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Exploring Emotion-Regulation and Autonomic Physiology in Metastatic Breast Cancer Patients: Repression, Suppression, and Restraint of Hostility

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Abstract

We examined relationships between three emotion-regulation constructs and autonomic physiology in metastatic breast cancer patients ($N = 31$). Autonomic measures are not often studied in breast cancer patients and may provide evidence of an increase in allostatic load. Patients included participated as part of a larger clinical trial of supportive-expressive group therapy. Systolic and diastolic blood pressure and heart rate were assessed at a semi-annual follow-up. We averaged 3 resting assessments and used measures of Repression, Suppression, Restraint of Hostility, and Body Mass Index as predictors of autonomic response. We found that higher repression was significantly associated with higher diastolic blood pressure, while higher restraint of hostility was significantly associated with higher systolic blood pressure. A repressive emotion regulation style may be a risk factor for higher sympathetic activation possibly increasing allostatic load, while restraint of hostility may be a protective factor for women with metastatic breast cancer.

Keywords

emotion regulation; emotional expression; autonomic physiology; breast cancer; repression; suppression; hostility

Studies have demonstrated that diminished emotional expressiveness is associated with health risks. For example, research has linked repression (low anxiety and high defensiveness) and suppression (deliberate withholding of the expression of negative emotions) with faster cancer progression and shorter survival (Bahnsen, 1981; Fox, Harper, Hyner, & Lyle, 1994; Giese-Davis & Spiegel, 2003; Greer, 1985; Greer & Morris, 1975; Gross, 1989; Kneier & Temoshok, 1984). In addition, an epidemiological study found that subjects likely to suppress anger were at greater mortality risk from all causes across age, sex, and education groups (Julius, E., Cottingham, & Johnson, 1986). In the Julius study, suppressed anger significantly interacted with elevated blood pressure (BP) to predict the highest mortality risk (Julius et al., 1986). Three emotion inhibition processes may be associated with psychological and physiological indices of health in metastatic breast cancer patients: suppression, repression, and restraint of

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hostility (Giese-Davis et al., 2002; Giese-Davis, Sephton, Abercrombie, Duran, & Spiegel, 2004; Giese-Davis & Spiegel, 2001). Because cardiovascular measures are not often studied in cancer patients and may provide additional evidence of an increase in allostatic load (Giese-Davis et al., 2006; McEwen, 2004), we collected and calculated autonomic measures: BP, heart rate (HR), mean arterial pressure (MAP), pulse pressure (PP), and rate pressure product (RPP). Because DBP and SBP have each been shown to be predictors of cardiac events in older adults (Domanski et al., 2002; Psaty et al., 2001), we specifically examined their relationship to targeted emotion-regulation measures.

Repression is a defense mechanism in which a person is unable to remember or be cognitively aware of disturbing wishes, feelings, thoughts, or experiences ((Task Force on DSM-IV, 1994), pp. 756-757). Weinberger ((Weinberger, 1990a), p.341) states that, “repressive individuals are hypothesized to be persons who often believe that they are not upset despite objective evidence to the contrary.” Suppression differs from repression in that a person is aware of negative affect, but does not express it (DSM-IV, 1994). A suppressed individual holds all components of a conflict in mind, but postpones action, affective response, or ideational worry associated with that conflict (Vaillant & Vaillant, 1990). Restraint of hostility is the ability to “inhibit immediate, self-focused impulses in the interest of other, usually more long-term goals” ((Feldman & Weinberger, 1994), p.196). This includes restraint of impulsivity, verbal and physical aggression, and direct manipulation of others for personal gain. Autonomic hyperarousal has been associated with repression, suppression or inability to restrain hostile responses.

Repression

A number of studies demonstrate that repressors exhibit a discrepancy between low self-reported anxiety and high cardiovascular activity HR, SBP, and DBP (Asendorpf & Scherer, 1983; King, Taylor, Albright, & Haskell, 1990; Linden, Paulhus, & Dobson, 1986; Weinberger, Schwartz, & Davidson, 1979). Eysenck’s (Eysenck & Derakshan, 1997) theory of repressors indicates that they avoid attending to, and tend to interpret four sources of information (environmental stimuli, personal physiological reactivity, personal behavior, information in long term memory) in a non-threatening way. Therefore, they do not describe high activation in self-reports (due to an interpretive bias), but show elevated bodily physiological symptoms.

Suppression

Suppression is also linked to elevated cardiovascular physiology (Davidson, 1993; Julius et al., 1986; Vogelee & Steptoe, 1992). In Davidson (1993) suppressors exhibited higher systolic BP (SBP) than repressors. Gross and Levenson (Gross & Levenson, 1993, 1997) also found that when directed to suppress expression while watching arousing film clips, some cardiovascular parameters such as HR suggested deactivation, while others indicated increased sympathetic nervous system activity. In a second study (Gross & Levenson, 1997), suppression of neutral affect had no physiological consequences but suppression of positive or negative affect elevated sympathetic cardiovascular activation. A considerable number of studies (e.g., (Gallacher, Yarnell, Sweetnam, Elwood, & Stansfeld, 1999; Vögele, Jarvis, & Cheeseman, 1997; Vogelee & Steptoe, 1992)) also demonstrated that inhibition of anger expression elevated cardiovascular reactions to stress while over anger expression did not. A meta-analysis of 295 relevant effect sizes from studies of suppression obtained from 25,469 participants (Jorgensen, Johnson, Kolodziej, & Schreer, 1996) found that lower affect expression (as well as defensiveness) was associated with higher BP and greater hypertension.

Hostility

Research documents distinctions (cognitive, emotional, and overt behavior) (Buss, 1961) in the measurement of hostility and its relationship with cardiovascular disease (Graham et al.,

2006; Ruiz, Uchino, & Smith, 2006). Beginning with Meyer Friedman who noticed that cardiovascular patients sat on the edge of their chairs in an outpatient waiting room, the Type A personality posited that greater cardiovascular disease is associated with incessant busyness, high levels of frustration, high expression of hostility, and impatience with others (Friedman & Rosenman, 1960, 1964; Friedman, Rosenman, & Brown, 1963). In clarifying these constructs, the cognitive component of hostility refers to “negative beliefs about and attitude toward others including cynicism, mistrust, and denigration” (Miller, Smith, Turner, Guijarro, & Hallet, 1996) p. 323. Overt behavior refers to actual attacks either verbal or physical, including “insult, sarcasm, rudeness, and simple opposition” ((Miller et al., 1996) p. 323). Thus the ability to inhibit or restrain hostility at any of these levels may increase healthier cardiovascular response.

Current Hypotheses

In the current study, we assessed BP and HR in a convenience sample of metastatic breast cancer patients randomized in a clinical trial comparing supportive-expressive group therapy to an educational control condition. We tested hypotheses that those people high on repression and suppression would have higher, and those high on restraint of hostility would have lower cardiac risk as measured by SBP, DBP, and HR (when body mass index (BMI) was included in the equation). For the possible interest of the reader, we also included descriptive information about derivative measures MAP, PP, and RPP. Findings consistent with our hypotheses may indicate an additional physiological burden while coping with breast cancer.

Methods

Participants included 31 of 125 women with metastatic or recurrent breast cancer (see (Giese-Davis et al., 2002) for a thorough description). All participants gave written informed consent under approval by the Stanford I.R.B. before HIPAA requirements. All participants lived in the Greater San Francisco Bay Area, were English speaking and able to complete questionnaires. Women were excluded if they had no metastasis beyond positive supraclavicular lymph nodes, had active cancers within the past 10 years (other than breast cancer, basal cell or squamous cell carcinomas of the skin, in situ cancer of the cervix, or melanoma with a Breslow depth below 0.76mm), a Karnofsky score of less than 70 (Karnofsky & Burchenal, 1949), or a medical condition that could affect short-term survival.

Sixty-four of the 125 women were randomized to receive at least a year of supportive-expressive group therapy (SET). Women in the control and treatment groups all received educational materials. Baseline assessments included self-report of social support, distress, coping and physical activity. Samples of blood and saliva were also collected to assess immune and endocrine function. Post-baseline assessments were conducted every 4 months in the first year and every 6 months thereafter.

The 31 women included in this small study were part of a convenience sample. For a one-year period, each woman was asked if we could collect HR and BP at her routine semi-annual follow-up just prior to a blood draw. Women included in this sample, had this BP/HR assessment, and data for weight, height, suppression, repression, and restraint. One woman was lost from an initial 32 who met criteria due to no weight measurement. The women were in both treatment (N=22) and control (N=9) groups. Because of the nature of this convenience sampling period combined with the rolling enrollment in the study, this one-year collection meant that women had been in the study for varying lengths of time (from 1 to 5 yrs from baseline): 31.3% were assessed at one-year, 18.8% at two-year, 18.8% at three-year, 15.6% at four-year, and 15.6% at five-year follow-up. Analyses comparing the current sample ($n = 31$) with the rest of the sample not used ($n = 94$) indicated that this sample did not differ on any demographic or medical

variables with the exception that a greater percentage of women selected for this study had not been given chemotherapy as a treatment for their disease, $\chi^2 (1, N = 125) = 4.09, p < .05$.

Measures

Blood Pressure and Heart Rate—SBP/DBP and HR were assessed with a 7000-7001 Continuous Noninvasive Blood Pressure-02SAT Device. Subjects were assessed prior to a blood draw for immune measures. We averaged 3 resting (5 min. before), eyes-closed, BP and HR assessments. We allowed 20 sec. between the 3 assessments. Arm for the cuff we used was opposite the side of the body used for axillary lymph node dissection. Both SBP and DBP strongly predict cardiac events (Domanski et al., 2002; Miura et al., 2001).

Mean Arterial Pressure—MAP was calculated with the equation $(SBP + 2 \times DBP)/3$. MAP measures average pressure during the cardiac cycle. DBP is weighted twice as much as SBP because 2/3 of the cardiac cycle is spent in diastole. MAP with absolute accuracy can be computed by summing central venous pressure (CVP) and the product of systemic vascular resistance (SVR) and cardiac output (CO). However, at normal resting HR, MAP can be approximated by the above equation. An MAP of about 60 is necessary to perfuse coronary arteries, brain, kidneys, while the usual range is 70 to 110. It is a predictor of cardiac events (Miura et al., 2001).

Pulse Pressure—PP was calculated as SBP-DBP and measures the maximal change in aortic pressure during systole. PP is influenced by the compliance of the aorta as well as stroke volume. High PP increases risk of cardiovascular events in an older population (Blacher et al., 2000; Fernández-Escribano Hernández et al., 2007; Foucan et al., 2005).

Rate Pressure Product—RPP was calculated as $HR \times SBP$. RPP is strongly correlated with cardiac oxygen consumption, and indicates the load of the heart (Hinderliter, Miller, Bragdon, Ballenger, & Sheps, 1991). Increases in RPP are related to ischemic events in both normotensive and hypertensive men with CAD (Deedwania & Nelson, 1990).

Women completed a questionnaire at the time of BP and HR collection assessing family history of serious medical problems, consumption of a caffeinated beverage that morning, and average levels of consumption of coffee/tea, soda, tobacco, alcohol, and sleep, diet, exercise, and stress in past 24 hours. Weight and height were also collected.

We time-matched the emotion-regulation data collected closest to BP and HR assessments, because this sample of subjects was assessed at a number of different individual follow-up points.

Suppression—We used The Courtauld Emotional Control Total Score (CECS: (Watson & Greer, 1983)), a 21-item questionnaire measuring whether participants “smother” or “bottle-up” feelings of anger, depression, and anxiety. Coefficient alpha at baseline was .95. The time lag between completion of the CECS and having their BP assessment was relatively short ($M = 27.19$ days, $SD = 25.17$, $R = 0.0 - 88.00$).

Repression and Restraint—We used The Weinberger Adjustment Inventory WAI-Short Form: (Weinberger, 1990a, 1990b; Weinberger, 1997; Weinberger & Schwartz, 1990), a validated measure of repressive-defensiveness and restraint (Turvey & Salovey, 1993-94). We used the Restraint Scale comprised of Suppression of Aggression, Impulse Control, Consideration of Others, and Responsibility subscales, and the Repressive-Defensiveness Scale as continuous measures. The time lag between WAI completion and BP assessment was

relatively long ($M = 617.32$ days, $SD = 458.83$, $R = 1-1182$). This lag was longer than for the CECS because it was only given twice, at baseline and at one year.

Results

All independent variables were relatively non-collinear (no Pearson Correlation Coefficient above .19).

We tested SBP, DBP, and HR separately as dependent variables. In each regression model, we entered repression, restraint of hostility, suppression, and BMI as independent variables (Table 3).

Higher DBP was associated with repression with 23% of the variance predicted.

Higher SBP was associated with higher BMI and lower restraint of hostility with 25% of the variance predicted.

We could demonstrate no significant associations with HR.

Discussion

We found that higher repression was associated with higher DBP in breast cancer patients, while higher restraint of hostility was associated with lower SBP. These results confirm our hypotheses that relationships between these coping styles and autonomic activation (Asendorpf & Scherer, 1983; King et al., 1990) extend to the breast cancer population, and may increase allostatic load, potentially contributing to disease progression.

These results suggest physiological distinctions among emotion regulatory abilities in these cancer patients. Repression is associated with cognitive inability to assess that negative affect or stress is experienced (Weinberger & Davidson, 1994), and thus corrective action that might reduce the impact of stress on the body is short-circuited (Brown et al., 1996). Consequently, higher repression was associated with higher DBP and has been associated with both greater cardiovascular resting activity and reactivity to stress in other populations.

However, restraint of hostility is a positive emotion-regulatory ability associated with better social functioning (Feldman & Weinberger, 1994; Weinberger & Schwartz, 1990), and here linked with lower SBP. Much past research indicates that lack of restraint (e.g. the expression of hostility) is associated with greater cardiovascular disease risk, including greater inflammation (Graham et al., 2006), HR response to stress (Ruiz et al., 2006), BP (Davidson, 1993; Linden, Chambers, Maurice, & Lenz, 1993), hypertension (Yan et al., 2003), atrial fibrillation (Eaker, Sullivan, Kelly-Hayes, D'Agostino, & Benjamin, 2004), and CHD (Miller et al., 1996). Our results indicate that the ability to restrain hostility is associated with better sympathetic functioning in metastatic breast cancer patients.

We could not demonstrate that suppression of the expression of anxiety, anger, and depression using the CECS was associated with cardiovascular indices. Some studies finding strong effects of suppression on cardiovascular function asked participants to actively suppress expression in-the-moment (Gross & Levenson, 1993, 1997). Our study instead assessed trait-like suppression and examined resting cardiovascular measures. It is unclear whether in-the-moment suppression would be associated with cardiovascular function in this sample.

Our study is limited by the self-selection of participants who were part of a convenience sample of a larger study. However these women did not differ from the larger sample in most clinical and demographic variables, and we were able to demonstrate significant effects even with our

small sample. We cannot generalize beyond our sample which consisted predominantly of non-Hispanic Caucasians. Finally, we did not recruit matched controls without breast cancer, nor is there a comparable literature examining this hypothesis in the literature. Consequently, it is difficult to interpret absolute values of the physiological data. However, these limitations are counterbalanced by providing preliminary information on the relationship between cardiovascular function and emotion regulation in metastatic breast cancer.

In summary, we found evidence that repression was associated with higher DBP and the ability to restrain hostility was associated with lower SBP at rest in metastatic breast cancer patients. Future studies could address whether physiological activation at rest is a useful predictor for disease progression. If so, physiologically oriented treatments aimed at physiological deactivation, such as breathing training and muscle relaxation therapy, may be an effective addendum to emotion-focused psychotherapy (Classen et al., 2001; Giese-Davis et al., 2002; Goodwin et al., 1996; Spiegel, Bloom, Kraemer, & Gottheil, 1989; Spiegel, Bloom, & Yalom, 1981) and cognitively oriented interventions (Antoni & Carver, 2003) to improve mental and physiological well-being in breast cancer patients.

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Table 1Demographic, Medical, and Study Variables Measured at Baseline in Metastatic Breast Cancer Patients ($N=31$).

<i>(n = 31)</i>	
Characteristic	<i>M (SD)</i>
Diastolic Blood Pressure	74.46 (0.05)
Systolic Blood Pressure	117.04 (14.23)
Heart Rate	82.53 (13.40)
Mean Arterial Pressure	88.66 (9.06)
Pulse Pressure	42.58 (10.49)
Rate Pressure Product	9619.80 (1725.97)
Repression	35.90 (7.21)
Restraint	55.10 (3.72)
Suppression	15.71 (3.57)
Body Mass Index	25.08 (5.44)
Age at randomization	53.99 (12.34)
Disease-Free Interval (Months)	49.34 (35.50)
Time From Metastasis To Study Entry (Months)	31.14 (34.70)
Years of Education	16.61 (2.65)
Characteristic	%
Ethnicity	
Minority	3.2
Caucasian	96.8
Marital Status	
Single	35.6
Married	64.5
Household Income	
< \$20,000--79,999	64.5
\$80,000 --\$100,000 or >	32.3
Estrogen Receptor Negative	16.1
Treatment	
Chemotherapy	32.3
Hormone Therapy	80.6
Site of Metastasis	
Chest Wall	35.5
Bone	32.3
Viscera	32.3

Note. Repression and Restraint = Weinberger Adjustment Inventory. Suppression = Courtauld Emotional Control Scale.

Table 2Correlations Among Emotion-Regulation and Cardiovascular Measures For Women With Metastatic Breast Cancer ($N = 31$).

	DBP	SBP	HR	MAP	PP	RPP	Repression	Restraint
DBP	-----							
SBP	0.69***	-----						
HR	-0.05	-0.21	-----					
MAP	0.93***	0.91***	-0.14	-----				
PP	0.21	0.85***	-0.26	0.57	-----			
RPP	0.41*	0.47**	0.76***	0.47**	0.34	-----		
Repression	0.40*	0.19	-0.11	0.33	-0.03	0.02	-----	
Restraint	-0.12	-0.39*	-0.21	-0.27	-0.45*	-0.44*	0.02	-----
Suppression	-0.21	0.08	-0.11	-0.08	0.27	-0.01	0.05	0.16
BMI	0.22	0.38*	0.07	0.33	0.36*	0.32	-0.18	-0.12

Note. Repression and Restraint = Weinberger Adjustment Inventory. Suppression = Courtauld Emotional Control Scale. BMI = Body Mass Index.

Table 3

Summary of Simultaneous Regressions for Variables Predicting Cardiovascular Measures For Women with Metastatic Breast Cancer ($N = 31$).

Diastolic Blood Pressure	<i>B</i>	<i>SE B</i>	β
Repression	0.51	0.17	0.48**
Restraint	-0.09	0.34	-0.05
Suppression	-0.57	0.35	-0.27
BMI	0.48	0.23	0.34
Systolic Blood Pressure			
Repression	0.53	0.32	0.27
Restraint	-1.41	0.62	-0.37*
Suppression	0.35	0.65	0.09
BMI	1.00	0.43	0.38*
Heart Rate			
Repression	-0.17	0.36	-0.09
Restraint	-0.68	0.70	-0.19
Suppression	-0.28	0.73	-0.07
BMI	0.09	0.49	0.04

Note: DBP $R^2 = 0.23$, $F(4,26) = 3.22$, $p = .03$; SBP $R^2 = 0.25$, $F(4,26) = 3.50$, $p = .02$; HR $R^2 = -0.09$, $F(4,26) = 0.40$, $p = 0.80$. Repression and Restraint = Weinberger Adjustment Inventory. Suppression = Courtauld Emotional Control Scale. BMI = Body Mass Index.