TRAIT ANXIETY AS A PREDICTOR OF PSYCHOLOGICAL AND PHYSIOLOGICAL DISTRESS OVER TIME IN PATIENTS RECENTLY DIAGNOSED WITH BREAST CANCER

by

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Breast cancer has an unpredictable course, can be fatal, and many breast cancer treatments and prevention strategies are unpleasant and have aversive side effects. Anxiety is a normal reaction to a breast cancer diagnosis and may promote adaptive responses to new demands. However, anxiety can also have negative psychological and physiological consequences. Past research has suggested that trait anxiety may be an important determinant of psychological adjustment as well as physiological disease outcomes. The specific effects of trait anxiety on the course of psychological functioning during the initial period of adjustment to disease and on disease course following diagnosis are not well understood. The primary aim of this study was to evaluate the course and influence of trait and acute anxiety in patients diagnosed with breast cancer. This study included 58 women with a new diagnosis of breast cancer. Anxiety measures, psychosocial distress measures, and salivary cortisol measurements were collected at diagnosis, and 3 and 6 months post-diagnosis. Overall, anxiety at diagnosis was related to poorer psychosocial outcomes during the first six months following a diagnosis of breast cancer. Specifically, trait anxiety was more predictive of long-term distress than was state anxiety.

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I. INTRODUCTION

Among American women, there is approximately a 13% lifetime risk of developing breast cancer and a 3.3% risk of dying from the disease. In 2004, it is expected that over 217,000 new cases of invasive breast cancer and 55,700 new cases of in situ breast cancer will be diagnosed (American Cancer Society, 2004). Breast cancer has an unpredictable course, can be fatal, and many treatments and prevention strategies have aversive side effects. Anxiety is a normal reaction to a breast cancer diagnosis and may promote adaptive responses to new demands. However, anxiety can also have negative psychological and physiological consequences.

The prevalence and symptoms of anxiety have been broadly investigated in breast cancer prevention, early detection and diagnosis, and treatment (McKenna, Zevon, Corn, & Rounds, 1999; Kreitler, Kreitler, Chaitchik, Shaked, & Shaked, 1997). The importance of recognizing stress and anxiety in this population has been documented (Slaughter et al., 2000; Andrykowski, Cordova, McGrath, Sloan, & Kenady, 2000). Still, the course and negative sequelae of specific anxiety characteristics in this population are poorly understood. Recent research has suggested that psychophysiological aspects of anxiety can interact with physiological aspects of breast cancer in potentially important ways (Cameron, Leventhal, Love, & Patrick-Miller, 2002). Specifically, trait anxiety has been related to physiological disease characteristics.

Although stress and anxiety should be expected to wax and wane as one's disease or treatment outlook changes, trait anxiety may be an important determinant of psychological adjustment as well as physiological disease outcomes. This study investigated the impact of trait

anxiety in patients with a new diagnosis of breast cancer by following patients closely during the first six months after diagnosis. The primary aim of this study was the evaluation of the course and influence of trait and acute anxiety in patients diagnosed with breast cancer. Secondly, this study aimed to examine of the course of psychological distress and quality of life in this population and to examine the differential influence of trait and acute anxiety on these factors. Lastly, this study aimed to examine the relationship between anxiety and physiological markers of stress in patients with breast cancer.

A. Background

The presence of stress has been well identified in patients with cancer (Mettler & Mettler, 1947; Skarstein, Aass, Fossa, Skovlund, & Dahl, 2000). Generally, stress can be expected with any life change and at times has been shown to be adaptive and beneficial, motivating people to make difficult lifestyle changes (Kreitler et al., 1997; McCaul, Schroeder, & Reed, 1996). Stress in patients with cancer may be acute and dissipate as the situation becomes more normative (Epping-Jordan et al., 1999; Irvine, Brown, Crooks, Roberts, and Browne, 1991), however, when stress does not dissipate, there is an increased risk for a chronic and interfering psychological and physiological responses (e.g., Brabander & Gerits, 1999).

Physiological and psychological stress and the interplay between the body and mind have long piqued the interest of scientific minds. Walter Cannon, a physiologist, was the first to describe the body's reaction to stress in the early part of the twentieth century (Greenberg, 1990). Cannon described the "fight-or-flight" response as the way the body prepares itself when confronted with a threat. It is the process that determines whether an organism will stay and "fight" the threat or "flight" and work to avoid the threat. During the same time period, Hans Selye, the "father of stress", worked toward an understanding of the body and mind interaction.

Selye believed that stress was a shared component in all illnesses. He defined stress as the nonspecific response of the body to environmental demands. He examined stress and its effects on the physiological systems. He conceptualized chronic stress as "exhausting" to the body and causing compromised immune system functioning. Later, Lazarus and Folkman (1984) introduced and proposed specific psychological appraisal components involved in the physiological stress response. More recently, stress has been conceptualized as an emotional experience usually accompanied by physiological arousal that results from appraisal of information about a stressor and an individual's perceived ability to deal with it. The emotional and physiological reaction experienced either motivates one to change or forces one to accommodate to or reduce the effects of the stressor by creating safety from it (Craig, Brown, & Baum, 1995).

Research has suggested several ways in which stress may negatively affect physiological activity in individuals with disease (Lovallo, 1997; Cohen, Kessler, & Gordon, 1995). Pathways that have been suggested by which stress affects disease progression include the influence of stress on hormone levels and immune responses (Luecken & Compass, 2002; Cohen et al., 1995). Specifically, stress in women with breast cancer has been shown to be associated with natural killer cell activity, T and B cell activity, and lymphocyte counts (Andersen, Kiecolt-Glaser, & Glaser, 1994; Tjemsland, Soreide, Matre, & Malt, 1997). Data from laboratory and psychosocial interventions studies aimed at reducing stress have suggested that stress levels may even be associated with the progression of disease in patients with cancer (Forlenza, Latimer, & Baum, 2000; Spiegel et al. 1989).

Situations that are perceived as threatening (e.g., disease diagnosis) can cause stress and may trigger anxiety in the individual. Barlow (2000) characterizes anxiety as being a state of

helplessness, because of perceived inability to predict, control, or obtain desired results or outcomes in upcoming personally salient situations or contexts. The prolonged or chronic manifestation of anxiety is dependent on the nature of the stress, the coping ability of the individual, and the social context of the stressor. Anxiety can become problematic when it is so intense that it diminishes efforts at coping and attention is solely focused on reducing distress and fear. This reaction can potentially affect appraisal of future stressors and change the experience of stress in routine events (Craig et al., 1995).

Non-chronic anxiety has been defined as state (or acute) anxiety, which can be descried as a temporal cross section in the emotional stream of one's life, consisting of subjective feelings of tension, apprehension, nervousness, worry, and activation of the autonomic nervous system. Conversely, trait anxiety refers to individual differences in the stable and enduring tendency to perceive a wide range of situations as dangerous or threatening, and to respond with more frequent and intense elevations in state anxiety (Spielberger, Gorsuch, & Lushene, 1970). Stress has been measured in the context of event related anxiety, anxiety disorders, symptoms, and characteristics, yet there still remains little consensus as to when and how stress for the patient with cancer moves from a normal and expected stress reaction to one with negative consequences.

1. Anxiety and Breast Cancer. Elevated levels of anxiety can potentially cause a wide variety of psychological and physiological problems for patients with breast cancer. Anxiety has been associated with an increase in pain, interference with sleep, and increased negative response to chemotherapy treatment. Anxiety in has also been associated with an increase in somatic symptoms causing subsequent early termination of potentially curative treatments (Love,

Leventhal, Easterling, & Nerenz, 1989). Additionally, anxiety has been associated with decreased quality of life in patients with cancer as well as their family members.

Women with breast cancer that have elevated acute anxiety may experience more intrusive thoughts and less optimism than less anxious women (Epping-Jordan et al., 1999), both of which have been associated with negative psychological and physiological effects (Scheier, Carver, & Bridges, 1994; Baum, Cohen, & Hall, 1993). Intrusive thoughts have also been associated with a higher rate of health complaints, depression, sleep problems, and an elevated risk for distress as long as two years post breast cancer diagnosis (Bleiker, Pouwer, van der Ploeg, Leer, & Ader, 2000).

Intrusive thoughts are a hallmark characteristic of anxiety and Posttraumatic Stress Disorder (PTSD), which has been studied as a response to a diagnosis of breast cancer. Several clinical studies have reported that some patients with cancer and their family members exhibit PTSD-like symptoms following cancer diagnosis and treatment. Those expressing a more emotionally reactive and anxious personality type have exhibited a heightened risk of developing significant PTSD symptoms in response to cancer diagnosis and treatment (Smith, Redd, Peyser, & Vogl, 1999). These risk factors suggest that pre-existing chronic anxiety may increase distress and the likelihood of PTSD symptoms in patients with cancer.

Research on anxiety in patients with cancer often has assumed that it is high levels of state anxiety that are problematic in adjustment to disease. This is evident in the fact that most studies of anxiety symptoms in cancer populations are based on single assessments, are usually associated with an event or procedure and often focus on the diagnosis of an anxiety disorder (e.g., Slaughter et al., 2000; Smith, et al., 1999). These approaches imply transient increases in anxiety. However, research provides some evidence for a differential experience for patients with

cancer that have high levels of trait anxiety. High levels of trait anxiety may uniquely influence psychological and physiological disease mechanisms (Cameron, Leventhal, & Love, 1998; Cameron et al., 2002). Although chronically elevated levels of anxiety may not reflect clinical episodes, distress remains during the course of everyday activity and contributes to hyperactive endocrine responses (Sullivan, Kent, & Copeland, 2000).

2. Trait Anxiety. In women with breast cancer, trait anxiety was shown to be inversely related to feelings of well-being (Kaczorowski, 1989), and cause increased and persistent psychological and physiological distress related to chemotherapy treatment (Jacobsen, Bovbjerg, & Redd, 1993). Patients with cancer who have high levels of trait anxiety have different patterns of immune system change and have an increased capacity for developing conditioned nausea and vomiting in response to chemotherapy treatment (Fredrikson, Furst, Lekander, Rotstein, & Bloomgren, 1993). Additionally, the interactions between trait anxiety and the endocrine system have been reported to delay recovery and increase pain from surgery (Mathews & Ridgeway, 1981).

High levels of trait anxiety have been commonly described as a catalyst for over-reporting of symptom experience because of hypervigilance to external and internal environments for cues of danger or threat (Gray, 1982; Watson & Clark, 1984). Due to the necessity of success in cancer treatments, accurate symptom reporting and accurate conceptualizing of reported symptoms is crucial. Although over-reporting has been associated with patients who have high levels of trait anxiety, there is no clear evidence that this increase in symptom reporting is an inaccurate report of somatic activity (Cameron et al., 1998).

Cameron and colleagues (1998) examined the relationship between trait anxiety and physiological symptom reporting in a group of women with breast cancer that were participating

in a placebo-controlled trial of tamoxifen. They were interested in the influence of trait anxiety on the symptomatic side effects induced by tamoxifen. High levels of trait anxiety were associated with greater and more rapid increases in symptom reporting, however, high symptom reports were of symptoms expected to emerge from tamoxifen treatment and could be attributed to actual experience and not to over-reporting. These findings suggested that trait anxiety may be associated with a differential symptom experience.

Further investigations again found that although patients with high trait anxiety did report more symptoms, when directly tested there remained no evidence to suggest that patients with high trait anxiety were less accurate in their symptom report. Rather, it was found that patients were reporting an increased number of vague symptoms of treatment (tiredness, mood swings, fatigue), but that their initial and retrospective reports of concrete symptoms (hair loss, vomiting, mouth sores) were neither increased nor inconsistent. Trait anxiety was consistently and positively related to vague symptom report but not to concrete symptom report. This increased report and symptom experience, further suggested trait anxiety as a marker of endocrine reactivity (Rabin, Ward, Leventhal, & Schmitz, 2001). This system may already be being taxed by a chronically alert stress system in patients with high trait anxiety, and the interaction of the chemotherapy regimen may actually produce a greater endocrine symptom response.

These results suggested that trait anxiety may also affect the pharmokokinetics of tamoxifen and may contribute to differential outcomes of tamoxifen therapy. This is important as the presence of anxiety may alter the amount of circulating drug, reduce overall effectiveness of estrogen blockade, and affect how bothersome side effects may be. Cameron and colleagues (2002) hypothesized that trait anxiety may be a marker for a physiological substrate moderating

the estrogen-agonist effects of tamoxifen on lumbar spine bone mineral density (BMD) and levels of sex hormone binding globulin (SHBG).

They found that tamoxifen produced greater preservation of BMD of the lumbar spine in trait anxious women. Trait anxiety was also associated with a suppression of tamoxifen-induced increases in SHBG. This finding is consistent with the physiology of chronic anxiety that when the body's stress system is alerted other systems, such as the reproductive system which is import in the production of SHBG, shut down. Although tamoxifen significantly increased SHBG levels regardless of anxiety level, women with high trait anxiety exhibited smaller mean changes in SHBG. These data suggested that trait anxiety might be an indicator of a physiological substrate that affects estrogen-agonist effect of tamoxifen and provided evidence regarding the physiological mechanisms underlying trait anxiety. Neuroendocrine processes associated with high trait anxiety appear to interact with estrogenic agents in ways that influence physiological outcomes.

Physiological theories of persistent (or trait-like) anxiety suggest that there is a chronic alertness of the "stress system" (Barlow, 2000). When activated this system produces several hormones including catecholamines and cortisol, that are known to suppress immune function, and to have a range of other effects. The endocrine system is thought to be influenced by trait anxiety (Sullivan et al., 2000) and it has been hypothesized that the endocrine system may participate in growth of human breast cancer (Henderson, 1995). The effects of chronic stress have also been shown to decrease the activity of natural killer cells and other anti-tumor effectors (Murphy, Lawrence, & Lenhard, 1995). The brain's stress response system, specifically the processes implicated in trait anxiety, may interact in important ways with systems that have been implicated in the initiation or progression of breast cancer and the general disease process.

3. Anxiety and Cortisol. Cortisol is a hormone secreted by the hypothalamic-pituitary-adrenal (HPA) axis, which participates in a number of important functions in humans. The HPA axis affects several physiological response systems, including the immune system (Lovallo, 1997). Cortisol reduction is thought to represent changes in the HPA axis. The HPA system and cortisol are known to play a key role in the adaptation of the body to both physical and psychological stress (Van der Pompe, Antoni, & Heijnen, 1996). Increases in cortisol levels are typical biochemical signs of stress (Bohnen, Nicolson, Sulon, & Jolles, 1991). Cortisol has been used as a measure of anxiety levels and also as a marker of immune functioning (Cohen et al., 1995) and evidence from animal paradigms has suggested the involvement of cortisol on immune cell distribution in response to acute stress (Ottaway & Husband, 1992).High trait anxiety has been associated with significant elevations in cortisol levels in healthy subjects (van Eck, Berkhof, Nicolson, & Sulon, 1996), and elevated levels of plasma and urinary cortisol have been associated with increased levels of daily stress and increased levels of perceived stress (Brantley, Dietz, McKnight, Jones, & Tulley, 1988; Pruessner, Hellhammer, & Kirschbaum, 1999).

Alterations in HPA axis function have been reported in women with breast cancer indicated by flattening of the circadian rhythm of cortisol secretion and elevated plasma cortisol levels (Cruess et al., 2000). Such differences may be due to disease and treatment effects on the endocrine system or to psychological stressors associated with a cancer diagnosis and treatment. Additionally, the progression of breast cancer has been associated with abnormal HPA responses (Van der Pompe et al., 1996). These data suggest that cortisol levels may be related to disease status and that breast cancer may be associated with a hyperactive adrenal gland resulting in chronically increased cortisol levels.

B. Summary, Goals, and Hypotheses

The critical implication of these findings is the suggestion that trait anxiety may affect overall well-being as well as the course of disease in patients with breast cancer. Despite growing evidence of the importance of such variables, published reports focusing on the psychological correlates and physiological distress associated with trait anxiety in this medical population are limited. In clinical decision making it is likely that there is little differentiation made between trait and state anxiety. Trait anxiety may often be overlooked because it does not meet diagnostic criteria for an anxiety disorder and may not be differentiated from state anxiety when more acute measures of anxiety are used to assess distress. A thorough and accurate understanding of the psychological and physiological distress variables associated with trait anxiety will assist to inform treatment choices, adjunct psychological interventions strategies, and other clinical decision-making in patients with breast cancer. This research will attempt to provide a better understanding of these factors.

It was hypothesized that women with breast cancer would report higher levels of acute anxiety at diagnosis than at 3 or 6 months post-diagnosis, but trait anxiety would not fluctuate over time. It was also expected that trait anxiety at diagnosis would more strongly predict acute symptoms of anxiety at 3 and 6 months post-diagnosis than would acute anxiety at diagnosis. It was expected that measures of psychosocial distress (e.g., general stress, depression, general and medical worry, quality of life, and symptom reporting) would be increased at diagnosis compared to 3 and 6 months post-diagnosis, with trait anxiety at diagnosis accounting for more of the variance in psychosocial distress over time than would state anxiety at diagnosis. Quality of life directly influenced by physical functioning was expected to fluctuate during this time period due to the course of cancer treatments. Lastly, it was expected that increased levels of

physiological distress at diagnosis as measured by salivary cortisol levels would be related to self-report measures of anxiety at each time point and that salivary cortisol levels at diagnosis would account for a significant amount of the variance in self-reported anxiety at diagnosis and 3 and 6 months after diagnosis.

II. METHODS

In designing this study, careful attention was paid to difficulties in accrual of subjects, limiting subject response burden, and minimizing participant attrition. All eligible patients who were willing to consider participation met individually with the primary investigator who explained the study and obtained informed consent. Sixty-five participants were recruited in an eleven-month period and each subject was followed for six months at three month intervals beginning at baseline, time 1, (not more than four weeks post-diagnosis).

A. Participants

The 65 women with newly diagnosed primary stage breast cancer were recruited and provided informed consent for this study. Fifty-eight women completed all the study materials at Time 1. Participants ranged in age from 35-78 with a mean age of 56 (*SD* = 10). Participants were predominantly Caucasian (98%); reflecting the population seen at Magee Women's Hospital. Of these women, 60% were married, 12% were single, 19% were separated or divorced, and 9% were widowed. Sixty-five percent reported having a yearly family income of greater than $30,000 and 83% reported having some educational training beyond high school. About half reported working full or part time (53%), and 25% reported being retired, 15% were homemakers, and 7% were unemployed or on disability. Twenty-seven percent of this sample reported having at least one child under 18 living at home. The participants reported religious preferences were Catholic (42%), Protestant (48%), Orthodox (2%), and no affiliation (8%).

1. Cancer Information. Thirteen percent of women in this study were diagnosed with in situ (stage zero) breast cancer, 46% with stage one breast cancer, 30% with stage two breast cancer, and 11% with stage three breast cancer. Lumpectomy/segmental mastectomies were performed on 78% of these women, 15% had a total mastectomy, 3.5% had a modified radical mastectomy, and 3.5% had a bilateral mastectomy. Three months post-diagnosis, 69% of participants were receiving chemotherapy and 32% were receiving radiation treatment. Among participants, a total of 39% reported having a positive family history of breast cancer diagnosis in their mother (14%), a sister (9%), an aunt (26%), and/or child (2%). Regarding deaths due to breast cancer, 22% reported having a mother, grandmother, aunt, or sister die as a result of breast cancer.

2. Medical and Health Information. Nineteen percent of these women reported they were pre-menopausal, 15% reported they were peri-menopausal, 61% reported they were post-menopausal, and 5% did not report this information. Of the 58 participants, 83% reported no current psychological treatment, 16% were currently taking medication for psychological reasons, and 1% reported current psychological therapy. Reasons listed for psychological treatment were anxiety, depression, or relationship difficulties. Ten percent of participants reported being current smokers, 42% reported currently using alcohol at least occasionally, and 53% reported daily caffeine use. Regarding exercise, 10% reported never exercising, 44% reported that they exercised 1-2 times per week, 36% reported exercising 3-4 times per week, and 10% reported exercising 5 or more times per week.

3. Exclusion Criteria. Participants were eligible if they were over the age of 18, if this was their first diagnosis of cancer, if they had suspected early clinical stage (0,1,2) breast cancer, if they were able to read and write English, and if they were capable of informed consent. Individuals

were excluded if they exhibited a predominant medical condition other than the new breast cancer diagnosis, if there was behavioral evidence of significant psychiatric illness, psychosis, or organic brain syndrome, or if there was evidence of active suicidal ideation.

B. Procedures

All potential participants had been informed of their new diagnosis within the previous four weeks and had not yet had surgery or begun their cancer treatment regimen. Medical staff identified potential participants. If patients agreed to speak with an investigator, the patient was then informed of the purpose and requirements of this study and if willing to participate, patients were provided with an informed consent. After signing the informed consent, patients were given a packet of questionnaires and other study materials. Packets included a diary to record details for the cortisol measurements, materials to collect cortisol samples, and, if desired by the participant, a beeper was included to remind patients when to do their cortisol collections. Each set of questionnaires required approximately 45 minutes to complete.

Procedures for collecting saliva samples for measuring cortisol were explained. Each packet also contained written procedural instruction as well as specific instructions about the use of salivettes and recording diaries. The diaries served to remind participants of sampling times and to record and verify actual sampling times. Participants were asked to complete salivary cortisol samples for one day within one hour of waking, at 11 a.m., 3 p.m., 6 p.m., and 9 p.m. Patients were contacted at their beeper at each collection time to prompt them to complete their cortisol sampling if they desired. Participants were requested to complete all study requirements within four days and to return all study forms in a provided postage paid self-addressed envelope.

The second and third packets were sent to participants that completed the previous time point with a postage-paid return addressed envelope. Patients were contacted by phone two weeks prior to the mailing of each packet and reminded to expect questionnaires and saliva cortisol equipment by mail and to complete and return questionnaires and cortisol samples within four days after receiving them. Participants received $20.00 upon the return of each packet. Information on disease characteristics, treatment information, patient blood pressure reading, and height and weight measurements was collected from the patient's medical chart.

C. Measures

1. Control variables. All participants completed a sociodemographic questionnaire designed specifically for this study at all time points. Variables assessed to describe patient demographic characteristics were age, marital status, education, income, and number of children at home. The use of psychiatric medication, family cancer history, and health behavior information was obtained. Disease characteristics were determined through review of medical charts at Magee Women's Hospital. These variables were measured for descriptive purposes, as well as to allow for examination of potential alternative explanations for findings.

2. Predictor and Outcome Variables. Each variable was measured at all three time points in the study. Anxiety was measured using the State-Trait Anxiety Inventory (STAI; Spielberger et al., 1970). The STAI consists of two scales of items, with each scale comprised of 20 questions (40 total). One scale specifically measures state anxiety and the other scale measures trait anxiety. Superior internal consistency is shown with the alpha coefficients for both scales being uniformly high with median coefficients of 0.93 for the state anxiety scale and 0.90 for the trait

anxiety scale. Test-retest stability is also relatively high for the trait anxiety scale with median stability coefficients as high as 0.77. Test-retest stability is relatively low for the state anxiety scale, as would be expected, because of its purpose to assess transitory changes in anxiety (Spielberger, Sydeman, Owen, & Marsh, 1999).

The Depression Anxiety Stress Scale (DASS; Lovibond & Lovibond, 1995) was used to measure depression and distress. The anxiety scale (DASS-A) of this measurement was designed to eliminate symptoms of anxiety that could potentially overlap with depression and is conceptualized as a "pure" measure of anxiety. The DASS has 42 questions answered on a scale of 0 to 3. Analyses indicate that the DASS is a reliable and valid measure of anxiety, depression, and stress. The DASS's three-factor structure discriminates between anxiety and depression better than other commonly used measures (Brown, Chorpita, Korotitsch, & Barlow, 1997).

Worry was measured with the Worry Domains Questionnaire (WDQ; Tallis, Davey, & Bond, 1994). It is a widely used instrument that assesses the amount of non-pathological worry across five domains of everyday concern: relationships, lack of confidence, aimless future, work, and financial issues. Research has demonstrated that the WDQ shows high reliability and substantial validity, with good internal consistency (Cronbach's $\alpha > .90$) and with test-retest stability across four weeks being 0.85 (Stober & Joormann, 2001). Five questions were added to the WDQ that specifically addressed patient's worry of disease recurrence, apprehensiveness about treatment procedures and tests, and disease worry. In this study the alpha levels for these five questions were .91, .90, and .84 at each time point respectively. The medical specific questions were not added into the total score, but were considered separately in all analyses.

Quality of life was measured using the Medical Outcomes Study Short Form (MOS; Ware & Sherbourne, 1992). Quality of life was used as an outcome variable and was assessed with this

brief, comprehensive multi-item instrument measuring eight functional domains relating to health. Domains included: limitations in physical activities due to health problems, limitations in social activities due to physical or emotional problems, limitations in usual role activities due to physical health problems, bodily pain, limitations in usual role activities because of emotional problems, vitality, and general health perceptions. The MOS also generated one global score for mental health and one for physical health. The MOS measures have acceptable reliability for group comparisons across age, gender, ethnicity, education, and disease groups (McHorney, Ware, Lu, & Sherbourne, 1994). Responses were scored on a 5 point likert scale and transformed to a 0-100 scale. These scores were reversed in this study for consistency among measures so that 0 = best health and 100 = poorest health.

The Symptom Checklist 90, Revised (SCL-90; Derogatis, 1977) was used as an outcome variable to provide a global index of symptom reporting as well as intensity of distress scores for nine clinical problem dimensions. The subscales include somatic complaints, interpersonal problems, depression, anxiety, hostility, paranoia, phobia, and psychotic symptoms. Ninety items are rated on a five-point Likert scale of distress ranging from 0 = not at all to 5 = extremely. Internal consistency for the nine subscale ranges from .78 to .90 and one week test-retest reliability ranges between .78 and .90 (Derogatis & Melisaratos, 1983).

Physiological symptoms of distress were assessed through the measurement of salivary cortisol. Salivary cortisol was used as collected from participants at each assessment time point in the study and served as a physiological marker of anxiety. Historically it has been difficult to interpret the association of stress and cortisol levels because this measure has traditionally been collected with a blood sample. Blood sampling provides a very time limited “snapshot” of cortisol levels and some stress may be induced by vein puncture and the medical procedure itself.

However, salivary cortisol is collected several times during the day, is less reactive, and has been shown to reliably reflect levels of unbound cortisol in the blood and raised levels have been found to be associated with stress in normal subjects (Kirschbaum & Hellhammer, 1994).

Sampling spaced across the waking day was done to allow for the evaluation of the shape and periodicity of cortisol production over time, while avoiding typical lunch and dinner hours, reducing the potential effects of eating on cortisol levels. Collection of each cortisol sample requires very little time or burden; participants chew on a sterilized cotton wedge for 30 seconds, place it in a small test tube, and insert this into a salivette cover. The completed samples were returned to the medical center within 36 hours and centrifuged and frozen at –70 º C. Assessing for adherence to cortisol sampling is difficult, however, there is a normal diurnal curve that is expected throughout the day. In this sample, approximately 80% of participants with completed samples had results that followed this general pattern.

III. DATA ANALYSES AND RESULTS

Data were analyzed with SPSS version 11.0.1. Initially, the distributions, descriptive statistics (see Tables 1-4), and intercorrelations of all variables were examined to determine normality and multicollinearity. Additionally, intercorrelations among the various dependent variables were examined and any variables that were consistently and highly related to one another were combined or analyzed simultaneously as appropriate. In this way, the overall number of dependent variables as well as number of comparisons was reduced.

To correct for non-normal distributions, square-root transformations were used on all of the psychosocial self-report measures and logarithmic transformation was used on the cortisol variable. Zero-order correlations coefficients between self-reports and background variables (age, marital status, income, education, children in the home, psychological medication use), health behavior variables (exercise frequency, eating habits, smoking, alcohol use frequency, caffeine use frequency), family breast cancer history (mother, aunt, or sibling diagnosis and family death due to breast cancer), and medical variables (breast cancer stage, surgery type, radiation and chemotherapy treatment for cancer, blood pressure, weight) were examined to identify potential covariates. Significant correlations are listed in Tables 5-11. Most of these correlations were relatively small, accounting for less than 25% of the variance in any measure and the pattern of relationship was consistent with previous research. Based on these relationships, variables were used as covariates in analyses as appropriate.

Table 1

Means and Standard Deviations of Self-Report Full Scales for Completers at Each Time

Variable	Diagnosis *n* = 58		3 Months *n* = 51		6 Months *n* = 42	
	M	*SD*	*M*	*SD*	*M*	*SD*
Trait Anxiety	36	9.8	36	10	34	9
State Anxiety	37	14	33	11	30	10
DASS Anxiety	4.6	6.0	3.3	4.9	2.8	3.8
DASS Depression	6.0	7.1	5.0	6.5	3.9	5.3
DASS Stress	9.1	7.2	7.7	8.3	6.7	5.7
Worry Total	13.3	15.9	13.9	16.4	14.5	16.4
Worry Medical	10.5	5.8	7.8	5.4	6.9	4.2
SCL-GSI	.49	.44	.48	.48	.41	.40
MOS Mental Health	30	21	26	21	20	18
MOS General Health	31	18	35	18	31	16

Table 2

Means and Standard Deviations of Variables Among Full Completers (n = 42)

Variable	Diagnosis		3 Months		6 Months	
	M	*SD*	*M*	*SD*	*M*	*SD*
Trait Anxiety	36	11	36	10	34	9
State Anxiety	37	15	33	12	30	10
DASS Anxiety	4.4	6.1	3.0	4.3	2.8	3.8
DASS Depression	6.1	7.8	4.6	6.2	3.9	5.3
DASS Stress	8.9	7.3	6.8	6.3	6.7	5.7
Worry Total	13.7	16.6	12.8	15.1	14.5	16.4
Worry Medical	10.3	5.7	7.7	5.1	6.9	4.2
SCL-GSI	.50	.48	.45	.45	.41	.40
MOS Mental Health	29	21	24	20	20	18
MOS General Health	31	19	35	18	31	16

Table 3

Means and Standard Deviations of WDQ Subscales and SCL-90-R Subscales (n = 42)

Variable	Diagnosis		3 Months		6 Months	
	M	*SD*	*M*	*SD*	*M*	*SD*
WDQ Finances	4.07	5.01	3.62	4.40	4.64	5.37
WDQ Work	2.81	3.32	2.83	3.27	3.17	3.35
WDQ Aimless Future	2.86	3.85	2.48	3.49	2.81	3.70
WDQ Lack Confidence	2.69	3.70	2.33	3.33	2.55	3.33
WDQ Relationship	1.29	3.01	1.55	2.74	1.31	3.00
SCL Somatic	.49	.52	.62	.50	.63	.54
SCL OCD	.79	.67	.63	.58	.61	.53
SCL Interpersonal	.37	.56	.43	.60	.37	.47
SCL Depression	.68	.70	.62	.58	.53	.60
SCL Anxiety	.58	.68	.30	.49	.26	.45
SCL Hostility	.25	.44	.24	.37	.23	.35
SCL Phobia	.11	.34	.13	.32	.10	.30
SCL Paranoid	.28	.35	.26	.45	.23	.36
SCL Psychotic	.34	.41	.22	.34	.19	.31

Table 4
Means and Standard Deviations of MOS Scales (n = 42)

Variable	Diagnosis		3 Months		6 Months	
	M	*SD*	*M*	*SD*	*M*	*SD*
Physical Functioning	15	21	27	25	26	21
Physical Role	36	36	68	41	47	37
Body Pain	23	19	34	26	33	24
General Health	30	17	35	18	31	16
Vitality	42	20	53	23	47	20
Social Functioning	20	20	29	26	22	25
Emotional Role	37	41	30	42	22	32
Mental Health	29	21	24	20	20	18
Health Transitions	97	.84	98	1.0	98	1.1

Table 5

Correlations between background variables and self-report measures (n = 42)

	Age	Marriage	Income	Psych Med	Exercise	Eat Habits	Smoking
Trait Anxiety							
Diagnosis	.05	-.29	-.12	.45*	-.23	.22	.29
3 Month	-.10	-.23	-.04	.49*	-.12	.07	-.28
6 Month	.01	-.32*	-.22	.29	.12	-.08	-.09
State Anxiety							
Diagnosis	-.21	-.07	-.01	.21	-.17	-.07	-.21
3 Month	.07	-.13	-.01	.17	-.17	.20	-.31*
6 Month	.03	-.33*	-.16	.11	-.05	.15	-.17
DASS Anxiety							
Diagnosis	-.17	-.05	-.01	.45**	-.37*	-.17	.01
3 Month	-.09	-.24	-.22	.42*	-.23	.14	-.10
6 Month	.01	-.11	-.10	.24	-.24	.42**	.06
DASS Stress							
Diagnosis	-.24	-.24	-.07	.50**	.19	-.04	-.05
3 Month	-.17	-.06	.14	.40*	.03	.09	-.08
6 Month	-.20	-.30	-.15	.23	.08	-.03	.29
DASS Depression							
Diagnosis	-.29	-.24	-.07	.46**	-.19	-.03	-.09
3 Month	-.30	-.30	.03	.39*	-.10	.20	-.20

Table 5 (continued).

6 Month	-.15	-.41**	-.26	.20	-.20	-.01	.07
WDQ Total							
Diagnosis	-.08	-.37*	-.20	.35*	-.09	.07	-.16
3 Month	-.07	-.33*	-.18	.23	-.19	.16	-.21
6 Month	-.11	-.44**	-.36**	.14	-.07	.19	-.03
WDQ Medical							
Diagnosis	-.31	-.21	.15	.15	.01	.20	-.02
3 Month	-.32*	-.22	.01	.04	.10	.23	.01
6 Month	-.43**	-.33*	-.11	.03	-.18	-.02	-.04
MOS Mental Health							
Diagnosis	-.35*	-.06	.12	.36*	-.18	.10	-.12
3 Month	-.26	-.17	-.04	.26	-.25	.25	-.24
6 Month	-.22	-.19	-.06	.18	.11	.26	-.17
SCL GSI							
Diagnosis	-.12	-.21	-.07	.48**	-.24	-.01	-.18
3 Month	-.07	-.32*	-.15	-.32*	-.19	.21	-.22
6 Month	-.01	-.42**	-.35*	.20	-.02	.13	-.17

*p < *.05.* **p < *.01.* ***p < *.001.*

Table 6

Correlations between medical variables and self-report measures (n = 42)

	BC Stage	Systolic	Weight	Radiation	Chemotherapy
Trait Anxiety					
Diagnosis	-.30	.30	.27	-	-
3 Month	-.34*	.46*	.39*	-.11	.05
6 Month	-.40*	.29	.28	.23	-.33*
State Anxiety					
Diagnosis	-.20	.29	.11	-	-
3 Month	-.43**	.60**	.33*	-.06	-.20
6 Month	-.28	.44*	.22	.06	-.22
DASS Anxiety					
Diagnosis	.05	.10	.03	-	-
3 Month	-.25	.30	.40*	-.03	-.03
6 Month	-.07	.25	.19	-.11	.01
DASS Stress					
Diagnosis	-.14	.24	.09	-	-
3 Month	-.37*	.44*	.22	.43**	-.10
6 Month	-.29	.27	.06	.25	-.04
WDQ Total					
Diagnosis	-.26	-.04	.40**	-	-
3 Month	-.18	.26	.35*	.02	-.09
6 Month	-.17	.15	.28	-.12	-.26

Table 6 (continued).

MOS Gen Health					
Diagnosis	-.09	.44*	.11	-	-
3 Month	.04	.34	.14	.16	.21
6 Month	.07	.40*	.11	-.06	.19
MOS Men Health					
Diagnosis	-.08	.10	.08	-	-
3 Month	-.16	.43*	.18	-.02	-.09
6 Month	-.31*	.29	.02	-.01	-.22

*p < *.05.* **p < *.01.* ***p < *.001.*

Table 7

Correlations between background variables and WDQ Subscales (n = 42)

	Marriage	Income	Psych Med	Stage	Chemo	Systolic	Weight
Finances							
Diagnosis	-.33*	-.26	.12	-.19	-	.07	.38*
3 Month	-.26	-.30	.05	-.10	-.06	.02	.30
6 Month	-.30	-.40*	-.09	-.19	-.17	-.05	.17
Work							
Diagnosis	-.19	-.07	.41**	-.23	-	.36	.22
3 Month	-.13	.03	.25	-.17	.05	.34	.25
6 Month	-.26	.18	.15	-.07	-.19	.19	.15
Aimless Future							
Diagnosis	-.28	-.02	.28	-.16	-	.14	.33*
3 Month	-.25	-.06	.20	-.11	.01	.14	.38*
6 Month	-.42**	-.31	.05	-.16	.13	.10	.35*
Lack Confidence							
Diagnosis	.08	-.33*	.46**	-.31	-	.41*	.36*
3 Month	-.40**	-.27	.30	-.34*	-.38*	.42*	.30
6 Month	-.46**	-.35*	.30	.26	.26	.42*	.27
Relationship							
Diagnosis	.08	-.31	.31*	-.24	-	.25	.48**
3 Month	-.38*	-.22	.32*	-.11	-.01	.23	.28
6 Month	-.40**	.27	.28	-.16	-.09	.20	.40**

Table 8

Correlations between background variables and MOS Subscales (n = 42)

	Age	Income	Exercise	Eating Habits	Alcohol Use	Caffeine Use
Physical Function						
Diagnosis	.52***	-.14	-.30	-.05	-.33*	-.05
3 Month	-.031	-.28	-.24	.12	-.30	.01
6 Month	.06	-.37*	-.32*	.01	.52***	-.54***
Physical Role						
Diagnosis	-.24	.08	-.05	-.08	-.06	.07
3 Month	-.19	-.03	-.38*	-.02	-.24	-.09
6 Month	-.06	-.05	-.22	-.02	-.43**	-.30*
Body Pain						
Diagnosis	.14	-.38*	-.23	-.09	-.10	.11
3 Month	-.30	.13	-.20	.14	-.13	.10
6 Month	-.11	-.31	-.17	.13	-.19	-.38*
Vitality						
Diagnosis	-.31	.24	-.02	-.07	.12	.03
3 Month	-.38*	-.06	.32*	.02	-.03	.06
6 Month	-.27	.97	-.17	.11	-.15	-.35*
Social Function						
Diagnosis	-.25	-.21	.03	.05	-.39*	.05
3 Month	-.32*	.50	-.21	-.07	-.42**	-.08

Table 8 (continued).

6 Month	-.31*	-.20	-.21	-.09	-.58***	-.23
Emotional Role						
Diagnosis	-.31*	-.02	.03	.02	.10	.23
3 Month	.08	-.16	.39*	.14	-.20	.20
6 Month	-.06	-.16	.04	.46**	.06	.20

*p < *.05.* **p < *.01.* ***p < *.001.*

Table 9

Correlations between medical and family breast cancer history variables and MOS Subscales (n = 42)

	Systolic	Diastolic	Weight	Radiation	Surgery	Aunt Breast Cancer	Family Breast Cancer	Family Death Cancer
Physical Function								
Diagnosis	.12	.42*	.37*	-	-	.26	.19	.32
3 Month	-.02	-.35	.27	-.18	-.04	.48**	.35*	.21
6 Month	-.21	-.43*	.21	-.14	.19	.24	.25	.09
Physical Role								
Diagnosis	.13	-.24	.29	-	-	.08	-.02	.13
3 Month	-.05	-.27	-.05	-.16	.34*	.15	.04	-.17
6 Month	-.22	-.20	-.12	-.24	-.18	.01	.06	-.12
Body Pain								
Diagnosis	.17	-.21	.37*	-	-	.27	.02	.30
3 Month	-.01	-.49**	.22	-.06	-.09	.25	.09	.02
6 Month	.11	-.38*	.10	.01	.18	.06	.07	-.20
Vitality								
Diagnosis	.19	-.03	.17	-	-	.04	-.15	-.17
3 Month	.11	-.24	.07	.17	.16	.01	-.01	-.17
6 Month	.08	-.32	.01	-.33*	.20	-.05	.03	-.36*

Table 9 (continued).

Social Function								
Diagnosis	-.04	-.37*	.24	-	-	.05	-.09	.05
3 Month	-.04	-.28	-.03	-.07	-.09	.14	.09	-.23
6 Month	-.26	-.21	-.07	-.20	-.25	.30	.33*	-.09

*p < *.05.* **p < *.01.* ***p < *.001.*

Table 10

Correlations between background variables and SCL-90 Subscales (n = 42)

	Marriage	Income	Psych Med	Eating Habits	Alcohol Use
OCD					
Diagnosis	-.27	.03	.49**	.01	-.05
3 Month	-.30	-.01	.22	.22	.02
6 Month	-.42**	-.23	.23	.07	-.09
Interpersonal					
Diagnosis	-.39*	-.34*	.32*	.03	-.32*
3 Month	-.44**	-.30	.34*	.31*	-.26
6 Month	-.55**	-.48**	.15	-.02	-.23
Depression					
Diagnosis	-.23	.08	.43**	-.04	-.08
3 Month	-.26	-.08	.36*	.14	-.16
6 Month	-.41**	-.30	.21	.17	-.22
Anxiety					
Diagnosis	-.22	.10	.48**	-.10	-.03
3 Month	-.18	-.04	.34	.13	-.13
6 Month	-.32*	-.25	.21	.16	-.02
Hostility					
Diagnosis	-.13	-.19	.36*	-.09	-.39*
3 Month	-.10	-.37*	.17	-.02	-.06

Table 10 (continued).

6 Month	-.24	-.37*	.01	.05	-.01
Paranoid					
Diagnosis	-.32*	-.18	.32*	.12	-.31
3 Month	-.40**	-.37*	.23	.04	-.33*
6 Month	-.42**	.41*	-.01	-.15	-.35*
Psychotic					
Diagnosis	-.33*	.17	.42**	.03	-.16
3 Month	-.43**	-.28	.33*	.27	-.20
6 Month	-.42**	-.36*	.26	.02	-.24
Phobia					
Diagnosis	-.19	-.13	.53**	-.07	-.19
3 Month	.33*	-.26	.49**	.10	-.25
6 Month	.37*	-.33*	.42**	.18	-.30

*p < *.05.* **p < *.01.* ***p < *.001.*

Table 11

Correlations between medical variables and SCL-90 Subscales (n = 42)

	BC Stage	SBP	Weight	Chemo
OCD				
Diagnosis	-.14	.15	.14	-
3 Month	-.33*	-.52**	.29	-.13
6 Month	-.25	.35	.24	-.22
Interpersonal				
Diagnosis	-.40*	.36	.43**	-
3 Month	-.25	.33	.29	-.15
6 Month	-.24	.37	.35*	-.34*
Anxiety				
Diagnosis	-.13	.14	-.05	-
3 Month	-.25	.37	.19	-.09
6 Month	-.31*	.40*	.18	.32*
Paranoid				
Diagnosis	-.28	.24	.21	-
3 Month	-.34*	.15	.28	-.26
6 Month	-.31*	.31	.34*	-.31*
Psychotic				
Diagnosis	-.26	.28	.30	-
3 Month	-.28	.37*	.35*	.15

Table 11 (continued).

6 Month	-.34*	.30	.43*	-.32*

*p < *.05.* **p < *.01.* ***p < *.001.*

A. Participant Statistics

Sixty-five women were recruited for this study. A total of 58 women completed Time 1, 51 women completed Time 2, and 42 completed Time 3. Differences between completers and noncompleters at each time point were analyzed using one-way analyses of variance (ANOVA) for continuous dependent variables and chi-squares for categorical dependent variables. There were no significant differences in Time 1 self-report measures between completers and noncompleters at Time 2 or 3. There were no differences between those who completed and those that did not at any time points on age, marriage, income, education, number of children at home, psychiatric medication use, family history of breast cancer history, or current breast cancer staging. Surgery type did not differentiate groups at Time 1 or Time 3, however, a difference emerged between surgery type and completion at Time 2 ($\chi^2 = 11.4$, $p = .01$), although due to small cell sizes this should be interpreted with caution. Table 12 contains a summary of these results.

Table 12

Completers and Non Completers at Time 2 Based on Surgery Type

	Segmental Mastectomy	*Total Mastectomy*	*Modified Radical Mastectomy*	*Bilateral Mastectomy*
Completer (n = 50)	42 (84%)	6 (12%)	2 (4%)	0
Non Completer (n = 10)	6 (60%)	2 (20%)	0	2(20%)

B. Aim 1

The primary aim of this study was to evaluate the course of trait anxiety and acute symptoms of anxiety in patients newly diagnosed with breast cancer. It was expected that participants would report higher levels of acute anxiety at diagnosis than 3 or 6 months post-diagnosis. However, trait anxiety was not expected to decrease overtime from initial report. Furthermore, trait anxiety at diagnosis was expected to more strongly predict acute symptoms of anxiety overtime than was state anxiety at diagnosis.

Only participants who completed all anxiety measures (i.e., STAI, DASS) at all time points were included in these analyses except where noted. Correlational analyses were conducted to examine the bivariate association between trait anxiety and acute anxiety over-time. As depicted in Table 13, the correlations between trait anxiety and acute anxiety (STAI and DASS) over time ranged from low to high (r = .14 and r = .73). In the first six months after breast cancer diagnosis, there was a clear pattern of bivariate association between trait anxiety and acute anxiety. Over time trait anxiety at diagnosis maintained a stronger correlational relationship with acute measures of anxiety than did state anxiety at diagnosis.

Table 13

Correlations of Trait and Acute Anxiety at Diagnosis with Acute Anxiety Over Time

Anxiety variable	*Trait Anxiety at Diagnosis*		
	Diagnosis	3 months	6 months
State anxiety	.729***	.731***	.509**
DASS anxiety	.639***	.454**	.330*
	State Anxiety at Diagnosis		
State anxiety		.662***	.454**
DASS anxiety	.597***	.327*	.135(NS)

*p < *.05*. **p < *.01*. ***p < *.001*.

Within subjects ANOVA analyses were done to examine changes in trait anxiety and acute anxiety over time. Pairwise analyses of means used paired observation t-tests coupled with a Bonferroni control for experiment wise error rates. Covariates were used in these analyses as indicated by the results of the bivariate correlations (Tables 5 and 6). Figure 1 shows the changes in trait and state anxiety over time. When examining trait anxiety changes over time, psychiatric medication use and cancer staging were used as covariates. As expected, there were no main time effects on trait anxiety $F(2, 37) = .020$, $p = .980$. There was a main effect of time on state anxiety $F(2, 40) = 4.3$, $p = .020$. State anxiety was significantly higher at diagnosis than 6-months post-diagnosis ($M = 6.0$, $SE = .18$ and $M = 5.5$, $SE = .13$, respectively), $F(1.67, 68.45) = 6.50$, $p = .004$. When controlling for the use of psychiatric medication, there was no main effect

of time on the DASS-A. Without this covariate, however, there was a main effect for time on the DASS-anxiety measure over time $F(2, 40) = 3.2$, $p = .05$. Pairwise comparisons did not reveal any statistically significant differences. This relationship is displayed in Figure 2.

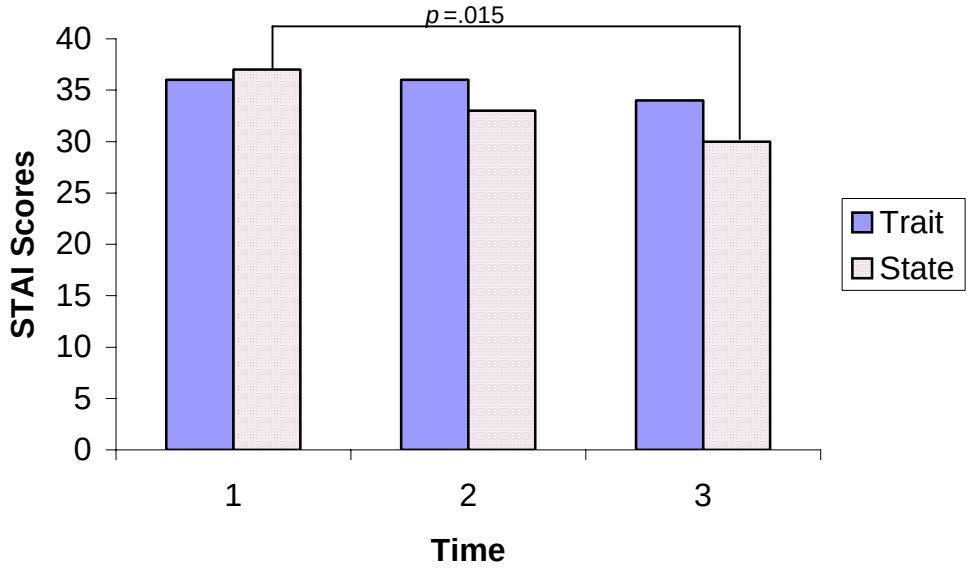

Figure 1. Changes in state and trait anxiety from diagnosis to 3 and 6 months post diagnosis.

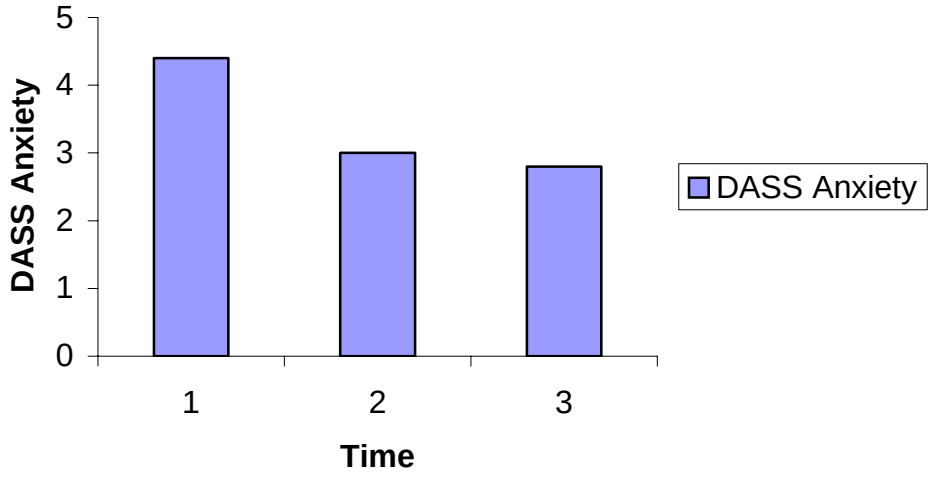

Figure 2. Changes in DASS-A from diagnosis to 3 and 6 months post diagnosis

As depicted in Tables 14, 15, and 16, a series of regression equations were constructed to examine the predictive relationship between trait anxiety and acute anxiety in the first six months following breast cancer. Based on the zero-order correlations of variables among participants used in these analyses, psychiatric medication use, cancer staging, and marital status were controlled for as appropriate and are noted when used. To control for shared variance between the dependent variables, trait and state anxiety were simultaneously entered into all equations. Additionally, due to the moderate correlations between the measures of trait anxiety and acute anxiety and the associated problem of multicollinearity of the predictors the regression analyses were additionally run with each type of anxiety entered separately. Any differences between the analyses, with the variables entered together versus separately are noted. In addition to analyzing the results for the 42 full completers, when investigating the relationship between only Time1 and Time 2 variables, analyses were run using participants that completed both Time 1 and Time 2, but not Time 3 ($n = 51$).

As shown in Table 14, when predicting anxiety 3 months after diagnosis, psychiatric medication use and cancer staging were controlled in all analyses. The regression equation was significant for predicting state anxiety 3-months post-diagnosis, accounting for 63% of the variance. Trait anxiety at diagnosis was the strongest predictor of state anxiety 3 months after diagnosis and cancer staging also significantly contributed to the model. The contribution of state anxiety at baseline and psychiatric medication were no more than expected by chance. When trait ($\beta = .650$, $sr^2 = .44$, $p < .001$) and state anxiety ($\beta = .555$, $sr^2 = .39$, $p < .001$) at diagnosis were entered separately, both were significant predictors of state anxiety 3 months post-diagnosis. Although the regression equation was significant to predict DASS-A 3 months post-diagnosis, none of the individual predictors reached statistical significance. When trait ($\beta = .365$,

sr^2 = .114, p = .035) and state anxiety (β = .227, sr^2 = .056, p = .147) at diagnosis were entered separately, only trait anxiety was a significant predictor of DASS-A 3 months post-diagnosis.

When entering all completers data in to the model, as shown in Table 15, trait anxiety at diagnosis was the strongest predictor of state anxiety 3 months after diagnosis, state anxiety and cancer staging also significantly contributed to the model. The contribution of psychiatric medication was no more than expected by chance. When trait (β = .640, sr^2 = .45, p < .00) and state anxiety (β = .592, sr^2 = .41, p < .00) at diagnosis were entered separately, both were significant predictors of state anxiety at 3 months post-diagnosis. The predictor model was significant to predict DASS-A 3 months post-diagnosis, although none of the individual predictors reached statistical significance. When trait (β = .402, sr^2 = .16, p = .006) and state anxiety (β = .293, sr^2 = .09, p = .041) at diagnosis were entered separately, both were significant predictors of DASS-A at 3 months post-diagnosis.

When predicting anxiety 6-months post-diagnosis, based on bivariate correlation analyses, psychiatric medication use and marital status were controlled for in all analyses (Table 16). Six-months post-diagnosis, the regression equation was significant in predicting state anxiety accounting for 33% of the variance. Neither type of anxiety at diagnosis uniquely significantly contributed to this equation. When trait (β = .515, sr^2 = .21, p = .003) and state anxiety (β = .428, sr^2 = .19, p = .004) at diagnosis were entered separately, both were significant predictors of state anxiety 6 months post-diagnosis. The regression equation did not significantly predict DASS-A 6 months post-diagnosis. When entered separately, neither trait nor state anxiety at diagnosis significantly predicted DASS-anxiety 6-months post-diagnosis.

Table 14

Regressions Predicting Anxiety at 3 months for completers of all time points (n = 42)

Variable	*β*	sr^2	p
STAI-State[a]			
Trait Anxiety at Diagnosis	*.417*	*.14*	*.021*
State Anxiety at Diagnosis	*.285*	*.08*	*ns*
Psychiatric Medication Use	*.082*	*.01*	*ns*
Cancer Staging	*-.246*	*.13*	*.026*
DASS Anxiety[b]			
Trait Anxiety at Diagnosis	*.380*	*.06*	*ns*
State Anxiety at Diagnosis	*-.018*	*.00*	*ns*
Psychiatric Medication Use at Diagnosis	*.127*	*.02*	*ns*
Cancer Staging	*-.131*	*.02*	*ns*

[a] $F(4,36) = 15.37$, $p < .001$, $R^2 = .63$

[b] $F(4,36) = 2.88$, $p = .036$, $R^2 = .24$

Table 15

Regressions Predicting Anxiety at 6 months for all Time 1 and Time 2 Completers (n = 51)

Variable	*β*	sr^2	p
STAI-State[a]			
Trait Anxiety at Diagnosis	*.411*	*.14*	*.009*
State Anxiety at Diagnosis	*.298*	*.09*	*.048*
Psychiatric Medication Use	*.027*	*.00*	*ns*
Cancer Staging	*-.263*	*.13*	*.011*
DASS Anxiety[b]			
Trait Anxiety at Diagnosis	*.393*	*.07*	*ns*
State Anxiety at Diagnosis	*.012*	*.00*	*ns*
Psychiatric Medication Use at Diagnosis	*.019*	*.00*	*ns*
Cancer Staging	*-.164*	*.06*	*ns*

[a] $F(4,44) = 16.14$, $p < .001$, $R^2 = .60$

[b] $F(4,44) = 3.16$, $p = .023$, $R^2 = .22$

Table 16

Regressions Predicting Anxiety 6 months post diagnosis (n = 42)

Variable	*β*	sr^2	p
STAI-State[a]			
Trait Anxiety at Diagnosis	*.326*	*.05*	*ns*
State Anxiety at Diagnosis	*.218*	*.03*	*ns*
Psychiatric Medication Use	*-.088*	*.01*	*ns*
Marital Status	*-.222*	*.06*	*ns*
DASS Anxiety[b]			
Trait Anxiety at Diagnosis	*.415*	*.06*	*ns*
State Anxiety at Diagnosis	*-.199*	*.02*	*ns*
Psychiatric Medication Use	*.122*	*.01*	*ns*
Marital Status	*-.015*	*.00*	*ns*

[a] $F(4,37) = 4.55$, $p = .004$, $R^2 = .33$

[b] $F(4,37) = 1.58$, $p = .200$, $R^2 = .15$

C. Aim 2

The second aim of this study included evaluating the course of psychosocial distress and quality of life related to physical functioning in patients diagnosed with breast cancer. It was expected that levels of psychosocial distress around diagnosis would be increased when compared to levels 3 and 6 months post-diagnosis. Measures targeting general stress, depression, general and medical worry, quality of life, and symptom reporting were evaluated. Additionally, it was expected that trait anxiety at diagnosis would more strongly predict psychosocial distress overtime than would state anxiety at diagnosis. Quality of life due to physical functioning was expected to fluctuate during the first 6 months due to the course of cancer treatments.

Only participants that completed all time points are included in these analyses. When evaluating the course of psychosocial distress and quality of life related to physical functioning, demographic and medical variables that were related to the outcome variable were used at covariates as indicated by bivariate correlations (Tables 5 –11) and each use is noted. Covariates were also included in the regression equations as they related to the predictor and outcome measures. Correlational analyses were conducted to examine the bivariate association between trait and state anxiety and the other distress dimensions over time. As depicted in Table 17, the correlations between trait and state anxiety and the dependent variables ranged from low to high overtime (r = .00 to r = .84). Trait anxiety at diagnosis, with few exceptions, more strongly correlated with other measures of distress at diagnosis and 3 and 6 months post-diagnosis. Within subjects ANOVA and multiple analysis of variance (MANOVA) analyses were done to examine changes in dimensions of psychosocial stress and quality of life due to physical

functioning overtime in this population. Pairwise analyses of means used paired observation t-tests coupled with a Bonferroni control for experiment wise error rates.

Table 17.

Correlations of Trait and State Anxiety at Diagnosis with Distress Overtime *(N = 42)*.

	Diagnosis		*3 months*		*6 months*	
	Trait	State	Trait	State	Trait	State
DASS Depression	.84***	.80***	.53***	.48**	.40**	.31*
DASS Stress	.69***	.61***	.51***	.47**	ns	ns
WDQ Medical	.49**	.45**	ns	ns	ns	ns
WDQ Total	.69***	.48**	.63***	.42**	.45**	.34*
Finances	.35*	ns	.34*	ns	ns	ns
Work	.56***	.56***	.60***	.50**	.38*	ns
Aimless Future	.69***	.55***	.60***	.50**	.47**	.44**
LackConfidence	.73***	.42**	.57***	ns	.53***	ns
Relationship	.60***	.36*	.60***	.36*	.35*	ns
SCL GSI	.86***	.76***	.67***	.54***	.61***	.47**
Somatic	.73***	.67***	.47**	.47**	.40***	.38*
Obsess Compulsive	.78***	.70***	.56***	.43**	.60***	.45**
Interpersonal	.61***	.36*	.66***	.47**	.59***	.41**
Depression	.80***	.79***	.58***	.49**	.58***	.41**
Anxiety	.74***	.74***	.59***	.48**	.51***	.42**
Hostility	.62***	.49**	.48**	.44**	ns	.34*
Phobia	.71***	.61***	.69***	.49**	.53***	.35*
Paranoid	.50**	ns	.67***	.45**	.50**	.30*
Psychotic	.79***	.66***	.64***	.51***	.57***	.37*

MOS General Health	.51**	.45**	.34*	.37*	.41**	.41**
MOS Mental Health	.76***	.79***	.60***	.65***	.47**	.43**
Physical Function	.32*	ns	ns	ns	ns	ns
Physical Role	ns	.34*	ns	ns	ns	ns
Body Pain	ns	ns	ns	ns	ns	ns
Vitality	.62***	.70***	ns	.32*	.41**	.33*
Social Function	ns	.36*	ns	ns	ns	ns
Emotional Role	.48**	.51***	ns	ns	ns	ns
Health Transitions	ns	ns	ns	ns	ns	ns

*p < *.05.* **p < *.01.* ***p < *.001.*

When using psychiatric medication use as a covariate, there were no significant time effects on measures of depression. Without the use of this covariate, however, there was a main effect of time on depression as measured by the DASS $F(2, 40) = 4.2$, $p = .022$. Depression was significantly higher at diagnosis than 6-months post-diagnosis ($M = 2.1$, $SE = .21$ and $M = 1.6$, $SE = .19$, respectively), $F(2,82) = 4.43$, $p = .015$. When examining stress over time, psychiatric medication use was used as a covariate. There was a main time effect for stress $F(2, 39) = 4.48$, $p = .018$. With stress being higher at diagnosis ($M = 2.71$, SE = .20) than at 3 or 6 months post-diagnosis ($M = 2.20$, $SE = .21$ and $M = 2.26$, $SE = .19$, respectively) $F(2,80) = 4.03$, $p = .021$. Figure 3 shows these relationships.

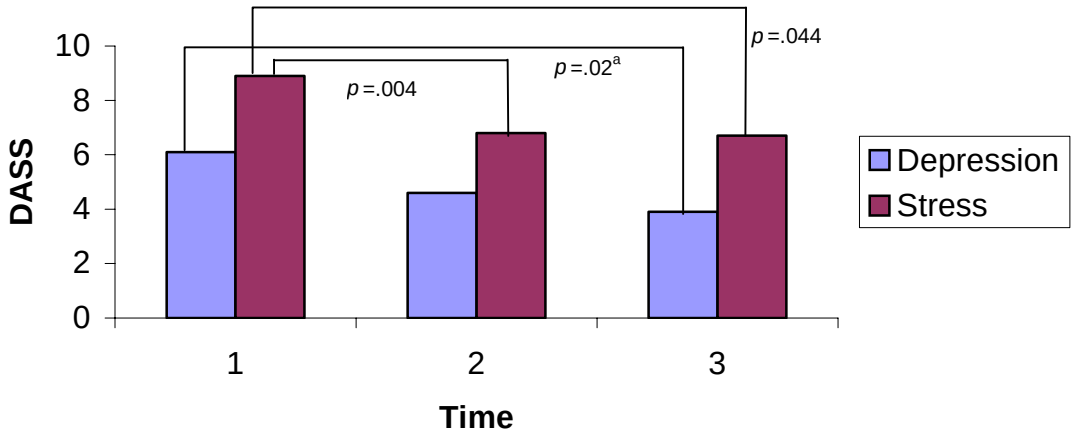

Figure 3. Changes from diagnosis to 3 and 6 months after diagnosis in depression and stress as measured by the DASS. [a]Depression changes are significant only without using psychiatric medication use as a covariate.

There was no main effect of time on total general worry with or without using marital status as a covariate. Additionally, when examining the subscales of the WDQ there were no time effects with (i.e., weight and marriage) or without the use of covariates. When using age as a covariate, there were no main effects of medical worry across time. When not controlling for age, there was a main time effect on medical specific worries $F(2,40) = 9.36$, $p < .001$. Medical worries were higher at diagnosis ($M = 3.04$, SE = .16) than at 3 or 6 months post-diagnosis ($M = 2.60$, $SE = .15$ and $M = 2.46$, $SE = .14$, respectively) $F(2,82) = 11.42$, $p < .001$. Figure 4 displays the across time patterns of worry.

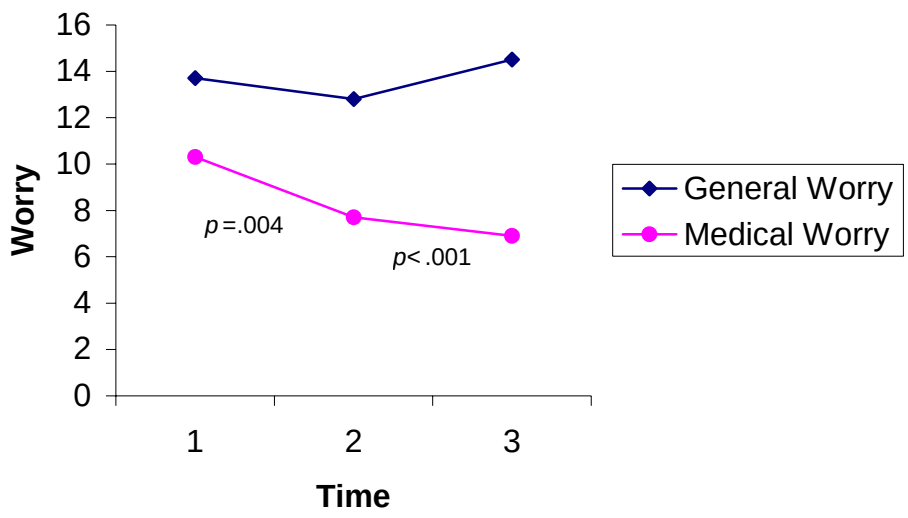

Figure 4. General worry and medical worry from diagnosis to 3 and 6 months post diagnosis. There are no significant changes in general worry across time and only when not using age as a covariate are the changes in medical worry significant.

There was no main time effect with or without the use of covariates (i.e., psychiatric medication use and marital status) on general symptom reporting as measured by the SCL-90. However, when the subscales of this measure were entered into a MANOVA, there was a multivariate effect of time $F(18, 18) = 2.81$, $p = .017$. Marital status and psychiatric medication were used as covariates in this analysis. There was a univariate time effect on somatic report $F(2,70) = 5.16$, $p = .008$. Somatic report was lower at diagnosis ($M = 1.21$, $SE = .03$) than at 3- or 6-months post-diagnosis ($M = 1.26$, $SE = .03$ and $M = 1.26$, $SE = .03$, respectively). The anxiety subscale also demonstrated a univariate time effect $F(2,70) = 7.0$, $p = .004$. Anxiety was higher at diagnosis ($M = 1.24$, SE = .04) than at 3 or 6 months post-diagnosis ($M = 1.13$, $SE = .03$ and $M = 1.11$, $SE = .03$, respectively). SCL-90 univariate effects are displayed in Figure 5.

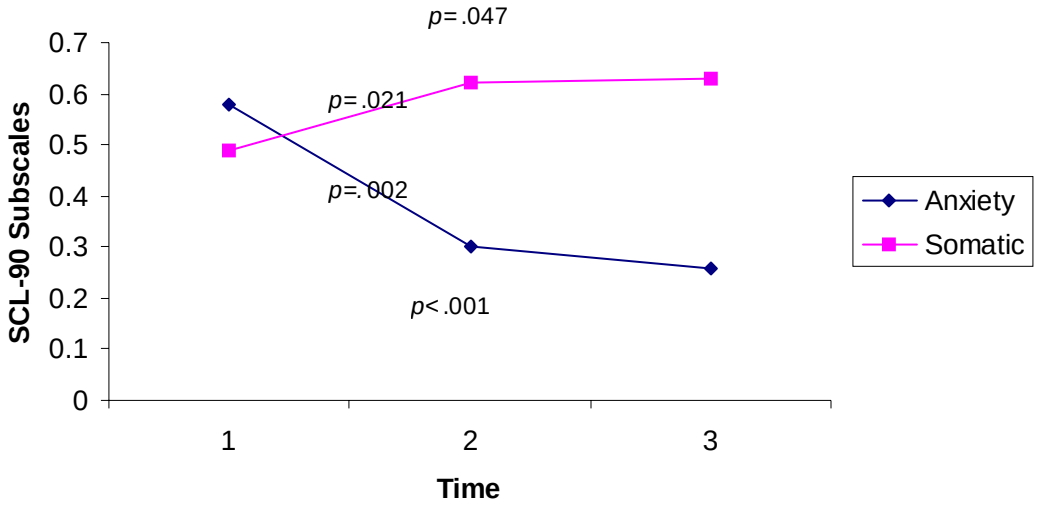

Figure 5. Changes in the SCL-90 subscales from diagnosis to 3 and 6 months post diagnosis. Anxiety and somatic scales are significantly changing from diagnosis to 3 months and 6 months.

Without the use of the covariates there was additionally a univariate time effect on the psychotic scale $F(2,38) = 8.01$, $p < .001$. Psychotic symptom report was higher at diagnosis ($M = 1.15$, SE = .03) than at 3 or 6 months post-diagnosis ($M = 1.10$, $SE = .02$ and $M = 1.09$, $SE = .02$, respectively) $F(2, 78) = 10.27$, $p < .001$. There were no differences over time on the obsessive-compulsive, interpersonal, depression, hostility, phobia, or paranoid scales of the SCL-90-R.

There was a main time effect for general mental health as measured by the MOS $F(2, 38) = 7.49$, $p = .002$. Consistent with the psychosocial measures, mental health distress was significantly higher at diagnosis than 6 months post-diagnosis ($M = 5.06$, $SE = .30$ and $M = 4.08$, $SE = .27$, respectively) $F(2,78) = 7.01$, $p = .001$. There was no main time effect on the general health scale. MANOVA analyses revealed a multivariate time effect for the subscales of the MOS-SF $F(14, 22) = 2.36$, $p < .035$. Family history of breast cancer, age, and frequency of alcohol use were used as covariates in this analysis. Univariate time effects were significant for physical functioning limitations $F(2, 70) = 11.14$, $p < .001$. Physical functioning limitations significantly increased from diagnosis to both 3 and 6 months post-diagnosis ($p < .001$). There was a univariate time effect for physical role limitations $F(2, 70) = 4.13$, $p = .02$. Physical role limitations significantly increased from diagnosis to 3 months post-diagnosis ($p < .001$). There was a univariate time effect for body pain $F(2, 70) = 3.72$, $p = .03$, however, pairwise comparisons were not significant. There was also a univariate effect for emotional role limitations $F(1.73, 60.55) = 4.02$, $p = .022$, again, pairwise comparisons were not significant. Significant effects are represented in Figure 6 and 7.

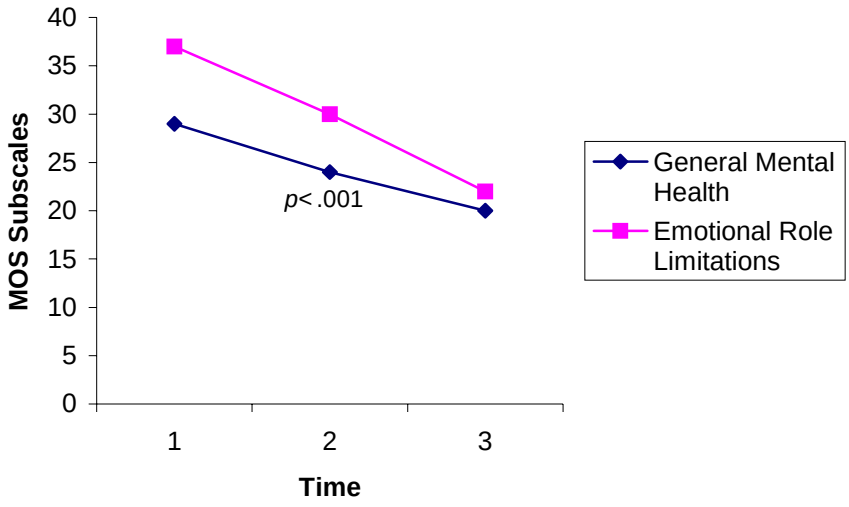

Figure 6. Significant changes across time on scales of the MOS. All change patterns are significant, with general mental health significantly changing from diagnosis to 6 months post diagnosis.

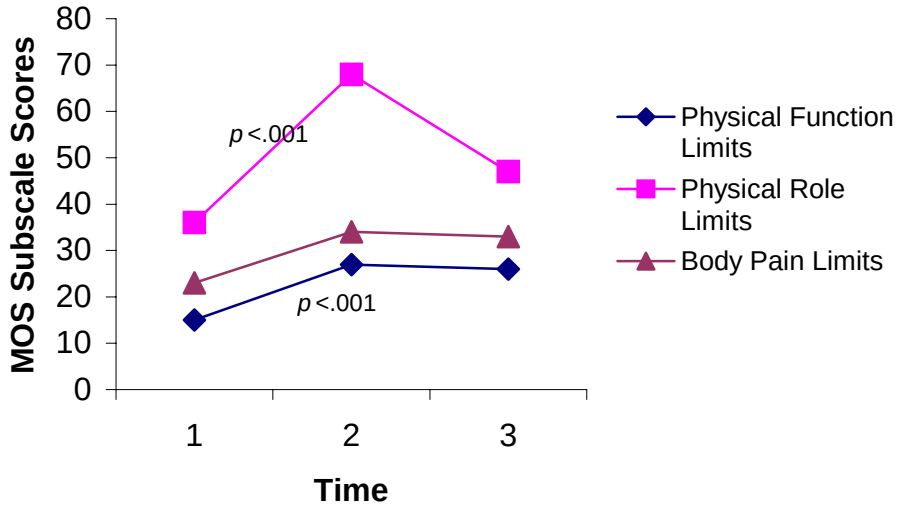

Figure 7. Physical Functioning subscales of the MOS. All main effects are significant. Additionally, there was an effect of from diagnosis to 3 months in physical role limitations and an effect from diagnosis to 6 months for physical functioning limits.

Lastly, regression analyses were used to test the hypothesis that trait anxiety at diagnosis would more strongly predict psychosocial distress over time than state anxiety. As depicted in Tables 18, 19, and 20, a series of regression equations were constructed to examine the predictive relationship between trait and state anxiety on other measures of psychosocial distress at and following a diagnosis of breast cancer. The same strategy as presented in Aim 1 was used to control for shared variance and address multicollinearity of the predictors in the regression equations. When predicting general stress 3 and 6 months post-diagnosis, psychiatric medication use and cancer staging were controlled for in the analyses. The regression equation was significant for predicting general stress 3-months post-diagnosis, accounting for 37% of the variance. None of the individual predictor variables significantly contributed to this model (Table 18). When trait (β = .403, sr^2 = .16, p = .013) and state anxiety (β = .356, sr^2 = .15, p = .014) at diagnosis were entered separately, both were significant predictors of general stress 3 months post-diagnosis. When predicting general stress 6-months post-diagnosis, the regression equation was not significant (Table 18). Neither regression equation was significant when trait and state were entered separately.

When predicting depression 3 and 6 months post-diagnosis, psychiatric medication use was controlled for in all analyses. The regression equation was significant for predicting depression 3-months post-diagnosis, accounting for 32% of the variance. None of the individual predictor variables significantly contributed to this model (Table 18). When trait (β = .509, sr^2 = .22, p = .002) and state anxiety (β = .442, sr^2 = .20, p = .003) at diagnosis were entered separately, both were significant predictors of depression 3 months post-diagnosis. When predicting depression 6-months post-diagnosis, the regression equation approached significance (Table 18). When trait

(β = .471, sr^2 = .18, p = .006) and state anxiety at diagnosis were entered separately, only the equation with trait anxiety was significant.

When predicting general worry 3 and 6 months post-diagnosis, psychiatric medication and marital status were controlled for in all analyses. The regression equation was significant for predicting general worry 3 months post-diagnosis, accounting for 42% of the variance. Only trait anxiety was a unique predictor in this model, the other predictors accounted for no more of the variance than expected by chance (Table 19). When trait (β = .598, sr^2 = .31, p < .001) and state anxiety (β = .362, sr^2 = .15, p = .014) at diagnosis were entered separately, both were significant predictors of general worry 3 months post-diagnosis. When predicting general worry 6-months post-diagnosis, the regression equation was again significant accounting for 32% of the variance (Table 19). In this equation, only marital status was a significant and unique predictor. When trait (β = .401, sr^2 = .11, p = .017) and state anxiety (β = .308, sr^2 = .11, p = .034) at diagnosis were entered separately, both were significant predictors of general worry six months post-diagnosis.

When predicting medical worry 3 and 6 months post-diagnosis, psychiatric medication and age were controlled for in all analyses. The regression equation did not significantly predict medical worry 3 months post-diagnosis. When trait (β = .354, sr^2 = .11, p < .038) and state anxiety at diagnosis were entered separately to predict medical worry at 3 months, only the model including trait anxiety significantly predicted medical worry 3 months post-diagnosis. The regression equation did, however, significantly predict medical worry 6 months post-diagnosis accounting for 33% of the variance. Trait anxiety, psychological medication use, and age were significant predictors of medical worry. The contribution of state anxiety was no more than expected by chance. (Table 19). When trait (β = .350, sr^2 = .12, p = .029) and state anxiety at

diagnosis were entered separately into equations, both equations were significant, with trait anxiety at diagnosis significantly contributing to its equation while state anxiety at diagnosis did not.

When predicting symptom reporting 3 and 6 months post-diagnosis, psychiatric medication and marital status were controlled for in all analyses. The regression equation was significant for predicting symptom reporting 3 months post-diagnosis, accounting for 49% of the variance. Only trait anxiety was a unique predictor in this model, the other predictors accounted for no more of the variance than expected by chance (Table 20). When trait ($\beta = .607$, $sr^2 = .34$, $p < .001$) and state anxiety ($\beta = .480$, $sr^2 = .28$, $p = .001$) at diagnosis were entered separately, both were significant predictors of symptom reporting 3 months post-diagnosis. When predicting symptom reporting 6-months post-diagnosis, the regression equation was again significant accounting for 46% of the variance (Table 20). Trait anxiety and marital status were significant individual predictors of symptom reporting. When trait ($\beta = .600$, $sr^2 = .32$, $p < .001$) and state anxiety ($\beta = .431$, $sr^2 = .22$, $p = .003$) at diagnosis were entered separately, both were significant predictors of symptom reporting 6 months post-diagnosis.

Table 18

Regressions Predicting Dimensions of Distress 3 and 6 months post diagnosis (n = 42)

Variable	*β*	sr^2	p
DASS Stress 3 months[a]			
Trait Anxiety at Diagnosis	*.238*	*.03*	*ns*
State Anxiety at Diagnosis	*.202*	*.02*	*ns*
Psychiatric Medication Use	*.167*	*.03*	*ns*
Cancer Staging	*-.247*	*.08*	*ns*
DASS Stress 6 months[b]			
Trait Anxiety at Diagnosis	*.020*	*.00*	*ns*
State Anxiety at Diagnosis	*.189*	*.02*	*ns*
Psychiatric Medication Use	*.163*	*.02*	*ns*
Cancer Staging	*-.225*	*.05*	*ns*
DASS Depression 3 months[c]			
Trait Anxiety at Diagnosis	*.331*	*.07*	*ns*
State Anxiety at Diagnosis	*.222*	*.03*	*ns*
Psychiatric Medication Use at Diagnosis	*.098*	*.01*	*ns*
DASS Depression 6 months[d]			
Trait Anxiety at Diagnosis	*.470*	*.09*	*.06*
State Anxiety at Diagnosis	*.001*	*.00*	*ns*
Psychiatric Medication Use at Diagnosis	*-.138*	*.02*	*ns*

[a] $F(4,36) = 5.28$, $p = .002$, $R^2 = .37$ [b] $F(4,36) = 1.74$, $p = .163$, $R^2 = .16$
[c] $F(3, 38) = 5.82$, $p = .002$, $R^2 = .32$ [d] $F(3, 38) = 2.81$, $p = .052$, $R^2 = .18$

Table 19.

Regressions Predicting Dimensions of Worry 3 and 6 months post diagnosis (n = 42)

Variable	*β*	sr^2	p
General Worry 3 months[a]			
Trait Anxiety at Diagnosis	*.643*	*.18*	*.006*
State Anxiety at Diagnosis	*-.053*	*.00*	*ns*
Psychiatric Medication Use	*-.031*	*.00*	*ns*
Marital Status	*-.145*	*.03*	*ns*
General Worry 6 months[b]			
Trait Anxiety at Diagnosis	*.303*	*.04*	*ns*
State Anxiety at Diagnosis	*.112*	*.01*	*ns*
Psychiatric Medication Use	*-.073*	*.01*	*ns*
Marital Status	*-.343*	*.12*	*.03*
Medical Worry 3 months[c]			
Trait Anxiety at Diagnosis	*.482*	*.08*	*ns*
State Anxiety at Diagnosis	*-.149*	*.01*	*ns*
Psychiatric Medication Use	*-.215*	*.04*	*ns*
Age	*-.424*	*.15*	*.016*
Medical Worry 6 months[d]			
Trait Anxiety at Diagnosis	*.590*	*.14*	*.02*
State Anxiety at Diagnosis	*-.281*	*.04*	*ns*
Psychiatric Medication Use	*-.376*	*.12*	*.03*

Table 19 (continued).

Age	*-.595*	*.28*	*.00*

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[a]F(4, 37) = 6.82, p < .001, R^2 = .42 [b]F(4, 37) = 4.35, p = .006, R^2 = .32

[c]F(4, 37) = 2.47, p = .062, R^2 = .21 [d]F(4, 37) = 4.51, p = .005, R^2 = .32

Table 20

Regressions Predicting Symptom Reporting 3 and 6 months post diagnosis (n = 42)

Variable	*β*	sr^2	p
Symptom Reporting 3 months[a]			
Trait Anxiety at Diagnosis	*.434*	*.11*	*.04*
State Anxiety at Diagnosis	*.201*	*.03*	*ns*
Psychiatric Medication Use	*.095*	*.01*	*ns*
Marital Status	*-.190*	*.06*	*ns*
Symptom Reporting 6 months[b]			
Trait Anxiety at Diagnosis	*.513*	*.13*	*.02*
State Anxiety at Diagnosis	*.100*	*.01*	*ns*
Psychiatric Medication Use	*-.100*	*.01*	*ns*
Marital Status	*-.262*	*.10*	*.06*

[a] $F(4, 36) = 8.79$, $p < .001$, $R^2 = .49$

[b] $F(4, 36) = 7.61$, $p < .001$, $R^2 = .46$

D. Aim 3

The final aim of this study was to explore the course of the relationship between physiological measures of anxiety and self-report measures of anxiety. First, it was expected that increased levels of physiological stress as measured by salivary cortisol would be related to self-report measures of anxiety at each time point. It was also expected that salivary cortisol levels at diagnosis would account for a significant amount of the variance in self-reported anxiety at diagnosis, 3- and 6-months post-diagnosis. The ability of self-report anxiety and physiological measures of anxiety at diagnosis to predict anxiety at 3 and 6 months post-diagnosis was explored.

Cortisol levels at each time point were calculated using an "area under the curve with respect to increase" formula (Pruessner, Kirshbaum, Meinlschmid, & Hellhammer, 2003). This measurement method disregards the distance from zero for all measurements, and emphasizes the changes over time, with greater decrease during the course of the day suggesting lower overall levels of cortisol. Past research has found this measurement formula to be more reflective of psychological stress than other commonly used formulas (Pruessner et al., 2003).

Participants that completed all questionnaires and cortisol measurements at any time point are included in these analyses. At diagnosis there were 38 participants that met these criteria, at 3 and 6 months post-diagnosis there were 29 and 24 respectively. In addition to cortisol measurements the core anxiety measures for this study (i.e., STAI, DASS-A) were used in these analyses. One-way ANOVAs and chi-squares tests were done to evaluate differences on

background variables or self-report measures among participants that did and did not have available cortisol data. At diagnosis and 6 months, there were no significant differences in these variables. At 3 months, none of the 4 participants that reported currently smoking had available cortisol data (22 total without cortisol data), while none of the 29 participants with cortisol data were smokers (χ^2 = .5.72, p = .017). Due to small cell sizes this statistic should be considered exploratory.

Correlational analyses were conducted to examine the association between cortisol levels and self-report anxiety measures and all background, health behaviors, and medical variables. There were only two significant relationships that emerged. At 3 months the use of psychiatric medication (n = 3) was negatively correlated with cortisol levels (r = .398, p = .032) and at 6 months cortisol levels were related to menopausal status (r = -.538, p = .012). Correlations were rerun excluding the three participants on medication, no differences were found. Among the anxiety self-reports, as expected, there was a relationship between DASS-A at diagnosis and cortisol measurement at diagnosis (r = .422, p = .008). However, no other relationships were significant. Due to the low number of participants who completed cortisol measurements at all three time points (n = 16), dependent T-tests were used to compare differences between each time point thus increasing the number of data points for each test. The results of these tests were not significant; no differences in cortisol measurements were seen between time points.

Based on the results of the bivariate correlations, a regression model was constructed to examine the predictive relationship of cortisol to DASS-A (Table 21). Initial correlations (Table 13) revealed that DASS-A at diagnosis and 3 months was related to both state and trait anxiety at diagnosis. For this reason, these two factors were also included in the regression equations. The results of the regression equation for predicting DASS-A at diagnosis were significant (Table

21). This equation accounted for 58% of the variance in DASS-A and trait anxiety and cortisol significantly contributed to the model. The contribution of state anxiety was no more than expected by chance. These variables were not significant in predicting anxiety at 3 months. However, anxiety variables at diagnosis did significantly predict anxiety 6 months post-diagnosis accounting for 26% of the variance in anxiety 6 months post-diagnosis.

Table 21

Regressions Predicting Anxiety with Cortisol (n = 38)

Variable	β	sr^2	p
DASS Anxiety at Diagnosis[a]			
Trait Anxiety at Diagnosis	.590	.26	.002
State Anxiety at Diagnosis	.064	.00	ns
Cortisol at Diagnosis	.353	.22	.003
DASS Anxiety at 3 months[b]			
Trait Anxiety at Diagnosis	.255	.03	ns
State Anxiety at Diagnosis	.204	.02	ns
Cortisol at Diagnosis	.094	.01	ns
DASS Anxiety at 6 months[c]			
Trait Anxiety at Diagnosis	.639	.26	.03
State Anxiety at Diagnosis	-.265	.00	ns
Cortisol at Diagnosis	.201	.22	ns

[a] $F(3,34) = 15.83$, $p < .001$, $R^2 = .58$

[b] $F(3,32) = 2.71$, $p = .061$, $R^2 = .20$

[c] $F(3,27) = 3.16$, $p = .041$, $R^2 = .26$

IV. SUMMARY AND DISCUSSION

This study focused on examining the impact of trait anxiety on distress overtime in patients with a new diagnosis of breast cancer. Specifically, this study differentiated between trait anxiety and acute anxiety symptom patterns. As expected, trait anxiety was stable in the first six months after the diagnosis of breast cancer, while acute measures of anxiety significantly decreased from diagnosis to 6-months after diagnosis. Consistent with research on stress reactions (Craig et al., 1995), at diagnosis elevated levels of state anxiety may be a normal reaction to the diagnosis of a life threatening disease and could promote the use of resources to cope with the stressful event. A decline in state anxiety across time may be unique to populations of patients with early stage breast cancer; other studies with cancer patients with varying degrees of disease have found state anxiety levels to be more stable across time (Brewin et al., 1998).

This study also investigated the course of other psychosocial distress in patients with a new diagnosis of breast cancer. As expected, measures of depression, stress, medical worry, and symptom reporting decreased from diagnosis to 6 months post-diagnosis. Measures of daily domains of general worry did not show a significant change from diagnosis to 6 months post-diagnosis. This finding may suggest distress as a result of breast cancer diagnosis may be specific to the diagnosis and not globalize into other areas of functioning. Additionally, when controlling for age there was no longer a time effect on measures of medical worry. This finding suggests a unique interaction between age and medical worry. This finding is consistent with past research documenting age effects in this population. Compas et al. (1999) found that younger women experience increased distress compared to older women around the time of diagnosis, but that distress dissipates with time.

When evaluating general symptom report (SCL-90), there was not a main effect across time. The anxiety subscale of this measures did, however, significantly decrease from diagnosis to six months after diagnosis, this is consistent with other acute anxiety measurements used in this study. The somatic subscale of the SCL-90 increased across time; this increase may be reflecting symptom reports that can be accounted for by the nature of cancer treatment. Differences in the direction of change on quality of life dimensions measured by the MOS (psychological distress decrease, physiological distress increase) also reflect this finding and are consistent with the initial unexpected stress of diagnosis and the subsequent course of physically challenging cancer treatment. Specifically, on the MOS, general mental health and emotional role limitations decreased over time, while limits in physical functioning, physical role, and limits due to body pain fluctuated during the course of the study.

As would be expected, the use of psychiatric medication was consistently related to several of the distress outcome variables. The only psychosocial outcome measures that psychiatric medication use was not related to at any of the time points were state anxiety and worries related to medical domains. This unique lack of relationship with state anxiety may further suggest that state anxiety is reflecting a transient and normative reaction to the diagnosis of a life-threatening disease. Marital status was consistently negatively related to several of the psychosocial outcome variables. Past research has documented the benefits of social support in this population (Glanz & Lerman, 1992; Lewis et al., 2001; Trunzo & Pinto, 2003). The presence of a spouse may provide social support and account for decreased distress found here among married women.

Unexpectedly, cancer staging and psychosocial distress were consistently negatively related. One possible hypothesis for this finding involves appraisal processes suggested in the stress response (Lazarus & Folkman, 1984). Although current medical treatment of early stage

breast cancer is often successful for recovery, women continue to fear a diagnosis of breast cancer more than other diseases. When a woman is told that her early stage cancer is likely to be successfully treated, this information may be incongruent with what she believes causing feelings of low mastery over the situation and uncertainty. These constructs are known to produce avoidant coping strategies that lead to higher levels of distress (i.e., Sweet, Savoie, & Lemyre, 1999). However, when a woman is told that she has more severe breast cancer, she may appraise the situation as what she would expect from a diagnosis and be able to use coping strategies that decrease distress. This hypothesis would be best tested with a wider degree of disease status at diagnosis than is found in this study.

A. Anxiety and Psychosocial Distress Across Time

Overall, anxiety at diagnosis was related to poorer psychosocial outcomes during the first six months following a diagnosis of breast cancer. Both state and trait anxiety were correlated with increased psychosocial distress at diagnosis and at 3 and 6 months post-diagnosis. Regression analyses revealed that only trait anxiety at diagnosis was predictive of state anxiety 3 months post-diagnosis, although this relationship did not hold at six months. State anxiety at diagnosis was not uniquely predictive of state anxiety at either follow-up time point. These findings were somewhat modified when trait and state anxiety were entered into regression analyses separately to account for multicollinearity. Both types of anxiety were predictive of 3- and 6-month state anxiety, however, trait anxiety uniformly accounted for more of the variance in each equation.

Regression equations predicting other measures of psychosocial distress revealed that trait anxiety was a unique predictor of general worry at 3 months, medical worry at 6 months, and general symptom reporting at 3 and 6 months. When findings were modified by entering trait and

state anxiety into separate equations, on all measures trait anxiety predicted more variance in each equation model than did state anxiety. Most of these differences were modest, however, when trait anxiety was entered separately it was uniquely predictive of depression at 6 months post-diagnosis, and uniquely predicted medical worry at 3 and 6 months.

These results provide additional information on specific anxiety patterns in women with a new diagnosis of breast cancer. Correlation analyses are consistent with past research (Bleiker et al., 2000; Schreier & Williams, 2004), in that trait and state anxiety at diagnosis are both related to distress at diagnosis and overtime. Although independently both trait and state anxiety at diagnosis were predictive of state anxiety at 3- and 6-months, when entered simultaneously into regression equations trait anxiety was the only unique predictor of state anxiety at anytime point. Likewise, trait and state anxiety were independently correlated with several of the other psychosocial distress measures, but when entered in the equation simultaneously, trait anxiety was consistently unique in accounting for variance in psychosocial outcome measures.

Caution must be used when interpreting these results. Although the measurement and differentiation of trait and state anxiety is theoretically and clinically important, as has been documented in past research (Kendler & Kocovski, 2001), it is difficult to accurately measure and interpret these constructs. Trait and state anxiety were highly correlated with each other and when entered simultaneously into the regression equation they were competing for shared variance. Bearing these factors in mind, these results may suggest that trait anxiety as opposed to state anxiety may put an individual at risk for long-term distress. In this population state anxiety may be more reflective of acute distress and may be a normative reaction to a life threatening disease diagnosis and provide energy to access resources that will promote coping behavior in the individual.

Research studies often fail to differentiate anxiety measures and do not report or account for trait effects on state measurements when using anxiety as a predictor or outcome measure (e.g., Epping-Jordan et al., 1998; Ozalp, Sarioglu, Tuncel, Aslan, & Kadiogullari, 2003). Epping-Jordan and colleagues reported that acute anxiety and depression at the time of diagnosis of breast cancer did not predict these symptoms 3 months after diagnosis, but levels of anxiety at 3 months did predict anxiety at 6 months. This may reflect the current hypothesis and finding that acute anxiety at diagnosis dissipates over time, explaining why there was no significant predictive relationship between baseline and 3 month anxiety levels. However, it may be that the high levels of anxiety at 3 months are more reflective of trait anxiety than state anxiety, thus being the reason 3-month anxiety is predicting 6-month anxiety. When measuring anxiety across time, anxiety that is uniquely state anxiety may decrease over time, but trait anxiety that is being reflected in measures of state anxiety may continue to be reflected in higher scores in state anxiety. Not accounting for this possibility could lead to interpretation of results and conclusions that are inconsistent with actual experience of distress.

At the time of diagnosis, cortisol levels were significantly related only to DASS-A. No relationship was found between other measures of anxiety and cortisol. The DASS-A was designed specifically considering the overlap of depression and anxiety symptoms and excluded items that could reflect depression symptoms as well as symptoms of anxiety (Crawford & Henry, 2003). Therefore, the DASS-A may be a more pure measure of strictly anxiety symptoms, thus accounting for the unique relationship of cortisol with this measure and not with other measures of anxiety. Varying degrees of relationships between physiological stress markers and biological disease markers have been reported (Lueken & Compas, 2002), yet information about the influence of specific psychological processes on physiological stress markers is not

well understood. The unique relationship between anxiety as measured by the DASS-A and cortisol at diagnosis provides additional information on the nature of these relationships.

B. Limitations and Future Directions

This research supports the main hypothesis suggesting that trait anxiety may be a unique construct that can predict long-term distress in patients with disease. While the findings presented here answer some questions, this research also raises several issues. One important issue is the differential outcomes among measures of the construct of anxiety. Appropriately evaluating and applying the measurement of anxiety to the hypothesis being tested and accurately reporting anxiety symptom patterns is an important consideration that should be more adequately addressed in research. This issue is not unique to medical populations, but is particularly important to consider in these populations where stress and anxiety have routinely, but broadly, been associated with disease variables.

Trait anxiety may become more predictive of long-term distress and the effects of state anxiety may continue to dissipate over a more extended period of time. Future research may want to consider these variables over a longer time period. Additionally, considering the current findings, future research should work toward identification of unique aspects and correlates of trait anxiety so that it can be distinguished from state anxiety. This may allow for an early determinant variable of long-term distress in disease populations. Interpretation of these results should consider that the levels of distress in this sample may be lower than distress in this general population. When considering participation in this study, the most anxious women might have declined participation. Additionally, due to the type and early stage of cancer in this sample, these findings may not generalize to more severe disease populations.

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