depressive/anxious symptoms were 17%, 13%, and 13%, respectively.

Table 3  
Unstandardized Coefficients (and Standard Errors) for Social Variables From Generalized Estimating Equations Predicting AL, all Social Variables Entered as Predictors

Variable	Baseline model <sup>a</sup>	Final model <sup>b</sup>
Friends and family (N = 949)		
Family contact	0.03 (0.03)	0.02 (0.04)
Friend contact	0.05 (0.03) <sup>†</sup>	0.05 (0.03) <sup>†</sup>
Family support	0.01 (0.07)	0.01 (0.07)
Friend support	-0.01 (0.08)	0.05 (0.07)
Family negativity	0.29 (0.10)**	0.21 (0.09)*
Friend negativity	-0.10 (0.10)	-0.09 (0.10)
Friends, family, spouse (N = 660)		
Family contact	0.03 (0.04)	0.03 (0.04)
Friend contact	0.05 (0.04)	0.06 (0.04) <sup>†</sup>
Family support	0.05 (0.11)	0.05 (0.10)
Friend support	0.01 (0.10)	0.05 (0.10)
Spouse support	-0.21 (0.12) <sup>†</sup>	-0.17 (0.11)
Family negativity	0.23 (0.12) <sup>†</sup>	0.16 (0.12)
Friend negativity	-0.07 (0.12)	-0.06 (0.13)
Spouse negativity	-0.02 (0.10)	0.04 (0.10)

<sup>a</sup> Baseline model adjusted for age, sex, race, and education. <sup>b</sup> Final model adjusted for age, sex, race, education, major chronic conditions, functional status, smoking, physical activity, depressive symptoms, and anxious symptoms.  
<sup>†</sup>  $p < .10$ . \*  $p < .05$ . \*\*  $p < .01$ . \*\*\*  $p < .001$ .

## Supplementary Analyses

**Gender differences.** We tested whether gender moderated the associations between social relationships and AL by adding gender interaction terms to the models. None of the gender interaction terms were significant (all  $ps > .10$ ).

**System-specific analyses.** Analyses examining the associations between social variables and each of the seven system risk scores included in the AL composite are also available online, and the associations were generally consistent with those reported earlier for the AL summary score, although they tended to be smaller in magnitude and many did not reach statistical significance. Details of these analyses are available in an online supplement.

**Change sensitivity analyses.** Analyses comparing those who reported large changes in social experience from M1 to M2 with those who remained stable revealed no group differences in AL for any social variable except family contact where decreases were associated with higher AL (concurrent with worsening health status). Details of these analyses are available in an online supplement.

## Discussion

Results of this study indicated that structural and functional aspects of social relationships were associated with allostatic load (AL), an index of cumulative multisystem biological dysregulation, in a large sample of middle-aged and older adults. When social experience was examined by relationship type, ties with spouse and family appeared to have more generalized associations with AL; higher levels of family negativity and spouse negativity were each associated with higher AL, and higher levels of spouse support were associated with lower AL, independent of age, sociodemographic factors, and health covariates. Both friend contact and friend support were somewhat surprisingly associated with greater AL, though a significant age interaction for friend support indicated that this association was largely seen among the older adults. Aggregated across the social network, higher levels of social contact and negativity were associated with higher AL, although the association between negativity and AL was marginal in the final model.

Taken together, these findings build upon existing literature, pointing to potentially important differences in family versus friend effects as well as, for the first time, highlighting the extent to which social influences may vary by age. The strong effects of social negativity were also notable. Although the associations between social relationships and AL observed here are small (standardized coefficients range from  $-0.08$  to  $0.08$ ), they are comparable in magnitude with traditional risk factors and are thus meaningful predictors with clinical relevance. For example, a 1 *SD* increase spouse support was associated with a predicted decrease of 0.06 units in AL, and a 1 *SD* increase in exercise was associated with a predicted decrease of 0.11 units in AL. In other words, a 1 increase unit on the spouse support scale (e.g., from *some* support to *a lot* of support) was associated with a 0.19 unit increase in AL, roughly comparable with the reduction in AL associated with 9 additional hr of exercise per week.

Higher levels of negativity—criticism, insensitivity, and interference—from family and spouse were each associated with higher AL, and negativity across the social network was associated with

marginally higher AL. These findings are consistent with studies reporting a positive association between demands or criticism and AL (Weinstein et al., 2003; Seeman et al., 2002), as well as evidence that close relationships are more closely tied to physiology than more peripheral ties (Robles & Kiecolt-Glaser, 2003).

Spouse support was strongly and negatively related to AL, independent of age and sociodemographic covariates. This finding is consistent with other evidence that positive aspects of relationships are associated with lower AL (Seeman et al., 2002; Singer & Ryff, 1999), and with evidence that marriage has a more potent effect on physiology than other relationships (Robles & Kiecolt-Glaser, 2003). More generally, this finding suggests that the support provided by a spouse may be a powerful antidote to stress-related wear and tear on physiological systems.

Given the impressive age range in the MIDUS study, this study is the first to document age interactions in the associations between social support and AL. We found that network support was associated with lower AL among the youngest adults in our sample (age 34–50), suggesting that social support may serve a protective function in this age group. Among the older groups (50–65, 65+), the coefficients associated with network support were not statistically significant; we speculate that the associations between support and AL may be overshadowed by other biological risk factors in older age. These findings highlight the importance of accounting for age when examining associations between support and AL.

It is worth noting that our findings about social support and older adults stand in contrast to those of Seeman, Singer, Ryff, Dienberg Love, and Levy-Storms (2002), who found that higher levels of social support were associated with lower AL in a sample of older adults from the MacArthur Study of Successful Aging. This discrepancy may be due to the fact that the MacArthur sample was selected to represent the healthiest of 70- to 79-year-olds, whose biological profiles may have been more similar to the individuals under 65 in our study. Future research that incorporates temporal dimensions of unfolding support and health dynamics is needed to clarify the likely reciprocal relationships between social support and health—especially at older ages when health-related needs are increasingly common.

Contrary to expectation, more frequent social contact was associated with higher AL, and this finding appeared to be driven by the effects of friend contact. Although previous studies have documented negative associations between indices of social integration and AL (Seeman et al., 2002, 2004), these studies assessed the number of social ties as an indicator of integration, yet we measured the frequency of contact with others. It is not clear why more frequent contact with friends would be associated with higher AL. The association did not vary as a function of potential confounding variables such as marital status, social network size, amount of emotional, instrumental and financial support provided, or social network stressors. Our contact measure may have captured information about social obligations or demands which may not be rated as unpleasant, but which may involve physiological challenge. Alternatively, high friend contact could be associated with higher AL if those friendships were characterized by moderate levels of ambivalence (Holt-Lunstad, Uchino, Smith, & Hicks, 2007). Future work is needed to understand why the frequency of contact with others may be associated with higher AL.

The effect of family negativity was particularly robust, as it was maintained in an integrated model with all other social variables

added. The remaining observed associations between social variables and AL were not significant in these integrated models, but a comparison of point estimates indicates that the effects were largely unchanged. The fact that we were not able to document statistically significant associations in the integrated final models may be due to the correlations among social variables which also contributed to an increase in the size of the standard errors in the integrated models, thereby reducing the significance levels of the estimates.

We included health status, health behaviors, and depressive/anxious symptoms as confounders in our models given their known associations with both social functioning and physiology. Although the temporal ordering of our data precluded rigorous tests of mediation, the inclusion of these covariates in our analyses provides some evidence consistent with potential pathways. For example, our analyses suggest that health behaviors may play a larger role as potential mediators as compared with depressive and anxious symptoms. Further research, however, is needed with longitudinal data to ascertain whether these variables function as mediators. More generally, the fact that many of the associations between social variables and AL were independent of these covariates is noteworthy, and suggests that there are other pathways linking social relationships and AL.

It is worth noting that there has been some debate as to the conceptualization and measurement of AL. However, a recent factor analysis found that the components of AL did in fact reflect a latent factor (McCaffery, Marsland, Strohacker, Muldoon, & Manuck, 2012). The fact that our findings regarding AL were not being driven by one or two physiological systems further highlights the utility of the AL construct as a measure of cumulative physiological dysregulation.

Although some previous studies (Seeman et al., 2002) have documented gender differences in the associations between social factors and AL, we did not detect such differences. Speculatively, any gender differences may have been obscured by the wide age range of our study. Generational differences in gender roles (i.e., increased participation in the work force among younger women) may impact the manner in which gender interacts with social relationships to influence health. Future research is needed to better understand whether this is in fact the case.

This study has several limitations worth noting. Our measure of social support assessed emotional but not instrumental or informational support, which may have different associations with physiology. Although the sample is diverse in respect to age, gender, and socioeconomic status, it is not technically nationally representative. In particular, the MIDUS data do permit us to test whether the associations among social relationship qualities and AL observed here extend to non-White Americans, and future research should better examine possible racial/ethnic differences. An additional limitation of this study is the fact that AL was measured at only one time point, which precluded our ability to control for AL at baseline or to examine potential influences of prior AL on social processes assessed at MIDUS 1 and 2. Although there may be concerns about the significant length of time across the two measurement occasions, the lengthy interval allows us to assess long-term exposure to levels of relationship quality and to examine how these exposures are related to a multisystem indicator of physiological well-being. Future work ought to employ repeated assessments of AL over time to test whether social relationship qualities

predict changes in AL over time and how AL may relate to dynamics of reported social contact, support and negativity.

Strengths of this study include the fact that it is among the first to examine the associations among structural and functional aspects of social relationships and AL across the majority of the adult life span, rather than in a limited range of older adulthood. Importantly, the age range in the MIDUS sample allowed us to test for age differences in the effects of social experience on AL. Also, whereas previous work has focused on the positive aspects of social relationships, this study extends previous work by comparing positive and negative aspects of relationships and by comparing specific sources of support and negativity. An additional strength of this study is the measurement of AL, which is the most comprehensive to date, including assessment of multiple indicators of autonomic, endocrine, cardiovascular, metabolic, and immune system activity (Gruenewald et al., 2012). The findings of this study are consistent with previous evidence that the structure and function of social relationships are related to health-relevant physiology, and our study documents the persistence of associations from young adults through middle and older ages. Our findings also indicate that the positive and negative aspects of social ties have independent associations with physiology, and that these associations vary based on the type of relationship and based on age.

## References

- Baker, B., Paquette, M., Szalai, J. P., Driver, H., Perger, T., Helmers, K., . . . Tobe, S. (2000). The influence of marital adjustment on 3-year left ventricular mass and ambulatory blood pressure in mild hypertension. *Archives of Internal Medicine*, *160*, 3453–3458. doi:10.1001/archinte.160.22.3453
- Brooks, K. P., & Dunkel Schetter, C. (2011). Social negativity and health: conceptual and measurement issues. *Social and Personality Compass*, *5*(11), 904–918.
- Brummett, B., Barefoot, J., Siegler, I., Clapp-Channing, N., Lytle, B., Bosworth, H., . . . Mark, D. B. (2001). Characteristics of socially isolated patients with coronary artery disease who are at elevated risk for mortality. *Psychosomatic Medicine*, *63*, 267–272.
- Buckwalter, J. G., Rizzo, A., John, B. S., Finlay, L., Wong, A., Chin, E., . . . Seeman, T. E. (2011, December). *Analyzing the impact of stress: A comparison between a factor analytic and a composite measurement of allostatic load*. Paper presented at the 2011 Interservice/Industry Training, Simulation, and Education Conference, Orlando.
- Carstensen, L. L., Isaacowitz, D. M., & Charles, S. T. (1999). Taking time seriously: A theory of socioemotional selectivity. *American Psychologist*, *54*, 165–181. doi:10.1037/0003-066X.54.3.165
- Clark, L. A., & Watson, D. (1991). Tripartite model of anxiety and depression: Psychometric evidence and taxonomic implications. *Journal of Abnormal Psychology*, *100*, 316–336. doi:10.1037/0021-843X.100.3.316
- Cohen, S. (2004). Social relationships and health. *American Psychologist*, *59*, 676–684. doi:10.1037/0003-066X.59.8.676
- Ditzen, B., Hoppmann, C., & Klumb, P. (2008). Positive couple interactions and daily cortisol: On the stress-protecting role of intimacy. *Psychosomatic Medicine*, *70*, 883–889. doi:10.1097/PSY.0b013e318185c4fc
- Glymour, M. M., Weuve, J., Berkman, L. F., Kawachi, I., & Robins, J. M. (2005). When is baseline adjustment useful in analyses of change? An example with education and cognitive change. *American Journal of Epidemiology*, *162*, 267–278. doi:10.1093/aje/kwi187
- Gruenewald, T. L., Karlamangla, A. S., Hu, P., Merkin, S. S., Crandall, C., Koretz, B., & Seeman, T. E. (2012). History of socioeconomic disad-



- vantage and allostatic load in later life. *Social Science & Medicine*, 74, 75–83. doi:10.1016/j.socscimed.2011.09.037
- Gruenewald, T. L., Seeman, T. E., Karlamangla, A. S., & Sarkisian, C. A. (2009). Allostatic load and frailty in older adults. *Journal of the American Geriatrics Society*, 57, 1525–1531. doi:10.1111/j.1532-5415.2009.02389.x
- Helgeson, V. S., Lopez, L. C., & Kamarck, T. (2009). Peer relationships and diabetes: Retrospective and ecological momentary assessment approaches. *Health Psychology*, 28, 273–282. doi:10.1037/a0013784
- Holt-Lunstad, J., Smith, T. B., & Layton, J. B. (2010). Social relationships and mortality risk: A meta-analytic review. *PLOS Medicine*, 7, e1000316. doi:10.1371/journal.pmed.1000316
- Holt-Lunstad, J., Uchino, B. N., Smith, T. W., & Hicks, A. (2007). On the importance of relationship quality: The impact of ambivalence in friendships on cardiovascular functioning. *Annals of Behavioral Medicine*, 33, 278–290. doi:10.1007/BF02879910
- Karlamangla, A. S., Singer, B. H., McEwen, B. S., Rowe, J. W., & Seeman, T. E. (2002). Allostatic load as a predictor of functional decline. MacArthur studies of successful aging. *Journal of Clinical Epidemiology*, 55, 696–710. doi:10.1016/S0895-4356(02)00399-2
- Kiecolt-Glaser, J. K., Gouin, J. P., & Hantsoo, L. (2010). Close relationships, inflammation, and health. *Neuroscience and Biobehavioral Reviews*, 35, 33–38. doi:10.1016/j.neubiorev.2009.09.003
- Kiecolt-Glaser, J. K., Malarkey, W. B., Chee, M., Newton, T., Cacioppo, J. T., Mao, H. Y., & Glaser, R. (1993). Negative behavior during marital conflict is associated with immunological down-regulation. *Psychosomatic Medicine*, 55, 395–409.
- Love, G. D., Seeman, T. E., Weinstein, M., & Ryff, C. D. (2010). Bioindicators in the MIDUS national study: Protocol, measures, sample, and comparative context. *Journal of Aging and Health*, 22, 1059–1080.
- McCaffery, J. M., Marsland, A. L., Strohacker, K., Muldoon, M. F., & Manuck, S. M. (2012). Factor structure underlying components of allostatic load. *PLoS ONE*, 7, e47246.
- McEwen, B. S. (1998). Protective and damaging effects of stress mediators. *The New England Journal of Medicine*, 338, 171–179. doi:10.1056/NEJM199801153380307
- Okun, M. A., & Lockwood, C. M. (2003). Does level of assessment moderate the relation between social support and social negativity? A meta-analysis. *Basic and Applied Social Psychology*, 25, 15–35. doi:10.1207/S15324834BASP2501\_2
- Pinquart, M., & Duberstein, P. R. (2010). Associations of social networks with cancer mortality: A meta-analysis. *Critical Review of Oncology/Hematology*, 75, 122–137. doi:10.1016/j.critrevonc.2009.06.003
- Radler, B. T., & Ryff, C. D. (2010). Who participates? Accounting for longitudinal retention in the MIDUS National Study of Health and Well-Being. *Journal of Aging and Health*, 22, 307–331.
- Radloff, L. (1977). The CES-D scale: A self-report depression scale for research in the general population. *Applied Psychological Measurement*, 1, 385–401. doi:10.1177/014662167700100306
- Robles, T. F., & Kiecolt-Glaser, J. K. (2003). The physiology of marriage: Pathways to health. *Physiology & Behavior*, 79, 409–416. doi:10.1016/S0031-9384(03)00160-4
- Schuster, T. L., Kessler, R. C., & Aseltine, R. H. (1990). Supportive interactions, negative interactions, and depressed mood. *American Journal of Community Psychology*, 18, 423–438.
- Seeman, T. E., Gleib, D., Goldman, N., Weinstein, M., Singer, B., & Lin, Y. H. (2004). Social relationships and allostatic load in Taiwanese elderly and near elderly. *Social Science and Medicine*, 59, 2245–2257.
- Seeman, T. E., McEwen, B. S., Rowe, J. W., & Singer, B. H. (2001). Allostatic load as a marker of cumulative biological risk: MacArthur studies of successful aging. *Proceedings of the National Academy of Sciences of the United States of America*, 98, 4770–4775. doi:10.1073/pnas.081072698
- Seeman, T. E., Singer, B. H., Rowe, J. W., Horwitz, R. I., & McEwen, B. S. (1997). Price of adaptation—allostatic load and its health consequences. MacArthur studies of successful aging. *Archives of Internal Medicine*, 157, 2259–2268. doi:10.1001/archinte.1997.00440400111013
- Seeman, T. E., Singer, B. H., Ryff, C. D., Dienberg Love, G., & Levy-Storms, L. (2002). Social relationships, gender, and allostatic load across two age cohorts. *Psychosomatic Medicine*, 64, 395–406.
- Singer, B., & Ryff, C. D. (1999). Hierarchies of life histories and associated health risks. *Annals of the New York Academy of Sciences*, 896, 96–115. doi:10.1111/j.1749-6632.1999.tb08108.x
- Smith, T. W., & Ruiz, J. M. (2002). Psychosocial influences on the development and course of coronary heart disease: Current status and implications for research and practice. *Journal of Consulting and Clinical Psychology*, 70, 548–568. doi:10.1037/0022-006X.70.3.548
- Smith, T. W., Ruiz, J. M., & Uchino, B. N. (2004). Mental activation of supportive ties, hostility, and cardiovascular reactivity to laboratory stress in young men and women. *Health Psychology*, 23, 476–485. doi:10.1037/0278-6133.23.5.476
- Taylor, S. E. (2007). Social support. In H. S. Friedman & R. S. Silver (Eds.), *Foundations of health psychology* (pp. 145–171). New York, NY: Oxford University Press.
- Troxel, W. M., Matthews, K. A., Gallo, L. C., & Kuller, L. H. (2005). Marital quality and occurrence of the metabolic syndrome in women. *Archives of Internal Medicine*, 165, 1022–1027. doi:10.1001/archinte.165.9.1022
- Uchino, B. N. (2004). *Social support and physical health: Understanding the health consequences of relationships*. New Haven, CT: Yale University Press.
- Weinstein, M., Goldman, N., Hedley, A., Yu-Hsuan, L., & Seeman, T. (2003). Social linkages to biological markers of health among the elderly. *Journal of Biosocial Science*, 35, 433–453. doi:10.1017/S0021932003004334
- Wills, T. A. (1991). Social support and interpersonal relationships. In M. S. Clark (Ed.), *Prosocial behavior. Review of personality and social psychology* (Vol. 12, pp. 265–289). Thousand Oaks, CA: Sage Publications, Inc.

Received January 19, 2012

Revision received July 11, 2013

Accepted August 2, 2013 ■