Gender, Social Support, and Cardiovascular Responses to Stress

Laura M. Glynn, MA, Nicholas Christenfeld, PhD, and William Gerin, PhD

Objective: Laboratory research indicates that the presence of a supportive other can reduce physiological responses to a stressor. Whether there are gender differences, either on the part of the provider or the recipient, in this social support effect is explored. Such differences might shed some light on the frequent epidemiological reports of gender differences in social support and health. **Methods:** Male and female subjects gave an impromptu speech and received either standardized supportive or nonsupportive feedback from a male or female confederate. Blood pressure and heart rate were monitored continuously during baseline and speech periods. **Results:** Speakers with a supportive female audience showed a systolic increase of 25 mm Hg over baseline. Those with a nonsupportive female audience increased 36 mm Hg. A supportive male audience led to increases of 32 mm Hg, and a nonsupportive male audience 28 mm Hg. There was no significant effect of gender of subject. **Conclusions:** Results indicate that social support provided by women reduced cardiovascular changes for both male and female speakers compared with presence of a nonsupportive female audience. Social support from men did not. These findings suggest a possible mechanism that might help explain the epidemiological literature on the relationship between gender, social support, and health. The findings are consistent with the notion that married men are healthier because they marry women. Women do not profit as much from marriage or suffer as much from separation, in terms of health outcomes, because the support they gain or lose is the less effective support of a man. These findings render more plausible the possibility that differences in social support might contribute to health differences, through the dampening of cardiovascular responses to stress. **Key words:** social support, cardiovascular reactivity, gender, blood pressure, stress.

BPM = beats per minute.

INTRODUCTION

There is a substantial body of evidence demonstrating a relationship between social support and a variety of health outcomes. In general, individuals who have access to social networks, or feel less lonely, are healthier than their counterparts who lack these social ties (1–7). The positive effects of social support are associated with all-cause mortality, including the reduction of risk for cardiovascular disease (8–11).

Although associations between social support and health outcomes are fairly well established, existing research provides only limited insight concerning the mechanisms of this effect. One possibility is that social support directly affects physiological responses to stress (12, 13). Because exaggerated reactivity in response to laboratory stressors has been linked to hypertension and coronary heart disease (14–16), the dampening of such responses may provide one avenue through which social support operates. Several studies have shown that the presence of a supportive other can attenuate physiological responses in individuals facing a stressor (12, 13, 17–22), although not all studies find this effect (23, 24).

Although these studies demonstrate the positive effects of social support on cardiovascular outcomes, they do not address whether the effects of social support depend just on the supportive behaviors or also on the actor's construal of the meaning of these behaviors. Two factors that could alter the effectiveness of supportive behaviors are the nature of the recipient and the nature of the source.

Focusing on the nature of the recipient, Lepore (25) has shown that high cynicism individuals do not benefit from social support in terms of their cardiovascular responses to stress. However, low cynicism subjects exhibited smaller responses with supportive feedback than with neutral feedback. This finding provides one piece of evidence suggesting that subjects' construal of the situation influences the effectiveness of the social support.

Christenfeld et al. (22) examined whether the nature of the source makes a difference by comparing support offered by a friend to support offered by a stranger. Subjects gave a speech to a supportive friend, a supportive stranger, or a neutral stranger. The findings indicated that the supportive behaviors from a friend resulted in smaller cardiovascular responses than the same supportive behaviors offered by a stranger, which resulted in smaller responses than neutral feedback from a stranger. As with the Lepore (25) study, this suggests that not all supportive behaviors have the same effect—in this case, it matters who offers the support.

The finding that nature of the source of the support

From the Department of Psychology (L.M.G., N.C.), University of California-San Diego, La Jolla, California; and Cardiovascular Center, Cornell University Medical College-The New York Hospital (W.G.), New York, New York.

Address reprint requests to: Nicholas Christenfeld, PhD, Department of Psychology, 9500 Gilman Dr., University of California-San Diego, La Jolla, CA 92093-0109. E:mail: nicko@ucsd.edu

Received for publication July 2, 1997; revision received October 6, 1998.

0033-3174/99/6102-0234

Copyright © 1999 by the American Psychosomatic Society

can influence its effectiveness hints that other differences in the source might matter as well. One candidate is gender. Do supportive behaviors offered by a woman produce a different effect from the same behaviors coming from a man? There are various reasons to think this may be the case. Not only do gender differences pervade the psychological literature (26–29), but these differences also exist in the areas directly relevant to social support. In the epidemiological vein, there is evidence that marriage provides health benefits for men. Wiklund et al. (30), for example, have found that the estimation of risk of death for single men who have suffered a first myocardial infarction is roughly double that of their married counterparts. The studies that have examined the relationship between marriage and health for both genders have found for women either that the effects are substantially smaller than those for the men, or, more commonly, that there is no relationship. Berkman and Syme (1) reported that the relative risk ratio of premature mortality for unmarried compared with married men ranged from 2.1 to 2.9, depending on age, whereas the risk ratio was 1.4 for women. Similarly, House et al. (2) found a relative risk ratio for men of 1.9 and the corresponding ratio for women was 1.07.

Paralleling the research on the benefits of marriage, the epidemiological research on the effects of bereavement and widowhood suggests that the loss of a spouse seems to have more negative outcomes for men than it does for women (31). Helsing et al. (32) compared widowed with married individuals and found that the adjusted mortality rate (per 1000 person-years) for married men was 65.3 whereas the rate for widowed men was 51.8, demonstrating the detrimental effects of bereavement for men. For women, there was no corresponding negative effect of widowhood. The adjusted mortality rate (per 1000 person-years) for married women was 24.1 and for widowed women it was 23.2. Similarly, Martikainen and Valkonen (33) found effects of bereavement across a wide range of causes of death, and found that these effects are greater for men than for women.

Other than the work on marriage, the epidemiological evidence on the benefits of social support do not reveal clear gender patterns. People with larger social networks seem to live longer, healthier lives (4). Sometimes this effect is larger for women (1) and sometimes it is larger for men (2), but, overall, the gender findings are inconsistent (3, 34, 35). However, these data do not generally address the gender composition of the social networks, and so the source and the recipient effects could easily be confounded or canceled.

The epidemiological literature on marriage and bereavement provides evidence that the effects of social support differ with respect to gender. There are at least two reasons why it is difficult to draw conclusions about these gender differences. First, the social support behaviors that accompany marriage may differ depending on gender. Without experimental control or even reports from the subjects, it is impossible to rule out this possibility. Men may benefit more simply because wives are more supportive than husbands. Second, even if it were the case that husbands and wives were matched on the amount and type of support offered, without the fully crossed gender design, it is not possible to determine why this difference exists. It may be that women are unable to benefit from support, that men are unable to provide effective support, or that this is an interactive phenomenon.

Although the epidemiological evidence cannot directly address the exact nature of the gender difference, laboratory experiments may begin to shed some light on the issue. Epidemiological evidence indicates that people with better social support are healthier. The laboratory studies indicate that social support can reduce cardiovascular responses to stress. The laboratory social support paradigm thus illustrates one possible avenue through which social support can exert its beneficial effect—the dampening of cardiovascular responses. The epidemiological evidence also indicates that there are reliable gender differences in the effects of marriage. Are there, then, gender differences in the laboratory social support paradigm that could suggest a mechanism for the observed gender differences in marriage? That is, does the pattern of gender differences in cardiovascular responses to social support mirror the pattern of health benefits in marriage?

If gender differences in cardiovascular responses to social support were found, they would not necessarily be the mechanism underlying marriage effects any more than the social support and cardiovascular response findings must be the mechanism for general social support effects. However, finding a pattern of gender differences in laboratory social support that corresponds to those in the real world would render this mechanism more plausible. If these gender differences do not appear in the laboratory, it would tentatively suggest that patterns of cardiovascular responses to stress do not contribute to the observed epidemiological gender differences in social support. It is possible, of course, that reduced cardiovascular responses might still be implicated in the general social support effects, but might not be a mechanism underlying the gender differences.

A laboratory experiment conducted by Kirschbaum et al. (36) found a pattern of gender differences in physiological responses to social support that is consistent with the epidemiological findings on marriage.

Men who had their female live-in partner providing support during the anticipation of a stressor exhibited lower cortisol levels than those who did not. In contrast, for the women, there was no benefit of having their male live-in partner present during the anticipation of a stressor. This provides additional evidence for the existence of the gender difference but provides little insight into the source of the difference—the support behaviors were not standardized and this was not a fully crossed gender design.

In the domain of the cardiovascular response paradigm, the effects of gender on social support are largely unknown because the bulk of the work in this area has used only female subjects (12, 13, 19, 20, 22, 37, 38). Two experiments studied both men and women (18, 25), and, using same-sex dyads in a social support paradigm, failed to detect any significant effects of gender on the social-support effect. As discussed earlier, two other experiments, also using same-sex dyads, failed to uncover social support effects for either male or female pairs (23, 24). There is work outside the laboratory social support paradigm that documents gender differences in cardiovascular responses to social interaction (39–41), but this work, for the most part, serves to provide more evidence for gender differences rather than to answer questions about the mechanisms that may produce these differences. To date, we are not aware of a social support and blood pressure study that has used a fully crossed gender design.

The question raised by the epidemiological evidence is not satisfactorily resolved: Are the observed patterns due to a gender difference in the recipient of the support, a gender difference in the one offering the support, or an interaction of these two? The epidemiological findings suggest three plausible patterns of gender differences in social support: that women do not benefit from support, in terms of cardiovascular responses; that men do not provide effective support; or, more specifically, that men do not provide effective support to women. We are not suggesting that these are the only possible patterns, or that one of these patterns must emerge, but just that these three are all consistent with the epidemiological evidence, and thus worth special attention.

The present study is designed to explore whether there are gender differences in laboratory social support and whether these patterns are consistent with the epidemiological findings. To investigate this, we use a fully crossed gender design. A confederate, male or female, gave standardized supportive or nonsupportive feedback to a subject, male or female, who gave an impromptu speech.

METHODS

Overview

The study used a support (support/nonsupport) by gender of subject (female/male) by gender of observer (female/male) factorial design. Subjects gave a 5-minute impromptu speech to one observer, a confederate of the experimenter. The observer behaved in either a supportive or nonsupportive manner. Subjects' blood pressure and heart rate were continuously monitored during baseline and speech periods.

Subjects

Participants in the study were 109 undergraduates at a large university. The experiment included subjects from 18 to 25 years old (mean = 19.7 years), and there were 57 women and 52 men. All were normotensive (resting blood pressure $< 140/90$ mm Hg), and none reported either being in poor health or taking any medications that might affect cardiovascular measurements. Subjects received course credit in exchange for participation.

Recording of Physiological Measures

Systolic and diastolic pressures, as well as heart rate were collected using an Ohmeda Finapres 2300 blood pressure monitor, which takes beat-to-beat pressures in a noninvasive manner, using the Peñaz method. This method uses a finger cuff, worn on the third finger of the nondominant hand. The Finapres has been demonstrated to be a useful alternative to intraarterial blood pressure measurement in laboratory testing (42), as well as in clinical practice (43, 44). In addition, it has been shown to track intraarterial readings extremely well, even during sudden changes of blood pressure (45), making it a good candidate for use during reactivity testing. The Finapres collects a large number of readings, enhancing reliability (46).

Manipulation of Social Support

Confederates serving as the audience were trained to behave either supportively or nonsupportively, depending on the condition. In the support conditions, the confederate wished the subject good luck at the beginning of the speech, and then sat attentively throughout the speech with an upright but relaxed posture, and with arms at his or her side. The confederate nodded in agreement with statements made by the subject about every 30 seconds. In addition, twice during the course of the 5-minute speech, the confederate murmured "good point" in response to a statement made by the subject. The confederate also smiled when the subject smiled, and laughed if the subject initiated laughter.

In the nonsupportive condition, the confederate did not provide the subject with any positive feedback. The confederate's facial expressions were completely neutral, and his or her demeanor was generally inattentive. The confederate in this condition sat throughout the speech slouching, and with arms crossed in front of him or her. If the subject initiated smiles or laughter, the confederate did not respond.

Nine different undergraduate assistants served as confederates. Five of these were women, and four men. All were rigorously trained to act in exactly the same fashion during the speech. The timing and content of all utterances and nonverbal behaviors were standardized.

Procedure

Male and female subjects were randomly assigned to receive either supportive or nonsupportive feedback from either a man or a woman. Shortly after arriving at the laboratory, the subject was joined by a confederate who acted as though he or she was also there to participate in the session. The experimenter explained to the subject and confederate that they would take turns performing a task while their blood pressure and heart rate were being monitored. No allusion was made at this time as to the nature of the task. Subjects read and signed a consent form, which indicated that they might be asked to perform one of a long list of tasks, and indicated that they might be tape-recorded.

After the description of the experiment, cards were drawn to determine who would perform the task first. The cards were rigged so that the real subject always went first. The subject then had the blood pressure cuff applied, and a 10-minute baseline was taken. The subject was asked to sit quietly and not move around during the baseline period. The experimenter and the confederate both remained in the room during this period, reading quietly and not looking at the subject.

After the baseline period, the subject and the confederate were informed that they would each be giving a 5-minute impromptu persuasive speech, but would not know each other's topic until the beginning of each speech. The confederate was then asked to leave the room, so that the experimenter could reveal to the subject the topic of his or her speech. After the confederate left, the subject was advised that he or she would have 6 minutes to prepare a speech on euthanasia. The subject could choose whether to take the pro or con position, and was provided with a list of arguments on both sides of the issue to help him or her to speak for the entire 5-minute speech period. The experimenter answered any questions the subject asked, and then left the room under the pretense that she was going to tell the other subject his or her topic. It was at this time that the experimenter informed the confederate whether he or she would be providing supportive or nonsupportive feedback. In this way, the confederate was kept blind to the support manipulation during baseline.

Approximately 2 minutes later, the experimenter returned and sat quietly reading until the end of the 6-minute preparation period. At this time, the subject was informed that his or her speech would be tape-recorded, and that the experimenter would not be present during the 5-minute speech period. The experimenter then placed the tape-recorder on a table in front of the seated subject. The experimenter fetched the confederate, and seated him or her across from the subject. The experimenter turned on the tape-recorder, told the subject to begin the speech, and left the room.

At the end of 5 minutes, the experimenter reentered the room, removed the blood pressure cuff from the subject's finger, and fitted the cuff to the confederate's finger, to maintain the illusion that it was now the confederate's turn to give the speech. The confederate then began to sit through a sham baseline. During this period, the subject completed a postsession questionnaire. On completion of the questionnaire, the experiment was ended, and the subject was debriefed and thanked for his or her participation.

Speech Performance

To check that the support manipulation did not affect the amount of talking, the speeches were tape-recorded. Using the recordings, the amount of time each subject spent talking during the 5-minute speech period was timed, and the durations compared across the experimental conditions. Ten of the speeches were timed by two people to check the reliability of the coding.

Subjective Measures

To check the effectiveness of the social support manipulation, subjects rated the audience on the following series of 7-point Likert scales: friendly-unfriendly, supportive-unsupportive, accepting-rejecting, close-distant, warm-cold, and helpful-unhelpful.

In the postsession questionnaire, subject's appraisals of stress during the speech were obtained, using a series of 7-point Likert scales for the dimensions: stressed-relaxed, nervous-not nervous, angry-not angry, uncomfortable-comfortable, annoyed-not annoyed, anxious-not anxious, excited-calm, and worried-content.

Data Reduction and Analysis Procedures

Three cardiovascular measures were examined: systolic blood pressure, diastolic blood pressure, and heart rate. The cardiovascular dependent measures were change scores, computed using the difference between the mean of the speech task, and the mean of the pretask baseline measurements. These means were computed using the pulse-based technique (47). It has been shown that a 10-minute baseline, taken with the Finapres, allows enough measurements to be taken to provide excellent reliability (46).

The systolic blood pressure change scores constituted the primary physiological dependent measure, inasmuch as these have been shown to be more sensitive to interpersonal manipulations than other cardiovascular measures (12, 18, 22, 35, 36, 48), and are the most reliable change scores when assessed using the Finapres (49). Diastolic and heart rate change scores were also examined. Raw change scores rather than residualized change scores were used as recommended by Llabre et al. (50).

RESULTS

Manipulation Check

The subjects' answers to each Likert question rating the confederates' support during the speech were averaged into an overall support index score. This score showed high internal consistency, with Cronbach's $\alpha = .94$. Analysis of the subjects' ratings on the social support scale revealed that the social support manipulation was effective. The mean rating for the nonsupportive feedback was 2.7, whereas the mean in the support condition was 4.5, $F(1,99) = 94.86$, $p < .0001$. (It is also the case that each individual item showed a significant effect of the support manipulation, with all p values $< .0001$). There was a slight tendency for female confederates to be rated as more supportive, regardless of whether they were offering support or not. Female confederates received an average index score of 3.87, whereas males received a score of 3.41, $F(1,99) = 7.43$, $p < .01$. However, there was no sign that the difference between support and nonsupport varied by the gender of the confederate, $F(1,99) = 0.17$, NS.

Duration of Speech

The coding of how much time was spent speaking achieved satisfactory reliability, $r\ (9) = .83$. On average, the speakers spent 236 seconds actually talking (of the 300-second speech period). There were no significant effects of the experimental conditions on speaking time (all p values $> .2$).

Baseline Measures

There were no significant effects of the support manipulation or of audience gender on resting levels of any of the three physiological measures. Main effects of the gender of the subject for both systolic and diastolic blood pressure, $F(1,101) = 6.94$, $p < .05$; $F(1,101) = 8.16$, $p < .05$, respectively, indicated that men had higher resting blood pressure than women. Men averaged 133.9/81.7 and women averaged 125.8/76.6 mm Hg. There was no significant effect of subject gender on resting heart rate, with a mean of 77.4 BPM for men and 76.6 BPM for women.

Effect of Experimental Conditions on Blood Pressure and Heart Rate Changes

Systolic Blood Pressure. The physiological change scores were analyzed using a three-factor analysis of variance (ANOVA), with gender of the audience, gender of the speaker, and support as the factors. For systolic blood pressure, the main effect of gender of the speaker was not significant, $F(1,101) = 2.52$, $p = .11$, although there was a slight tendency for men to exhibit greater blood pressure responses than women (33.2 mm Hg and 28.0 mm Hg, respectively). There was no main effect for the gender of the audience, $F(1,101) = 0.00$, NS, nor was there a main effect of support, $F(1,101) = 0.66$, NS.

There was a significant interaction of audience gender and support, $F(1,101) = 4.82$, $p < .05$. The blood pressure changes from supportive and nonsupportive male and female audiences are shown in Figure 1. Breaking the interaction down into simple effects by

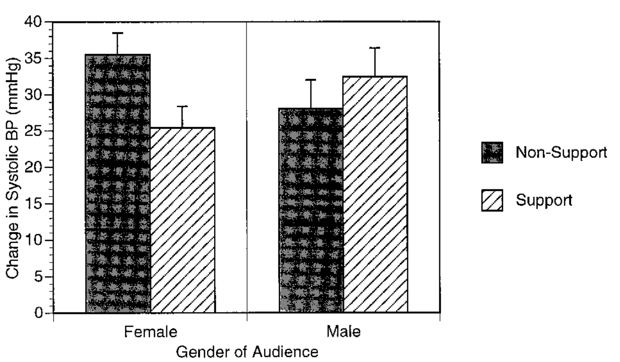

Fig. 1. The effects of support and nonsupport from a male or female audience on systolic blood pressure change from baseline (collapsed across gender of speaker).

audience gender reveals that social support from a female confederate resulted in lower arousal levels than nonsupport, $F(1,101) = 4.85$, $p < .05$. Support from a male confederate, however, compared with nonsupport from a male, produced no differences in the blood pressure responses, $F(1,101) = 0.92$, NS. With male confederates, not only did the supportive condition fail to result in lower blood pressure responses, but there was a slight tendency in the opposite direction.

The interaction of audience gender and support can also be broken down into simple effects by support. That is, one can examine whether the interaction is due to support from women producing lower systolic blood pressure than support from men, or is due to nonsupport from women producing higher blood pressure than nonsupport from men. Neither of these simple effects was significant ($F(1,101) = 2.70$, $p = .10$; $F(1,101) = 2.15$, $p = .15$), suggesting that the interaction was a combination of both effects.

There was no sign of an interaction between the gender of the speaker and support, $F(1,101) = 0.87$, NS). There was, however, a marginally significant three-way interaction, $F(1,101) = 3.62$, $p = .06$. The means for all eight cells are shown in Table 1. Whereas social support from a woman compared with nonsupport reduced blood pressure responses in both men and women, support from a man compared with nonsupport raised blood pressure in men, and had no effect on women. This backwards social support effect in the male dyads, which was not consistent with the prior research or the epidemiological literature, was not significant when using a Bonferroni correction to control the Type I error rate.

Diastolic Blood Pressure. Diastolic blood pressure changes exhibited the same pattern as the systolic changes. There were no main effects of subject gender, $F(1,101) = 0.06$, NS, or of audience gender, $F(1,101) = 1.79$ NS). Nor was there a main effect of support condition, $F(1,101) = 1.03$, NS. As with the systolic responses, the interaction between audience gender and support was significant, $F(1,101) = 8.23$, $p < .01$. Once again, an analysis of the simple effects by audience gender revealed that support from female confederates produced smaller responses than did nonsupport from female confederates, $F(1,101) = 7.64$, $p < .01$. Support from a male, however, did not alter the diastolic blood pressure response, $F(1,101) = 1.71$, NS. Looking at the simple effects by support condition reveals that those receiving support from a female audience had lower diastolic blood pressure responses than those receiving support from a male audience, $F(1,101) = 8.41$, $p < .005$. For the nonsupport conditions, there was no

TABLE 1. Mean (SD) Cardiovascular Change Scores for Male and Female Speakers Receiving Supportive or Nonsupportive Feedback From Male or Female Audience

	Female Audience				Male Audience			
	Support	*N*	Nonsupport	*N*	Support	*N*	Nonsupport	*N*
Systolic								
Female speaker	22.8 (16.8)	15	29.5 (15.8)	15	27.7 (21.0)	14	32.5 (12.8)	13
Male speaker	29.0 (21.4)	11	42.0 (11.0)	14	38.0 (18.2)	12	24.2 (17.3)	15
Diastolic								
Female speaker	15.7 (20.7)	15	25.3 (11.1)	15	24.9 (16.0)	14	24.7 (6.6)	13
Male speaker	16.0 (12.5)	11	27.6 (10.4)	14	29.8 (13.3)	12	19.9 (16.3)	15
Heart rate								
Female speaker	7.9 (4.1)	15	10.9 (7.2)	15	13.3 (10.9)	14	15.6 (7.2)	13
Male speaker	7.4 (5.4)	11	13.1 (10.9)	14	6.3 (5.3)	12	7.3 (7.0)	15

difference between male and female audiences, $F(1,101) = 1.24$, NS.

Consistent with the systolic changes, there was no two-way interaction of subject gender and support, $F(1,101) = 0.48$, NS. In the case of diastolic blood pressure changes (also shown in Table 1), there was no significant three-way interaction. However, the same trend for the male audience to produce a backwards effect on male speakers was observed, although again it was not significant, using the Bonferroni adjustment.

Heart Rate. Heart rate showed a somewhat different pattern of results from the blood pressure measures. The pattern of heart-rate changes is shown in Table 1. There was a significant main effect of subject gender, with women exhibiting larger heart-rate responses to the speech than men. Women's heart rate increased 11.8 BPM, whereas men's increased 8.7 BPM, $F(1,101) = 5.27$, $p < .05$. There was no effect of audience gender, $F(1,101) = 0.29$, NS. There was a main effect of support, $F(1,101) = 4.18$, $p < .05$, with the supported groups showing smaller increases than the nonsupported groups.

The two-way audience gender by support interaction was not significant, $F(1,101) = 0.85$, NS. The two-way interaction of subject gender and support was also not significant, $F(1,101) = 0.06$, NS. This reveals that for heart-rate responses, the male dyads did not show the backwards pattern observed for the blood pressure measures. Consistent with this, there was no three-way interaction, $F(1,101) = 0.48$, NS.

Differences Between Confederates

There was no difference within audience gender, either as a main effect or an interaction, on any dependent variable, depending on which confederate served as the audience. Furthermore, the overall pattern (female confederates providing effective social support to both genders and male confederates not providing effective support to either) was present for all of the confederates with two exceptions: one male confederate produced a social support pattern for female speakers, and one female confederate failed to produce a social support effect for female speakers. However, the female confederate provided nonsupport to only two female speakers, and the male confederate provided support to only two female speakers. Overall, the consistency of the pattern of gender differences supports the explanation that it is the gender of the confederate, and not just particular characteristics of the people who served as confederates, that is the important dimension.

Self-Reported Stress

Answers to each Likert-type question addressing the subjects' ratings of their level of stress during the speech were averaged into an overall stress index score. This showed good internal consistency, with Cronbach's $\alpha = .79$. As with many previous studies, however, the subjective stress ratings did not follow the same pattern as the blood pressure changes. There was no correlation between the stress index and the physiological change scores (systolic blood pressure, $r = .12$; diastolic blood pressure, $r = .12$; heart rate, $r = .04$). Furthermore, there was no hint of the same interaction of confederate gender with support that appeared with the physiological data, $F(1,99) = 0.27$, NS. (There was also no hint of such an interaction for any of the eight individual stress questions, all p values $> .1$) Nor was there any hint of an interaction of subject gender with support, $F(1,99) = 2.23$, NS. One effect that was significant was that women, on average, reported more stress than men, $F(1,99) = 15.23$, $p < .0005$.

DISCUSSION

The data indicate that, compared with nonsupportive feedback, social support from a woman is more effective at reducing blood pressure responses to stress in both men and women than support from a man. The finding that speakers show smaller blood pressure responses when a female audience behaved supportively than when she acted nonsupportively is consistent with earlier social support findings (13, 18, 22, 25). When the audience was male, overall, the speakers who saw supportive behaviors showed no less physiological activation than those who received nonsupportive feedback. In fact, there was some tendency for the men who received support from a male audience to show greater blood pressure changes than the men who received nonsupport. Additional research might explore whether the pattern obtained with a male audience and male speaker is reliable, and whether it is a general negative reaction to support, or if it is more particular to the kind of support offered in this paradigm.

The social support effects on cardiovascular responses that we observed in this study may underestimate the potential effects of having an ally. When the person offering social support is not a stranger, but someone with whom the speaker has a prior relationship, the cardiovascular effects of the support are magnified (22). In this study, it was not possible to use close friends, because doing so would prevent random assignment of speakers to the two audience genders (the closeness and gender of friends cannot be assumed to be independent). However, it is possible that the gender difference we observed in the effectiveness of support is smaller than the difference that may exist among close friends or spouses.

The patterns of the self-report data do not offer much insight into the processes that produced our effects. Speakers rated the supportive feedback of both male and female audiences as supportive, and rated the nonsupportive feedback of both male and female audiences as nonsupportive, and there was no accompanying interaction between support and audience gender. These self-report scores suggest that support from men is perceived, but that it is not translated into attenuated blood pressure responses. The self-reports of stress are no more helpful than the supportiveness ratings in providing insight. Subjective reports of stress failed to show any sensitivity to the social support manipulation or to confederate gender. This lack of correspondence between self-reports and physiological measures is typical of social support studies (12, 13, 17, 18, 36).

Our data do not indicate why it should be that support from women is more effective than support from men in reducing cardiovascular responses. In the epidemiological findings, and in the Kirschbaum et al. (34) study, there may well have been differences in the actual supportive behaviors from men and women. Hall (51), for example, has documented stable gender differences in nonverbal behavior. However, the actual physical behaviors in the present work were the same for our male and female confederates, and so the gender difference cannot be due to a difference in the nature of the support that the men and women provided. The difference in physiological responsivity in the present work depends instead on the speaker's construal of the meaning of the behaviors. The smiles and nods of a woman mean something different from the smiles and nods of a man. Although these supportive behaviors from a man during a speech did not buffer cardiovascular responses, there may be situations where men are capable of providing effective social support. The present findings do not provide any hints about what those situations might be.

Our data do not answer the question of whether the observed social support effect was due to supportive feedback from female audiences lowering blood pressure responses, or nonsupportive feedback increasing those responses. Lepore et al. (18) found evidence for both effects. Subjects who gave a speech with social support showed smaller blood pressure responses than those who gave the speech alone, and they, in turn, showed smaller responses than those who gave the speech to a nonsupportive audience. However, in a fundamentally social task, such as our public-speaking stressor, an "alone" condition may seem somewhat artificial to the subjects. There are other sorts of stressors that people could reasonably face with a supportive ally, with a nonsupportive other, or alone. Designs using such tasks can disentangle the nature of the social-support effect, by comparing support and nonsupport conditions to the alone condition. In our task, not only might it be somewhat peculiar to give a speech to nobody, but also a large part of the stress is delivering a speech in front of a stranger. Both designs with social and asocial stressors may provide reasonable models for the epidemiological findings on the benefits of social support. People with poor social networks may face more stressful situations alone, and also face more social stressors without a supportive ally.

Part of the audience-gender pattern in our study could be due to speakers expecting more friendly support from female audiences than from male audiences, and so being more alarmed when this support is absent. The simple effects analyses are ambiguous on this point. For systolic blood pressure responses, the pattern seems to be a combination of the benefits of support from a woman, compared with support from a man, and the costs of nonsupport from a woman compared with nonsupport from a man. For the diastolic blood pressure responses, the data favor the beneficial aspects of support from a woman.

The general findings concerning the effects of social support on cardiovascular responses provide a possible mechanism for the epidemiological evidence that people with better social networks are at reduced risk for cardiovascular disease. The gender findings of the present study suggest a possible mechanism that might help explain the epidemiological literature on the relationship between gender, social support, and health. Our findings are consistent with the notion that married men are healthier because, for the most part, they marry women. Women do not profit as much from marriage or suffer as much from separation in terms of health outcomes, because the support they gain or lose is the less effective support of a man. That is, if one accepts that reducing the magnitude of blood pressure responses to stress can have a protective effect on the cardiovascular system, then people with friends, especially female friends, should suffer less heart disease.

REFERENCES

1. Berkman LF, Syme SL. Social networks, host resistance, and mortality: a nine-year follow-up study of Alameda County residents. Am J Epidemiol 1979;109:186–204.
2. House JS, Robbins C, Metzner HL. The association of social relationships and activities with mortality: prospective evidence from the Tecumseh Community Health Study. Am J Epidemiol 1982;116:123–40.
3. Orth-Gomer K, Johnson J. Social network interaction and mortality: a six year follow-up study of a random sample of the Swedish population. J Chronic Dis 1987;40:949–57.
4. House JS, Landis KR, Umberson D. Social relationships and health. Science 1988;241:540–5.
5. Yasuda N, Zimmerman SI, Hawkes W, Fredman L, Hebel JR, Magaziner J. Relation of social network characteristics to 5-year mortality among young-old versus old-old white women in an urban community. Am J Epidemiol 1997;145:516–23.
6. Penninx BWJH, van Tilberg T, Kriegsman DMW, Deeg DJ, Boeke AJ, van Eijk JT. Effects of social support and personal coping resources on mortality in older age: the longitudinal aging study in Amsterdam. Am J Epidemiol 1997;146:510–9.
7. LaVeist TA, Sellers RM, Brown KAE, Nickerson KJ. Extreme social isolation, use of community-based senior support services, and mortality among African American elderly women. Am J Community Psychol 1997;25:721–32.
8. Reed D, McGee D, Yano K. Social networks and coronary heart disease among Japanese men in Hawaii. Am J Epidemiol 1983; 117:384–96.
9. Blumenthal JA, Burg MM, Barefoot J, Williams RB, Haney T, Zimet G. Social support, Type A behavior and coronary artery disease. Psychosom Med 1987;49:331–9.
10. Seeman TE, Syme SL. Social networks and coronary artery disease: a longitudinal analysis. Soc Psychol Q 1987;48:237–48.
11. Williams RB, Barefoot JC, Califf RM, Haney TL, Saunders WB, Pryor DB, Hlatky MA, Siegler IC, Mark DB. Prognostic importance of social and economic resources among medically treated patients with angiographically documented coronary artery disease. JAMA 1992;267:520–4.
12. Kamarck TW, Manuck SB, Jennings JR. Social support reduces cardiovascular reactivity to psychological challenge: a laboratory model. Psychosom Med 1990;52:42–58.
13. Gerin W, Pieper C, Levy R, Pickering TG. Social support in social interaction: a moderator of cardiovascular reactivity. Psychosom Med 1992;54:324–36.
14. Krantz DS, Manuck SB. Acute psychophysiologic reactivity and risk of cardiovascular disease: a review and methodologic critique. Psychol Bull 1984;96:435–64.
15. Krantz DS, Manuck SB. Psychophysiologic reactivity in coronary heart disease and essential hypertension. In Matthews KA, Weiss SM, Detre T, Dembroski TM, Falkner B, Manuck SB, Williams RB, editors. Handbook of stress, reactivity, and cardiovascular disease. New York: Wiley; 1986.
16. Lovallo WR, Wilson MF. The role of cardiovascular reactivity in hypertension risk. In Turner JR, Sherwood A, Light KC, editors. Individual differences in cardiovascular response to stress. New York: Plenum; 1992. p. 165–86.
17. Snydersmith MA, Cacioppo JT. Parsing complex social factors to determine component effects: I. Autonomic activity and reactivity as a function of human association. J Soc Clin Psychol 1992;11:263–78.
18. Lepore SJ, Allen KAM, Evans GW. Social support lowers cardiovascular reactivity to an acute stressor. Psychosom Med 1993;55:518–24.
19. Gerin W, Milner D, Chawla S, Pickering TG. Social support as a moderator of cardiovascular reactivity: a test of the direct effects and buffering hypothesis. Psychosom Med 1995;57:16–22.
20. Kamarck TW, Annunziato B, Amateau LM. Affiliation moderates the effects of social threat on stress-related cardiovascular responses: boundary conditions for a laboratory model of social support. Psychosom Med 1995;57:183–94.
21. Uchino B, Cacioppo JT, Kiekolt-Glaser JK. The relationship between social support and physiological processes: a review with emphasis on underlying mechanisms and implications for health. Psychol Bull 1996;119:488–531.
22. Christenfeld N, Gerin W, Linden W, Sanders M, Mathur J, Deich JD, Pickering TG. Social support effects on cardiovascular reactivity: is a stranger as effective as a friend? Psychosom Med 1997;59:388–98.
23. Sheffield D, Carroll D. Social support and cardiovascular reactions to active laboratory stressors. Psychol Health 1994;9: 305–16.
24. Sheffield D, Carroll D. Task-induced cardiovascular activity and the presence of a supportive or undermining other. Psychol Health 1996;11:583–91.
25. Lepore SJ. Cynicism, social support, and cardiovascular reactivity. Health Psychol 1995;14:210–6.
26. Maccoby EE, Jacklin C. The psychology of sex differences. Stanford, CA: Stanford University Press; 1974.
27. Deaux K. From individual differences to social categories: analysis of a decade's research on gender. Am Psychol 1984;39: 105–16.
28. Deaux K. Sex and gender. Annu Rev Psychol 1985;36:49–81.
29. Hyde JS, Linn MC. The psychology of gender: advances through meta-analysis. Baltimore: Johns Hopkins University Press; 1986.
30. Wiklund I, Oden A, Sanne H, Ulvenstam G, Wilhelmsson C, Wilhelmsen L. Prognostic importance of somatic and psychosocial variables after a first myocardial infarction. Am J Epidemiol 1988;128:786–95.
31. Stroebe MS, Stroebe W. Who suffers more? Sex differences in health risks of the widowed. Psychol Bull 1983;93:279–301.
32. Helsing KJ, Szklo M, Comstock GW. Factors associated with mortality after widowhood. Am J Public Health 1981;71:802–9.

33. Martikainen P, Valkonen T. Mortality after the death of a spouse: rates and causes of death in a large Finnish cohort. Am J Public Health 1996;86:1087–93.
34. Schoenbach VJ, Kaplan BH, Fredman L, Kleinbaum DG. Social ties and mortality in Evans County, Georgia. Am J Epidemiol 1986;123:577–91.
35. Shye D, Mullooly JP, Freeborn DK, Pope CR. Gender differences in the relationship between social network support and mortality: a longitudinal study of an elderly cohort. Soc Sci Med 1995;412:935–47.
36. Kirschbaum C, Klauer T, Filipp S, Hellhammer DH. Sex-specific effects of social support on cortisol and subjective responses to acute psychological stress. Psychosom Med 1995;57:23–31.
37. Allen KM, Blascovich J, Tomaka J, Kelsey RM. Presence of human friends and pet dogs as moderators of autonomic responses to stress in women. J Pers Soc Psychol 1991;61:582–9.
38. Edens JL, Larkin KT, Abel JL. The effect of social support and physical touch on cardiovascular reactions to mental stress. J Psychosom Res 1992;36:371–82.
39. Brown PC, Smith TW. Social influence, marriage, and the heart: cardiovascular consequences of interpersonal control in husbands and wives. Health Psychol 1992;11:88–96.
40. Linden W, Chambers L, Maurice J, Lenz JW. Sex differences in social support, self-deception, hostility and ambulatory cardiovascular activity. Health Psychol 1993;12:376–80.
41. Smith TW, Limon JP, Gallo LC, Ngu LQ. Interpersonal control and cardiovascular reactivity: goals, behavioral expression, and the moderating effects of sex. J Pers Soc Psychol 1996;70:1012–24.
42. Imholz BPM, Settels JJ, Meiracker AH, Wesseling KH, Wieling W. Noninvasive continuous finger blood pressure measurement during orthostatic stress compared with intra-arterial. Cardiovasc Res 1990;24:214–21.
43. Gorback MS, Quill TJ, Lavine ML. The relative accuracies of two automated noninvasive arterial pressure measurement devices. J Clin Monit 1991;7:13–22.
44. Wieling W, Harkel ADJ ten, Lieshout J J van. Spectrum of orthostatic disorders: classification based on an analysis of the short-term circulatory response on standing. Clin Sci 1991;81:241–8.
45. Parati G, Casadei R, Groppelli A, Di Rienzo M, Mancia G. Comparison of finger and intra-arterial blood pressure monitoring at rest and during laboratory testing. Hypertension 1989;13: 647–55.
46. Gerin W, Pieper C, Pickering TG. Measurement reliability of cardiovascular reactivity change scores: a comparison of intermittent and continuous methods of assessment. J Psychosom Res 1993;37:493–501.
47. Glynn LM, Christenfeld N, Gerin W. Implications of alternative methods of computing blood pressure means. Blood Pressure Monit 1997;2:175–8.
48. Smith TW, Nealey JB, Kircher JC, Limon JP. Social determinants of cardiovascular reactivity: effects of incentive to exert influence and evaluative threat. Psychophysiology 1997;34:65–73.
49. Gerin W, Christenfeld N, Pieper C, DeRafael DA, Su PO, Stroessner SJ, Deich J, Pickering TG. The generalizability of cardiovascular responses across settings. J Psychosom Res 1998;44: 209–18.
50. Llabre MM, Spitzer SB, Saab PG, Ironson GH, Schneiderman N. The reliability and specificity of delta versus residualized change as measures of cardiovascular reactivity to behavioral challenges. Psychophysiology 1991;28:701–11.
51. Hall JA. Nonverbal sex differences: accuracy of communication and expressive style. Baltimore: Johns Hopkins University Press; 1994.