SOCIAL SUPPORT AND AGE-RELATED DIFFERENCES IN CARDIOVASCULAR FUNCTION: AN EXAMINATION OF POTENTIAL MEDIATORS[1]

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ABSTRACT

The investigators examined the potential influence of social support on age-related differences in resting cardiovascular function and the potential mediators responsible for such associations in 67 normotensive women and men. Consistent with prior research, age predicted increased resting systolic blood pressure (SBP) and diastolic blood pressure (DBP). More importantly, regression analyses revealed that social support moderated age-related differences in resting SBP and DBP, as age predicted higher resting blood pressure for individuals low in social support, but was unrelated to blood pressure for individuals high in social support. An examination of potential pathways revealed that these results were not mediated by various health-related variables, personality factors, or psychological processes. Implications for the study of social support and health are discussed.

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INTRODUCTION

Chronological age is associated with reliable changes in physiological function that confers increased vulnerability to physical health problems (1). One interesting aspect of these age-associated changes is the interindividual variability that exists in biological function (2,3). One major challenge of modern aging theories is to explain these sources of heterogeneity, but many theories of aging focus on molecular, cellular, and/or systemic mechanisms at the biological level of analysis (4,5). The potential influence of social factors on such age-related differences in biological function has not been adequately examined (3,6). The major aims of this study were to examine the role of social support in predicting age-related differences in cardiovascular function and to test potential mediators responsible for these associations at differing levels of analysis (7).

Age-related differences in cardiovascular function are well-documented. For instance, research on chronological age and cardiovascular functioning suggests that age-related differences in the myocardium produce declines in cardiac performance to challenges (8,9). In addition, increasing age is associated with a decrease in the concentration (10) and sensitivity (11,12) of β-adrenergic receptors that further impact on myocardial performance. Resistance to blood flow also tends to increase (13,14). As a result, chronological age is typically associated with increases in resting systolic blood pressure (SBP) and diastolic blood pressure (DBP) (15–18). Importantly, cardiovascular disease risk increases in a linear fashion with rises in blood pressure (19). Thus, age-related differences in resting blood pressure may have important health implications even in normotensive populations.

Although age-related differences in cardiovascular function are well-documented, these differences are not isomorphic with chronological age, and variability exists between individuals in biological function (2,3,8,20). Psychosocial factors are often superimposed on chronological age; therefore, an examination of such factors may foster a more complete understanding of sources of variability in age-related biological differences. Of these psychosocial factors, there are important conceptual reasons to suspect that the quality of one's social relationships may be an important factor to consider. Social support has sufficient temporal stability to potentially influence disease states with a relatively long-term etiology. For instance, Sarason, Sarason, and Shearin (21) found that social support was stable over at least a 3-year period in a young adult population. Consistent with this notion, both the quality and quantity of one's social relationships have been associated with lower rates of morbidity and mortality (22–24).

Social relationships may also influence biological processes thought to underlie the development of physical health problems (25–28). In a recent review, Uchino, Cacioppo, and Kiecolt-Glaser (28) examined 81 studies that directly assessed the association between social support and aspects of the cardiovascular, endocrine, and immune systems. Social support was reliably related to beneficial effects on cardiovascular function (e.g. blood pressure regulation) in cross-sectional studies, intervention studies with both normotensives and hypertensives, and laboratory reactivity studies. In regards to endocrine function, social support appeared to be related to lower catecholamine levels. Finally, social support was consistently related to aspects of immune function, particularly in older adults. Importantly, the physiological systems reviewed by Uchino and colleagues may play important roles in the leading causes of death, including cardiovascular disorders, cancer, and infectious diseases.

In our earlier studies, we provided initial evidence that social support may moderate age-related differences in cardiovascular function (29,30). For instance, Uchino, Cacioppo, Malarkey, Glaser, and Kiecolt-Glaser (30) examined the role of social support on resting blood pressure in young (*M* age = 19) and older (*M* age = 67) adult women. Results revealed that age predicted increased resting blood pressure for individuals low in social support. In contrast, age did not predict resting blood pressure for individuals high in social support. Indeed, older adults high in support had comparable blood pressures to individuals who were on average 50 years younger.

An important issue raised by our earlier research concerns the potential mediators responsible for the associations between social

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TABLE 1
Sample Demographic Characteristics ($N = 67$)

Variable	Sample
Education	
Completed high school	100%
Partial college completion	98%
Completed college	43%
Partial graduate or professional school completion	21%
Completed graduate or professional school	12%
Yearly Income (Median)	$15,000 to $19,999
Ethnicity	
White	89%
Hispanic/Latino	3%
Asian/Pacific Islander	8%
Marital Status	
Single/never married	40%
Married/living with partner	36%
Divorced/separated	22%
Widowed	2%

support and age-related differences in cardiovascular function. An examination of potential pathways linking social support to health outcomes is considered a critical research agenda (7,28,31). In our prior research, such data were generally not examined. Therefore, an important aim of this research was to replicate our earlier results but also examine more precise health-related variables, personality factors, and psychological processes that could explain potential links between social support and age-associated differences in cardiovascular function.

There are possible behavioral pathways by which social support may predict age-related differences in cardiovascular function. For instance, the social control hypothesis suggests that social support may be beneficial because significant others exert pressures on close individuals to adopt healthier life-styles (32). Health-related variables such as body mass, exercise, or alcohol consumption may, in turn, influence blood pressure. However, consistent with past research indicating that such health-related variables are not the sole vehicles by which social support influences physical health (24,28,33), we predicted that the effects of social support on age-related differences in cardiovascular function will be evident while statistically controlling for these health-related variables.

There are also conceptually important personality and psychological factors by which social support might promote better health (7). For instance, personality factors such as neuroticism and extraversion may influence perceptions of support, whereas hostile individuals may elicit interpersonal transactions that impede effective social support (34,35). These personality processes may, in turn, influence resting cardiovascular function given evidence linking these factors to cardiovascular mortality (36,37). It is also possible that social support may promote better health due to decreased life stress or greater satisfaction with life (7), although less data exist linking these psychological processes to cardiovascular endpoints. By and large, elucidating such personologic and psychological pathways have presented some difficulty for the larger social support and physical health literature (28,33). However, in comparison to many prior studies (28) that have only examined a limited set of possible pathways, we examined a relatively wide range of personologic and psychological mediators. All of these measures have well-established psychometric properties that should increase our power to detect mediation.

METHOD

Participants

Thirty-six women and 31 men were recruited from the community to participate in a study of age-related differences in cardiovascular function (Age range = 20 to 70; M age = 38.3, $SD = 13.8$). Participants were paid $5.00 or they received psychology extra course credit for approximately 1 hour of participation. Consistent with our prior research, the following self-reported exclusion criteria were utilized for the present study: existing hypertension, cardiovascular prescription medication use, past history of chronic disease with a cardiovascular component (e.g. diabetes), past history of psychological disorder, tobacco use, and consumption of more than 10 alcoholic beverages a week. The sample demographic characteristics are detailed in Table 1.

Measures

Cardiovascular Assessments: A Dinamap Model 8100 Monitor (Critikon Corporation, Tampa, Florida) was used to measure SBP, DBP, and heart rate (HR). The Dinamap used the oscillometric method to estimate blood pressure. Prior studies have validated the Dinamap against both intra-arterial and standard ausculatory blood pressure assessments (38–42). Mean SBP, DBP, and HR were calculated by averaging across each assessment period to increase reliability (43). In the present study, mean SBP was 118.88 mm/Hg ($SD = 11.40$), mean DBP was 68.42 mm/Hg ($SD = 9.00$), and mean HR was 64.74 beats per minute ($SD = 9.58$).

Interpersonal Support Evaluation List (ISEL): The ISEL contained 40 questions and assessed total social support and the specific dimensions of appraisal, self-esteem, belonging, and tangible support. Cohen et al. (44) reported that the internal consistencies of the scales ranged from .60 to .92, with a 4-week test–retest reliability of .87 for the total scale. The reliability of the ISEL has also been established over a 6-month period (44). In the present study, the internal consistency of the total ISEL was .93 and the mean score was 2.27 ($SD = .43$).

Health Behavior Questionnaire (HBQ): A health behavior questionnaire provided information on the following potential health-related variables (45): average hours per week of exercise ($M = 3.85$, $SD = 3.73$); sleep ($M = 47.21$, $SD = 8.46$); alcohol consumption ($M = 1.24$, $SD = 2.82$); and caffeine intake ($M = 6.70$, $SD = 8.59$). Height and weight were measured using a Health-o-Meter scale from which body mass index (BMI) was calculated using the quetelet index, weight in kg / (height in meters)^2. Mean BMI in the present study was 25.65 ($SD = 6.21$).

Eysenck Personality Inventory (EPI): The short-form of the EPI was used to measure the independent personality dimensions of neuroticism and extraversion (46). As evidence of the reliability of the short-form, Eysenck (46) replicated the two-factor structure of the EPI and reported adequate split-half reliabilities for each scale. In addition, the 6-month test–retest reliability of the short-form EPI has been reported at .64 (47). The internal consistency for the neuroticism dimension was adequate in the current study (Chronbach's alpha = .79) and the mean score was 2.80 ($SD = 2.05$). In contrast, the internal consistency of the extraversion dimension was low (.40). As a result, analyses utilizing the extraversion dimension should be viewed as preliminary. The mean extraversion score in the present study was 3.64 ($SD = 1.45$).

Aggression Questionnaire (AQ): The 29-item AQ assessed the cognitive, affective, and behavioral aspects of trait aggression. Confirmatory factor analyses revealed evidence for the multidimensional structure of the AQ (48). The AQ has good 2-month test–retest reliabilities (*rs* = .72 to .80) and is characterized by adequate internal consistencies of .72 to .89 (48). Due to time constraints, we only assessed the Hostility subscale of the AQ ($M = 1.96$, $SD = .67$) that had an internal consistency comparable to prior research (.79).

Beck Depression Inventory (BDI): The BDI short-form contained 13 clinically derived items and appears to be a reliable and valid measure of depression (49,50). Beck et al. (49) reported a split-half correlation of .86 for the BDI. The internal consistency of the BDI short-form in the present study was good (.83) and the mean score was 4.96 ($SD = 5.05$).

Perceived Stress Scale (PSS): The PSS measured the extent to which individuals appraised their life to be stressful. Participants completed the 10-item version of the PSS that has been characterized by good internal consistency (i.e. alpha coefficient of .90 in the present study). In addition, the PSS is associated with increased stress as measured by similar measures, poorer health, and more health service utilization (51). In the current study, the mean score of the PSS was 1.72 ($SD = .73$).

Satisfaction With Life Scale (SWLS): The SWLS contained five items that assessed global life satisfaction (52). Diener and colleagues (52) reported an internal consistency of .87 and retest correlation of .82. The internal consistency of the SWLS in the current study was similarly high (.88) and the mean score was 4.34 ($SD = 1.5$). As evidence for the validity of the SWLS, individuals satisfied with their life were well-adjusted and relatively free of psychopathology (53).

Procedure

Potential participants were recruited via newspaper ads and flyers posted around the community. All participants were first screened by telephone according to the criteria detailed above. Qualifying individuals were scheduled for an appointment and upon arrival completed an informed consent document, demographic sheet, and health behavior questionnaire. Following completion of these questionnaires, the participant's height and weight were obtained using a standard medical scale.

The participant was then escorted to a sound-attenuated room and seated in a comfortable chair. Cardiovascular assessments were obtained by first measuring the participant's arm and positioning a properly sized adult occluding cuff on the upper left arm. In accord with manufacturer's specifications, the cuff was snug, with the transducer positioned over the artery. Either the senior author (B.N. Uchino) or the fourth author (R. Betancourt) performed the cuff placement procedure. The fourth author was trained in the cuff placement procedure by the senior author for a 1-week period and directly supervised for his first five participants. Accurate cuff placement minimizes error codes due to participant movement. It is worth noting that less than 4% of the obtained readings resulted in error messages, and most of these were due to excessive participant movement.

Following the blood pressure cuff placement, individuals were instructed to relax for the next 10 minutes while resting measures of cardiovascular function were obtained. A preliminary blood pressure reading was obtained at the beginning of the rest period simply to sensitize individuals to the assessment device.

TABLE 2

Results of Simultaneous Regression Analyses Examining the Prediction of Resting SBP and DBP by Age and Social Support

Variable	*B* (SBP)	*SE B*	β	*B* (DBP)	*SE B*	β
Gender	6.76	2.64	.30*	2.24	2.07	.12
Age (A)	.22	.10	.27*	.22	.07	.34**
Social Support (SS)	−2.12	3.03	−.08	−1.62	2.37	−.08
A × SS	−.47	.21	−.25*	−.44	.16	−.30**

Notes: Model $R^2 = .23$; adjusted $R^2 = .18$ ($p < .01$) for SBP. Model $R^2 = .24$; adjusted $R^2 = .20$ ($p < .01$) for DBP. * $p < .05$, ** $p < .01$.

Cardiovascular assessments of SBP, DBP, and HR were next obtained once per minute during the final 5 minutes of the resting assessment. The internal consistencies of these five resting assessments were high (Chronbach's alphas of .97 for SBP, .97 for DBP, and .99 for HR), suggesting good minute by minute measurement reliability. Thus, the 5-minute cardiovascular assessments were aggregated across minutes to increase reliability (43). Following the resting cardiovascular assessments, the participant completed the BDI, SWLS, PSS, EPI, and the Hostility subscale of the AQ. Participants were then compensated, debriefed, and thanked for their participation.

RESULTS

Preliminary Analyses

We first attempted to replicate prior research on age-related differences in resting cardiovascular function. In all analyses, an effect with a $p < .05$ was considered statistically significant. Consistent with prior research, age predicted increased resting SBP ($r = .30$, $p < .02$) and increased resting DBP ($r = .36$, $p < .01$). Also consistent with past research, no significant age-related difference in resting HR was found. Analyses also revealed that there were no significant three-way interactions involving age, social support, and gender. Therefore, all analyses reported were conducted statistically controlling for gender.

Does Social Support Predict Age-Related Differences in Cardiovascular Function?

To examine the prediction of age-related differences in cardiovascular function via social support, we utilized moderated regression procedures in which the main effect variables were centered (54). In these analyses, we entered the main effects of age and social support and the age × social support cross-products. As shown in Table 2, replicating our prior research social support moderated age-related differences in resting SBP ($\beta = -.25$, $p = .03$) and resting DBP ($\beta = -.30$, $p = .01$). The effect sizes for these age × social support interactions were small to moderate but nevertheless reliable (55).

The pattern of these statistical interactions were depicted by graphing the predicted values using scores one standard deviation above and below the means (54). Simple slope *t*-test analyses were also conducted (54). As depicted in Figures 1 and 2, age predicted increased resting SBP ($t = 3.30$, $p < .01$) and DBP ($t = 4.11$, $p < .01$) for individuals low in social support. In contrast, individuals high in social support had relatively low and comparable SBP and DBP as a function of age (*ts* < .5, n.s.).

Are Demographic Factors Mediators of the Social Support and Age-Related Blood Pressure Link?

Before we examined the mediators of main conceptual interest, we first analyzed whether the sample demographic factors

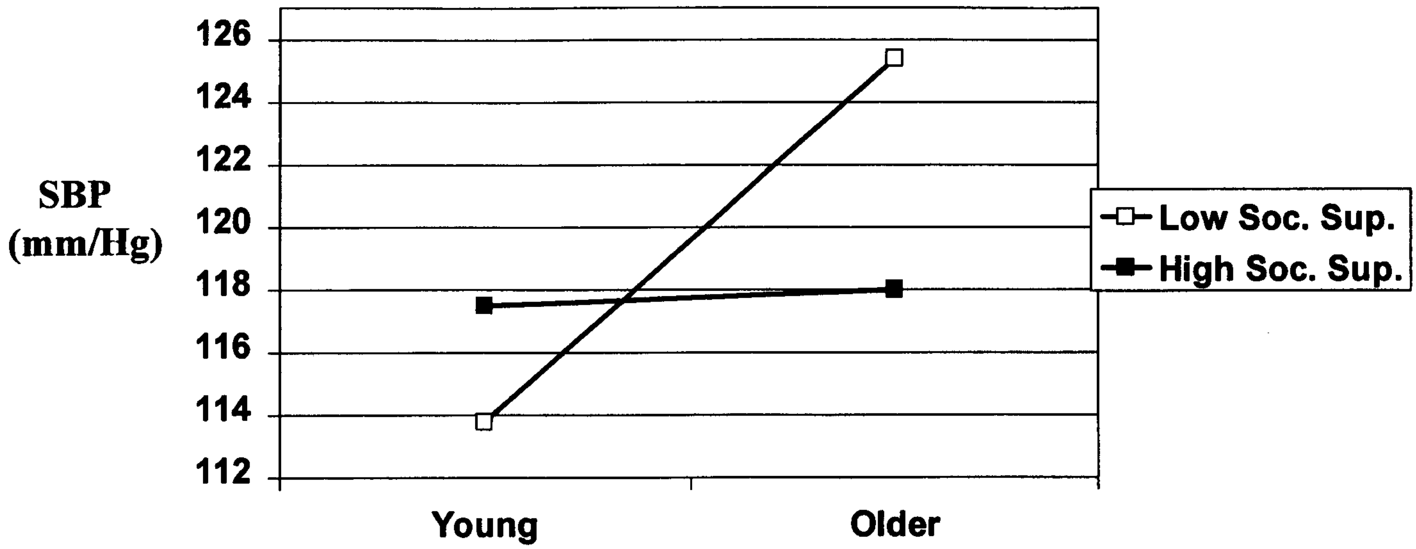

FIGURE 1: Predicted SBP as a function of social support and chronological age (one *SD* above and below the mean).

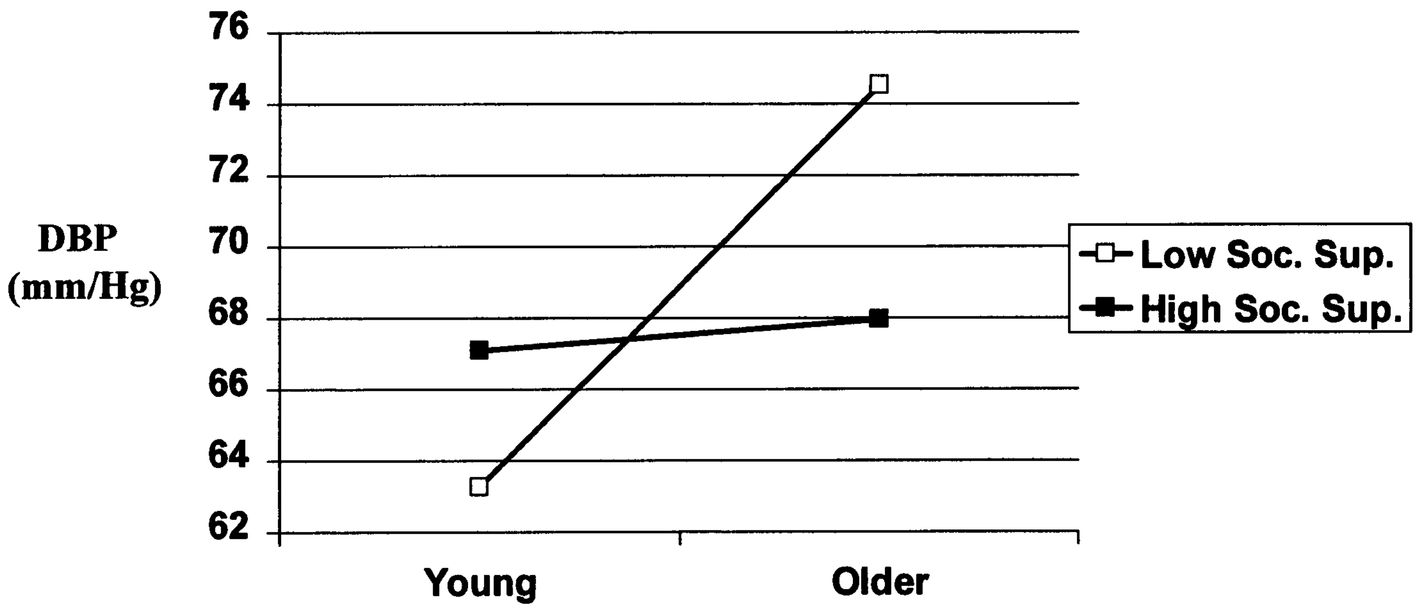

FIGURE 2: Predicted DBP as a function of social support and chronological age (one *SD* above and below the mean).

of income, marital status, and educational status were potentially responsible for the results reported above. These analyses were important due to evidence linking such factors to cardiovascular disease risk (56). According to Baron and Kenny (57), three conditions are needed to establish mediation once an association has been documented. First, the independent variable (IV) should be related to the proposed mediator(s). Second, the proposed mediator should be associated with the dependent variable (DV) while statistically controlling for the IV. Finally, the association between the IV and DV should be reduced when statistically controlling for the proposed mediator(s). Relatively strong evidence for mediation is present when a previously significant association is rendered nonsignificant. Evidence for partial mediation may be obtained if the association remains significant but the regression weight is reduced in magnitude (57).

According to the guidelines provided by Baron and Kenny (57), if these demographic variables were major pathways responsible for the age × social support interactions, then statistically controlling for these variables should render the interactions nonsignificant or decrease the relative size of the β weights. We examined these issues as a first step to demonstrating mediation. Prior to these analyses (and all subsequent analyses reported below), the main effect variables were first centered before inclusion into the model (54). Results of these simultaneous regression analyses revealed that statistically controlling for these demographic factors did not influence the age × social support interactions for either SBP ($\beta = -.25, p = .04$) or DBP ($\beta = -.33$, $p = .01$). Comparison of β weights between the original (unmediated) statistical interaction (see Table 2) with these analyses if anything revealed a stronger statistical interaction between age and

TABLE 3

Results of Simultaneous Regression Analyses Examining the Influence of Health-Related Variables on the Age × Social Support Interactions (N = 67)

Variable	B (SBP)	SE B	β	B (DBP)	SE B	β
BMI	.65	.20	.35**	.53	.16	.37**
Average Sleep	.20	.14	.14	.12	.12	.11
Average Exercise	−.04	.34	−.01	−.18	.29	−.07
Average Caffeine	.34	.14	.26*	.09	.12	.08
Alcohol	−.00	.46	−.00	.25	.39	.08
Gender	7.53	2.55	.33**	3.01	2.12	.17
Age (A)	.15	.09	.18	.17	.08	.26*
Social Support (SS)	.12	2.79	.00	−.45	2.31	−.02
A × SS	−.52	.19	−.28**	−.49	.16	−.34**

Notes: Model $R^2 = .46$; adjusted $R^2 = .37$ ($p < .001$) for SBP. Model $R^2 = .42$; adjusted $R^2 = .32$ ($p < .001$) for DBP. * $p < .05$, ** $p < .01$.

social support for DBP when controlling for these demographic factors.

Are Health-Related Variables Mediators of the Social Support and Age-Related Blood Pressure Link?

The first conceptual set of mediational variables examined the possibility that health-related variables might be pathways by which social support moderates age-related differences in blood pressure. Therefore, we repeated our analyses reported above while statistically controlling for BMI, average hours of sleep per week, average hours of exercise per week, average caffeine consumption per week, and average alcohol consumption per week. Results of these simultaneous regression analyses shown in Table 3 revealed that none of the significant interactions involving age and social support were altered by statistically controlling for these health-related variables. In fact, the significance levels decreased and the β weights increased in the age × social support interactions for SBP and DBP. All else being equal, it should be noted that the inclusion of proposed mediators into the model could result in a small decrease in the age × social support interaction independent of any conceptual mediation due to the resulting decrease in degrees of freedom. However, this simple statistical explanation cannot account for the results observed for these mediational analyses.

It is also important to note that in these simultaneous regression analyses, BMI was an independent predictor of greater SBP ($p < .01$) and DBP ($p < .01$). Similar results were found for average caffeine consumption in predicting SBP ($p < .02$). Therefore, we were able to replicate prior research involving some of these health-related variables suggesting adequate measurement sensitivity.

Are Personality Factors or Psychological Processes Mediators of the Social Support and Age-Related Blood Pressure Link?

We next examined the possibility that personality factors might be responsible for our findings involving social support. To test these hypotheses, we examined the age × social support interactions while statistically controlling for extraversion, neuroticism, and hostility. As shown in Table 4, these simultaneous regression analyses revealed small influences on the age × social support interactions for both SBP ($p = .06$) and DBP ($p = .03$). The slight decrease in significance levels is consistent with the small reduction in the β weights. Further analyses (not depicted) revealed that neuroticism was the sole component responsible for the decreased significance level of the age × social support interaction. However, an additional but necessary demonstration of mediation would require that the proposed mediator independently predict blood pressure. Results revealed that neuroticism did not significantly predict blood pressure in these analyses (*ps* > .23).

A final conceptual issue in this study relates to more specific psychological pathways by which social support may moderate age-related differences in cardiovascular function. We thus examined the age × social support interactions for SBP and DBP while statistically controlling for satisfaction with life, perceived stress, and depression. None of the analyses were appreciably altered by these statistical controls, suggesting little psychological mediation via these factors (see Table 5).

TABLE 4

Results of Simultaneous Regression Analyses Examining the Influence of Personality Factors on the Age × Social Support Interactions (N = 67)

Variable	B (SBP)	SE B	β	B (DBP)	SE B	β
Neuroticism	−.76	.72	−.14	−.68	.56	−.16
Extraversion	.16	.94	.02	.38	.74	.06
Hostility	3.90	2.22	.23	2.66	1.75	.20
Gender	6.03	2.76	.27*	1.47	2.17	.08
Age (A)	.25	.10	.30*	.23	.08	.36**
Social Support (SS)	−1.03	3.45	−.04	−1.31	2.71	−.06
A × SS	−.43	.23	−.23	−.39	.18	−.27*

Notes: Model $R^2 = .27$; adjusted $R^2 = .19$ ($p < .01$) for SBP. Model $R^2 = .28$; adjusted $R^2 = .20$ ($p < .01$) for DBP. * $p < .05$, ** $p < .01$.

TABLE 5

Results of Simultaneous Regression Analyses Examining the Influence of Psychological Processes on the Age × Social Support Interactions (N = 67)

Variable	B (SBP)	SE B	β	B (DBP)	SE B	β
Perceived Stress	−1.56	2.89	−.10	−.64	2.23	−.05
Depression	.14	.39	.06	−.29	.30	−.16
Life Satisfaction	−.75	1.36	−.10	−1.59	1.05	−.26
Gender	7.21	2.96	.32*	2.74	2.28	.15
Age (A)	.24	.10	.29*	.23	.08	.34**
Social Support (SS)	−1.36	3.72	−.05	−.84	2.86	−.04
A × SS	−.46	.22	−.25*	−.39	.17	−.27*

Notes: Model $R^2 = .24$; adjusted $R^2 = .15$ ($p < .05$) for SBP. Model $R^2 = .28$; adjusted $R^2 = .19$ ($p < .01$) for DBP. * $p < .05$, ** $p < .01$.

DISCUSSION

Aging is associated with important changes in physiological function that confers increased vulnerability to physical health problems. The data reported in this study suggest that these changes may not be isomorphic with chronological age and social processes may be important factors to consider in research on age-related differences in biological function (2). We should emphasize that the associations between age, social support, and blood pressure appear reliable (29,30). Our initial analyses also revealed that the pattern of results did not differ as a function of gender. However, due to the homogeneous ethnic composition of our sample, future research would be needed to demonstrate the generalizability of our findings across diverse ethnic groups.

One interesting question relates to the approximate age range in which social support was predictive of resting blood pressure. Our moderate sample size precluded definitive analyses as a function of discrete age categories. We nevertheless conducted ancillary analyses to investigate this question by examining the prediction of blood pressure via social support at differing age ranges. For individuals between the ages of 20 to 40 ($n = 38$), social support does not predict resting blood pressure ($r = -.01$ for SBP, $r = .04$ for DBP). However, social support appears to be a progressively stronger predictor of resting blood pressure in the older groups. For instance, social support predicts lower SBP ($r = -.23$) and DBP ($r = -.33$) for individuals over 40 ($n = 29$). In persons over 50 ($n = 15$), social support is associated with lower SBP ($r = -.50$) and DBP ($r = -.44$). Future research using large samples across broad age ranges will be necessary to adequately address this question.

An important goal of the present study, and social support research more generally, is the identification of mediators responsible for the health effects of social relationships. For instance, Cohen, Doyle, Skoner, Rabin, and Gwaltney (33) exposed individuals to the common cold virus and quarantined them for 5 days. Results revealed that individuals with more diverse social networks were less likely to be biologically infected by the common cold virus. However, statistical controls for health-related variables and personality processes revealed that these factors were not mediators of the association between social ties and infections. Similar to Cohen and colleagues (33), we found little evidence for the pathways responsible for the associations between social support, age, and blood pressure despite the well-documented psychometric properties of many of our measures. These data are consistent with a growing number of studies that have been unable to characterize the potential pathways responsible for the health-related effects of social relationships (24,28,58,59).

There are several important issues we should discuss that may have influenced the mediational analyses in this study. We suspect that these issues also have implications for examining statistical mediation in the larger social support and physical health literature. One important issue relates to the psychometric properties of our health behavior assessments. The reliability of these measures may be limited due to the single-item assessments. Despite this limitation, we were still able to detect significant associations between some of our health-related variables and resting blood pressure, thus suggesting adequate sensitivity. In addition, the BMI represents, in part, the cumulative effects of many health-related practices (e.g. diet). However, the model that included statistical controls for BMI did not alter the prediction of age-related differences in blood pressure via social support. Nevertheless, future research examining such health behaviors as potential pathways will need to consider the psychometric properties of their measures. In addition, physiologic assessments of health status thought to reflect health behaviors (e.g. serum albumin as a nutritional marker) may provide converging data for these assessments.

An examination of psychological mechanisms also revealed little evidence for the pathways responsible for our findings. According to Kenney, "Aging may be defined as the sum of all changes that occur . . . with the passage of time and lead to functional impairment and death" (60, p. 15). If these cross-sectional results indeed represent the cumulative long-term influence of having a strong social support network, then the temporal stability of these psychological mechanisms need to be measured at a comparable level. Ultimately, longitudinal designs that measure both biological function and psychosocial processes over an appreciable period of time may be necessary to adequately examine such questions.

Although our measures of personality factors and psychological processes were well-validated, we only examined a limited set of such factors. A large literature has documented psychological antecedents and consequences of social support (61). For instance, Rowe and Kahn (2) suggest that autonomy and personal control may be other important factors predicting age-associated differences in physiologic function. Rowe and Kahn (2) also draw the intriguing distinction between social support that enhances autonomy and social support that decreases autonomy. At least in Western cultures, social support that enhances autonomy may be a more effective form of social support. Future studies should examine an expanded set of conceptually relevant personality and psychological pathways based on the larger social support literature.

There are several additional limitations of the present study that need to be discussed. We utilized a cross-sectional design, so whether or not these data would translate to age-related changes cannot be determined. As noted above, well-designed longitudinal studies may be necessary to evaluate this question. In addition, although comparable to studies examining the physiological processes associated with social support, the present sample size was not large. It should be noted, however, that the relatively small sample size does not appear to provide a simple explanation for our reported lack of mediation. As noted earlier, the inclusion of mediators into the model could decrease the age $\times$ social support interaction and masquerade as mediation simply due to the resulting loss in degrees of freedom, especially in smaller samples. Of course, this reasoning does not explain why we failed to find evidence for mediation in the present study. Future studies that utilize larger samples would be beneficial to allow for more complex modeling procedures (e.g. interactive models using covariance structural modeling). The exact conceptual nature of these more complex models, however, is presently beyond the scope of the existing social support and physical health literature.

We also limited the present investigation to a normotensive population; therefore, the immediate biological significance of these findings is unclear. However, cardiovascular disease risk is related to blood pressure in a linear fashion, with no clear threshold effect within the normal range for DBP (19). These data have led some researchers to suggest that normotensives should also be included in clinical trials involving antihypertensive therapy (62). Based on these findings, one might speculate that without intervention, these middle-aged to older adult individuals low in social support may be at increased cardiovascular risk. It is hoped that the future identification of mediators may help to guide conceptually based interventions aimed at utilizing social relationships to promote positive health outcomes.

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