DIFFERENCES IN APPRAISAL, COPING STYLES, AND ANGER TRAITS BETWEEN FIBROMYALGIA SUBGROUPS

THESIS

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INTRODUCTION

Fibromyalgia syndrome is defined as a "painful, but not articular (not present in the joints), condition predominately involving muscles, and as the most common cause of widespread musculoskeletal pain" (Starlanyl & Copeland, 1996). Fibromyalgia is considered to be a chronic pain syndrome, which means the syndrome is a lifelong pain disorder. Fibromyalgia syndrome has had a controversial history, with major debates about whether the syndrome truly exists and its pathogenesis.

Some of the research focusing on fibromyalgia (FM) has been concentrated on treatment modalities (Turk, Farber, Starz, & Sinclair, 1996). Most of the treatment research suggests that there might be subgroups of fibromyalgia patients that present different clinical pictures and therefore respond to treatment differently. Treatment for fibromyalgia patients has not developed into a set protocol since patients differ in their symptoms and severity of symptoms (Maurizio & Rogers, 1997).

Fibromyalgia patients do seem to have similar characteristics depending on the nature of the syndrome's onset. The types of onset include traumatic and idiopathic. Turk and colleagues (1996) have found differences within the onset subgroups on such factors employment status, MMPI scores, presence and degree of disability compensation, and levels of physical activity.

Research on appraisal, coping styles, and anger traits has been done with other non-FMS chronic pain patients. Turk and Okifuji (1999) hypothesize that assessing and treating a person's pain may require knowledge of the patient's current mood and style of

coping with the symptoms. Anger has been found widely in chronic pain patients (Air, Neumann, Bor, Shir, Rubinow, & Buskila, 2000). Air and colleagues (2000) found that chronic pain patients tended to report a greater use of an avoidant coping style, more state anger, and more anger turned inward. An avoidant coping style means that chronic patients tend to distract themselves from the pain or problems dealing with their illness. State anger assesses the chronic pain patient's current, situational-based intensity of angry feelings, whereas trait anger assesses the more permanent, stable disposition of the patient to experience anger. Anger turned inward means that a person typically coverts anger, or does not express feelings of angry when he or she is upset. However, no research has been found that examines these traits in of FM based on idiopathic vs. traumatic onsets.

STATEMENT OF PURPOSE

The purpose of the thesis is to investigate if subgroups of fibromyalgia patients report different appraisal techniques, coping styles, and anger traits. This thesis will address several research questions:

RQ: Do fibromyalgia patients with different types of onset differ on appraisal techniques?

Research on appraisal techniques for fibromyalgia patients was not found. This study will investigate if subgroups of fibromyalgia patients differ in appraisal techniques. Appraisal describes the way a person assesses a certain psychologically distressing situation. Appraisal techniques will be measured by using the Appraisal Dimensions Scale by Folkman and Lazarus that has six appraisal subscales: control, salience, novelty, duration, causality, and predictability. A total appraisal score will be used to find group

differences. This investigator hypothesizes that patients with ETO and IO of fibromyalgia will have higher (suggesting maladaptive) scores than patients with PTO. "Maladaptive" appraisal techniques means the subject will report higher scores of feeling out of control, causing fibromyalgia, and not being able to predict what course their illness will take. These differences in appraisal will be due to the fact that patients with IO and ETO do not have tangible justification for why they have developed widespread pain, whereas physical trauma patients have a clearly understood, physical event to link their development of fibromyalgia. Therefore, IO and ETO patients will have a higher score on the appraisal score than PTO patients.

RQ: Do subgroups of fibromyalgia patients with different type of onset differ in coping styles?

Regarding coping styles among fibromyalgia patients, research is scarce (Air, et al, 2000). This present study will investigate if subgroups of fibromyalgia patients will differ in coping styles. Coping styles will be measured using the Ways of Coping Checklist Revised. The scales on this instrument are: problem-focused, seeks social support, blamed self, wishful thinking, avoidance, blamed others, count your blessings, and religiosity. Two scores will be used from this instrument: the problem-focused coping scale and an emotion-focused scale will be formed (based on collapsed scores on self-blame, wishful thinking, avoidance, and counting one's blessings. This investigator hypothesizes than patients with IO and ETO will score higher on emotion-focused coping responses than patients with PTO. Patients with PTO will score higher on the problem-focused coping scale than patients with ETO or IO.

Problem-focused and emotion-focused coping styles serve as mediators between appraisal and psychological distress (Mishel & Sorenson, 1993). Problem-focused coping involves active cognition or behavioral changes, such as changing one's behavior or environmental conditions, in effort to deal with the stressor. Patients without an external environmental aspect of the stressor will cope with the stressor internally.

RQ: Do subgroups of fibromyalgia patients differ on expressions of anger? Anger in pain patients has been recognized for decades (Air, et al, 2000). However, review of the literature did not yield any research was found on anger styles and subgroups of FM patients. This study will also investigate if FM onset subgroups differ on expressions of anger. The State-Trait Anger Expression Inventory will be used to measure the experience and expression of anger. The hypothesis is that patients with IO and ETO will score higher on Anger-in (anger turned inward) scores than patients with PTO. Patients with no tangible cause of their pain will turn anger inward, which may even contribute to increasing the intensity of the perceived pain. Another hypothesis is that patients with ETO and IO will score higher on anger control scores than patients with PTO since ETO and IO patients will tend to control their expressions of anger by turning their anger inward. State anger and Trait anger will also be explored within the FM onset subgroups.

JUSTIFICATION

The thesis topic is important for several reasons. First, this thesis will be one of the first systematic investigations of appraisal techniques, coping styles, and anger traits between FM onset subgroups. While such research has been published on other chronic pain syndromes, none has been found on FM specifically. Research of appraisal, coping

styles, and anger traits is important to discover potential differences between subgroups for treatment purposes.

If FM subgroups are found to have similar appraisal techniques, coping styles, and anger traits, then tailored psychological interventions will be easier to implement and be more effective. Maladaptive coping strategies that a patient uses can be better identified and healthy coping strategies can be taught in a targeted manner.

In the long run, research on FM onset subgroups can help make treatment overall more cost-effective and beneficial. If onset subgroups' techniques differ, more research obviously is needed to improve tailored treatment. Treatment for FM has been considered "trial-and-error" and has been done on a one-on-one basis (Maurizio & Rogers, 1997). By identifying subgroups, health care professionals can have a better idea where to begin treatment. A protocol for psychological interventions could be implemented for each type of onset subgroup.

CHAPTER ONE

REVIEW OF LITERATURE

DEFINITION

In 1987, the American Medical Association recognized fibromyalgia as a "true" illness and a major cause of disability. The official definition of fibromyalgia came from the World Health Organization as the Copenhagen Declaration. The Copenhagen Declaration defines fibromyalgia as a "painful, but not articular (not present in the joints), condition predominately involving muscles, and as the most common cause of widespread musculoskeletal pain" (Starlanyl & Copeland, 1996). Other definitions include fibromyalgia syndrome characterized by "widespread pain, stiffness, fatigue, and low pain thresholds at specific anatomic sites, termed tender points" (Aaron, Bradley, Triana-Alexander, Alexander, Martin, & Alberts, 1997). Fibromyalgia is considered a chronic pain syndrome, which means the syndrome is a lifelong pain disorder. Fibromyalgia (FM) syndrome is also described as a functional, non-degenerative disorder since there is no structural damage to the body. This syndrome is not considered a disease since a disease must meet the criteria of being a medical condition with a well-understood pathology (van Why, 1997). FM is labeled a "syndrome" since pathogenesis of FM, whether having a biological or psychological origin or a combination, is still under debate.

Fibromyalgia has been estimated to afflict 2-8% of the world population with about 90% of patients being female (van Why, 1997). In America, it has been estimated that 3-6 million people suffer from fibromyalgia (Turk, Okifuji, Starz, & Sinclair, 1996a). Fibromyalgia is one of the most common disorders seen in outpatient rheumatology clinics (Turk et al, 1996a). However, Kossoff (1999) reported that patients typically suffer from fibromyalgia symptoms for about eight years before receiving a diagnosis of fibromyalgia. This may be why fibromyalgia syndrome is usually diagnosed in the adult population, although FM has been reported to manifest in childhood and adolescence. (van Why, 1997). Most patients report that their FMS symptoms began gradually after the traumatic event (Aaron et al, 1997). There is no known cure for FM, and the syndrome usually does not progress. While patients do not become crippled or die from this syndrome, the pain, fatigue, and exhaustion that patients may experience can significantly lower their quality of life.

SYMPTOMS

The four symptoms that best characterize FM are: 1) chronic pain, 2) chronic fatigue, 3) morning stiffness or joint stiffness following inactivity, and 4) non-restorative sleep. The type and severity of symptoms may differ for each FM patient. FM symptoms for each individual may change continually each day or throughout the day (Secord, 1998). Chronic pain afflicts every quadrant of the body, and most FMS patients describe the pain as "aching," "gnawing" or "burning" (van Why, 1997). Pain has been reported in the occiput (back of the skull), neck, shoulder, low back, buttocks, elbows, knees, and hips (Maurizio & Rogers, 1997; Millea & Holloway, 2000). The level of reported pain is

different for each FM patient, ranging from being an irritant or so severe the patient cannot perform simple, everyday tasks (Secord, 1998).

Chronic fatigue has been reported in 85% of FM patients (Hulme, 1998). Chronic fatigue refers to exhaustion following levels of physical exertion, which patients formerly reported being able to tolerate. Patients with chronic fatigue also report feeling tired upon awakening from sleep. Several factors contribute to chronic fatigue such as poor quality of sleep, insufficient sleep, and muscle tension or skeletal spasm. Some studies suggest that muscle tension and skeletal spasm may deplete the metabolic resources needed for energy and sustained activity, so FM patients consequently always feel tired (van Why, 1997).

Morning or joint stiffness has been reported in 80% of FM patients (Hulme, 1998). Joint stiffness can manifest itself after any rest period, not only after a period of sleep. Interestingly, fibromyalgia is the opposite of Degenerative Joint Disease where joint stiffness increases with activity and decreases with rest (van Why, 1997). With FM, joint stiffness increases with rest and decreases with activity.

Sleep disturbances have also been reported in 80% of FM patients (Hulme, 1998). It may take one to two hours for FM patients to fall asleep at night, and patients report waking up anywhere from two to twenty-nine times during the night. Any noise, smell or any other sensory stimuli may arouse them (Hulme, 1998).

FM patients that have non-restorative or non-refreshing sleep report that after awakening, they feel like they have not slept at all. This is due to alpha incursion into delta (Stage 4) sleep and during the deepest stages of non-dreaming, non-REM sleep (van Why, 1997; Hulme, 1998). There are four stages of sleep that are characterized by

NREM (non-rapid eye movement). Stages 1 and 2 are the beginning of sleep, and the person can easily be awakened. After thirty to forty-five minutes of falling asleep, a person enters Stage 3 of sleep where high and low frequencies of delta waves begin to appear. During these deepest levels of sleep, heart rate, respiration, temperature, and blood flow to the brain are reduced, and growth hormones are secreted. According to the repair theory of sleep, sleep is a biological need that allows for replenishment to areas in the brain and body that are depleted during the day (*Gale Encyclopedia of Psychology, 2001).* Because of these sleep difficulties, FMS patients' muscles maintain a high level of activity at rest, and growth hormones are not released to repair cells for growth and metabolism (Hulme, 1998).

Neurovascular complaints or symptoms include muscular weakness, swelling, hypersensitivity to the cold or heat, numbness or tingling in legs and arms, mottled skin, and a reticular or "net-like" skin pattern (Baumstrak & Buckelew, 1992). Paraestheia, or the feeling of numbness or tingling in legs and arms has been reported in 50% of patients and hypersensitivity to noise, odors, heat or cold occurs in 50-60% of patients (van Why, 1997). Swelling has been said to be subjective or not always be noticeable to others.

Non-rheumatic symptoms are also commonly reported. These symptoms include headaches, depression, global anxiety, and dysmenorrhea (Baumstrak & Buckelew, 1992). Migraine or tension-type headaches occur in 70% of patients. Migraine headaches are one-sided, throbbing head pain usually associated with nausea and hypersensitivity to light and sound. Tension headaches produce a sensation of tightness or pressure across the forehead on both sides of the head and at the back of the neck extending into the

shoulder (van Why, 1997). Depression has been reported in 20-50% of patients, and global anxiety has been reported in 62% of FMS patients (van Why, 1997).

Research suggests that several factors can increase the severity of the non-rheumatic and neurovascular symptoms, although the mechanisms of influence are unclear. These factors are altered sleep, cold and heat exposure, anxiety, humidity, stress, fatigue, and sudden barometric pressure change (van Why, 1997).

COMORBIDITY

Fibromyalgia can occur concomitantly with other disorders, diseases, or illnesses. Irritable bowel syndrome has been reported by 70% of patients, pelvic pain dysmenorrhea by 43% of patients, Raynaud's phenomena (cold hands and feet) by 38%, and temporomandibular joint syndrome by 25%, and irritable bladder syndrome by 12% of patients (van Why, 1997). Other illnesses reported include rheumatoid arthritis, systemic lupus erythematosus, anklyosing spondylitis, Sjogren's syndrome, osteoarthritis, degenerative joint disease, hypothyroidism, and Cushing's disease (Hulme, 1998).

DIAGNOSIS

The American College of Rheumatology defined the official diagnosis of fibromyalgia syndrome in 1990 (Starlanyl & Copeland, 1996). Before the decision to diagnose a patient with FMS, all other illnesses and diseases must be ruled out via blood tests (Starlanyl & Copeland, 1996). Other potential diagnoses that must be excluded include autoimmune disorders, digestive disorders, musculoskeletal problems, and some cancers since the symptoms of these disorders can mimic the symptoms of fibromyalgia (Ser Vaas, 2000). To be diagnosed with FMS, the patient must meet the two criteria identified by the ACR. The two criteria needed to make a diagnosis of fibromyalgia are

as follows: (1) widespread pain for at least three months duration, and (2) reports of pain upon palpation of 11 of 18 specific tender points (Turk et al, 1996a). The "history of widespread pain" is defined as pain on the right and left sides of the body and axial skeletal pain (Baumstrak & Buckelew, 1992). Pain must also be above and below the waist (Kossoff, 1999).

Palpation of tender points causes local and referred pain. Local pain occurs when pressing on these pressure points causes pain at that trigger point while referred pain, causes pain both to the site (termed latent trigger point) and to other body parts (Starlanyl & Copeland, 1996). Location of these nine tender points occurring bilaterally include occiput (back of skull), low cervical (back of neck), trapezius (shoulders), supraspinatus (shoulder blade), second rib, lateral epicondyle (outside of knee), gluteal (buttocks), greater trochanter (hip), and knee. Pressure dolorimetry or direct digital palpation can measure tenderness of these specific anatomical sites. In dolorimetry, the tenderness is measured in kilograms per square centimeter using a mechanical device called a dolorimeter. In digital palpation, the tenderness is measured by pressing on each specific site (tender points) with fingers or thumb. The patient then rates the presence of pain on a scale rating of 0 to 4 (Jensen, Wittrup, Rogind, Danneskloid-Samsoe, & Bliddal, 2000). During physical examination of the specified tender points, other physical findings suggestive of a FM diagnosis include: changes in skin texture, increased resting muscle tension, and changes in the texture of the subcutaneous tissue (Millea & Holloway, 2000).

Minor criteria are also considered when diagnosing for fibromyalgia. Minor criteria include reports of disturbed sleep, fatigue, general anxiety, headaches, irritable

bowel syndrome, subjective swelling, numbness, and symptoms exacerbated by stress and/or anxiety (Baumstrak & Buckelew, 1992).

HISTORY

Although FMS seems to be a relatively new syndrome in popular media, this syndrome appears to have had a long evolution in history. Changes in the name and understanding of FM are partially due to the diagnostic criteria used to define the syndrome, speculative causes for the syndrome, and what type of health professional is diagnosing the syndrome (Maurizio & Rogers, 1997). In the past, FMS has been termed chronic or muscular rheumatism, soft tissue or non-articular rheumatism, fibrositis, neurasthenia, and myalgia (Starlanyl & Copeland, 1996; van Why, 1997).

During the 17th century, Galenic physicians were interested in looking at bodily humors to describe illnesses. The term "rheuma" was used to describe a pathophysiological process of the downward flow through the body of a morbid humor. Where this morbid humor stopped in the body caused a disease in that part of the body. This type of "chronic rheumatism" included painful, non-articular conditions of the musculoskeletal system (Quinter & Cohen, 1999).

In 1814, William Balfour, a Scottish surgeon and massage therapist, popularized the term "muscular rheumatism," a term which lasted for the rest of the 19th century. Muscular rheumatism eventually was rejected as a diagnosis, as it was seen as being too inclusive. Muscular rheumatism included regional myofascial pain syndromes as well as localized muscle pain problems like tendinitis and bursitis (van Why, 1997).

An American neurologist named Beard formulated a different concept of chronic rheumatism in 1869 and termed it "neurasthenia." Neurasthenia meant nervous

exhaustion, and Beard believed neurasthenia led to organic diseases if left untreated. Neurasthenia replaced muscular rheumatism as a somatic diagnosis for unexplained symptoms of physical and mental debility, headaches, sleep disturbances, muscular weakness, and widespread pain (Quinter & Cohen, 1999). Neurasthenia was also diagnosed for any widespread pain syndromes that were not considered to be due to a person malingering in order to avoid either accountability for his or her actions or for secondary gains like receiving attention (Berklow, 1997). Neurasthenia lost its popularity during the 1920s when theories of chronic pain focused more on conscious and unconscious psychological conflicts regarding sexual disturbances. Other researchers also refuted Beard, speculating that any organic disease could be complicated by but not caused by neurasthenia (Quinter & Cohen, 1999).

In 1904, Sir William Gowers provided FM with a new name, "fibrositis" to describe a musculoskeletal problem of the lower back. Fibrositis means inflammation of the white, fibrous connective tissues. The Scottish pathologist claimed to have viewed microscopic signs of inflammation of all the fibrous tissues (muscles, ligaments, and tendons) when he took tissue from his patients (Kossoff, 1999). The Septic Focus theory described the cause of the inflammation. The Septic Focus theory stated that Staphylococcus bacteria would infect organs such as teeth, parotid glands, gall bladder, or the appendix. The colonies of bacteria in these organs would spread into the bloodstream and migrate to sites in muscle and fibrous connective tissue, which would die due to the infection. While the bacteria were present in the living muscle and tissue, the bacteria would release metabolic toxins causing irritation and localized swelling. Gowers termed these sites as "fibrositic lesions" (van Why, 1997).

In the 1930s, Richard Gustein expanded on Beard's idea of neurasthenia but termed it "myodysneuria." Myodysneuria described a painful condition of the nerves serving muscles. Gustein believed the muscular and the nervous system caused the problem, and saw the syndrome as a dysfunction in the autonomic nervous system (van Why, 1997). However, fibrositis remained the more popular diagnosis until 1940 (Quinter & Cohen, 1999).

In the 1950s, Wallace Graham, a Canadian rheumatologist, introduced a new theory to explain fibrositis. He believed that fibrositis was not a disease itself, but a common end point for many disease processes. He called fibrositis a "final common pathway," but his views were largely ignored (van Why, 1997).

The first time the cause for fibromyalgia was described as being partially psychological was in the 1960s. Fibrositis became synonymous with "psychogenic rheumatism" which described a muscular manifestation of mental illness (van Why, 1997).

It was not until 1977 that interest in understanding fibromyalgia syndrome exploded, prompted by a seminar paper in the Bulletin of Rheumatic Diseases. Rheumatologist Hugh Smythe and psychologist Harvey Moldofsky, a student of Wallace Graham, published a paper on pain amplification syndrome, a disorder due to a grievous problem with the way the body's own pain control system inhibited pain (Kossoff, 1999; van Why, 1997). The two proposed diagnostic criteria for fibromyalgia, but the medical community was skeptical of their views. The skepticism stemmed from the fact that like all chronic pain syndromes, there was no known physiological explanation for the pain (Kossoff, 1999).

The first controlled study on FMS was published in 1981 which reported the diagnosis of fibromyalgia as a syndrome characterized by "widespread pain and increased sensitivity to pressure at various specific anatomical locations known as tender points" (Quinter & Cohen, 1999; Maurizio & Rogers, 1997). The study also identified many more symptoms besides the widespread pain at tender points (Maurizio & Rogers 1997).

THEORIES

Psychological Theories

Since the organic etiology of fibromyalgia is unknown, many doctors, scientists, and researchers believe this syndrome has more of a psychogenic origin. These researchers believe that FM is not an illness or syndrome, but a process due to a psychiatric disorder. Even after the 1981 controlled study on fibromyalgia syndrome was published, many researchers in the field continued to refute the idea that FM was a syndrome. FMS could still not be demonstrated objectively or demonstrate any pathological etiology (Maurizio & Rogers, 1997). Furthermore, physical examinations and laboratory studies such as complete blood counts, erythrocyte sedimentation rates, and EMG studies of FM patients yielded typically normal results, which further led researchers to believe that the etiology of fibromyalgia was psychological in nature (Baumstrak & Buckelew, 1992).

"Tender lady" syndrome

Most researchers also speculated that the syndrome had a psychological origin since most patients were female. Doctors have frequently dismissed FMS diagnosis and termed the condition as a "tender lady syndrome" (Kossoff, 1999). This idea derives from

the historic perspective that more women have psychiatric disorders because they are mentally "weaker." There may be many factors that contribute to the disproportional high prevalence rate of FMS among females compared to males. First, women may seek medical help more frequently for symptoms such as fatigue and idiopathic pain, or pain that does not have an identifiable cause. Since such symptoms have been attributed commonly as "all in the head" or "womanly" problems, men may choose not to seek medical help for these types of symptoms due to possible stigmatization. Furthermore, biologic evidence has shown that women tend to have lower serotonin levels than men. Serotonin is the neurotransmitter than regulates substance P, the neurotransmitter that aids in the transmission of pain signals from the spinal cord to the brain. If a person has high levels of substance P and low levels of regulating serotonin, pain signals may be transmitted more frequently or the body could be come more "attuned" to stimuli that cause pain. Therefore, women may subsequently have lower thresholds of pain or may be more biologically equipped to perceive smaller amounts of pain.

Psychopathology

Popular theories describing the etiology of FM as having a psychological basis were spurred by after the publication of a 1982 study by Maurizio and Rogers. FMS patients were compared to rheumatoid arthritis patients using the Minnesota Multiphasic Personality Inventory (MMPI). The MMPI is a standardized questionnaire that assesses psychiatric symptoms and personality characteristics. The authors reported significantly elevated scores on the MMPI in the FMS patients. The researchers concluded that FMS patients are "psychologically disturbed" and that FMS could possibly be linked to mental disorders (Maurizio & Rogers, 1997).

MMPI gives information on specific symptomology, not into a specific psychological diagnosis. The MMPI did not indicate that the patients had a psychiatric disorder, only elevated scored. As with all types of pain patients, MMPI scores are often elevated in anxiety and depression levels, due to situational distress, and are not necessarily indicative of a clinical disorder. Other research has shown that the MMPI is not a good battery to use for patients with somatic symptoms that are due to the non-psychiatric illness since these symptoms will elevate depressive and hypochondrical scales (Duhteman, 1966). Duhteman (1966) found that medical students who took the MMPI had elevated depressive and hypochondrical scores which was probably due to the distressful situation of being in medical school.

More recent studies have directly refuted the findings of the 1982 study. Another study conducted by Maurizio and Rogers (1997), the same authors of the 1982 study, used the Psychiatric Diagnostic Interview to assess mental status of the patients. The authors reported that the instrument did not find any major differences between FMS patients and rheumatoid arthritis patients.

Psychopathologic factors still cannot be proven to be the primary or sole cause of fibromyalgia. Much research has shown that the psychological aspects of FMS patients are similar to those with other chronic pain syndromes that are not believed to have a sole psychogenic origin (Maurizio & Rogers, 1997).

The following sections discuss research that has investigated FM links to specific types of psychopathology.

Mood Disorders

A study by Hudson and colleagues (1985) looked at the relationship between fibromyalgia and symptoms of major affective disorder. FMS patients exhibited symptoms of major affective disorder such as sleep disturbances, anergia, and anxiety. Also, patients with FMS exhibit phenomenologic similarities (or symptoms) to patients with chronic pain syndrome, which had been linked to several indices of a major affective disorder. Hudson and colleagues (1985) found a high rate of major affective disorder and a high family prevalence of major affective disorder in fibromyalgia patients. Therefore, a relationship between fibromyalgia and major affective disorder was suggested. Hudson and colleagues (1985) suggested that fibromyalgia might be a form of major affective disorder in which somatic symptoms are predominant. A family history of major affective disorder may constitute a preexisting vulnerability to the pain-prone patient, as well as exacerbate the condition.

More current studies have found contradictory results. When the frequency of psychiatric diagnosis was compared in FMS patients, rheumatoid arthritis patients, and subjects without pain, Ahles and colleagues (1991) found that there were no group differences in terms of lifetime history of any psychiatric disorders, major affective disorder, somatization disorder, or anxiety-based disorders. More importantly, this study showed that the Psychiatric Diagnostic Interview did not find differences in psychological symptomology between a disorder with a known organic cause (rheumatoid arthritis) and a disorder with no known organic etiology (fibromyalgia) (Ahles et al, 1991).

While fibromyalgia is no longer generally considered to be a derivative of major affective disorder, this theory positively contributed to the treatment of fibromyalgia by the introduction of antidepressant medications (Hudson et al, 1985).

Depression

One etiological theory of fibromyalgia suggests that fibromyalgia is a form of masked depression which is a Mood Disorder. Symptoms of masked depression include disturbances in appetite or sleep, either increasing or decreasing (Keller, 2001). However, Ahles and colleagues (1991) found that FMS patients did not report more vegetative signs of depression than rheumatoid arthritis patients or subjects without pain. Vegetative signs, a cardinal description part of masked depression, are a group of symptoms that refer to sleep, appetite, and weight regulation (Keller, 2001).

FMS patients differ from clinically depressed patients in terms of their cortisol levels, which has been related to depression. Cortisol is the most potent glucocorticoid produced by the adrenal gland and is part of the hypothalamic-pituitary-adrenal axis (HPA). HPA is a major determinant of the body's response to stress (Mercola, 2000). Cortisol affects immune functioning, mental processes, glucose regulation, vascular tone, and bone metabolism. FMS patients have been found to have an alteration in the hypopituitary-adrenal axis and a low production of cortisol, whereas people with depression alone have higher levels than normal of cortisol (Millea & Holloway, 2000).

Although most current research does not explicitly attribute fibromyalgia as being a psychological disorder, most research still recognizes the important role of psychological factors such as the role of depression in chronic pain syndromes. (Brown, 1990). Depression has been shown to be comorbid with chronic pain with an estimated

prevalence between 10% and 100% in chronic pain patients. Much research has been done in depression and its relation to chronic pain. The literature on chronic pain and depression has shown mixed results, with some studies indicating a strong relationship where others find none (Brown, 1990). Three hypotheses on the nature of chronic pain-depression relationship have been established in the research. These hypotheses are: (1) depression in exacerbates or contributes to the patient's pain by lowering his or her pain tolerance thresholds, (2) secondary depression occurs as an effect of chronic pain, and (3) the relationship between chronic pain and depression is that these symptoms occur simultaneously (Brown, 1990).

The first hypothesis that depression increases the patient's pain by lowering his or her pain tolerance threshold has been supported by some research. Brown (1990) found that the patients who tend to report higher levels of pain also have more depressive symptoms. No causal relationship can be established, however, so it is uncertain whether depression causes pain or pain causes depression.

The hypothesis that secondary depression occurs due to chronic pain seems plausible since chronic pain such as fibromyalgia is debilitating, lowers quality of life, and tends to reduce physical and social activities. Having a reduction in quality of life, physical activities, and social activities has been shown to cause depression (Brown, 1990). In addition, a patient realizing that he or she may have to view life though "pain-colored" glasses may be depressing in and of itself.

The third hypothesis that chronic pain and depression occur simultaneously is supported by these two constructs having similar psychological and biological origins. For example, as stated earlier, serotonin deficiency is related to depression. Serotonin

deficiency may be contributed to low thresholds of pain. Serotonin regulates substance P, the neurotransmitter that transmits signals of pain from the spinal cord to the brain. Although most patients with chronic pain do have higher levels of depression, there is no direct evidence that depression causes chronic pain.

Psychosomatic

Research findings about the pathogenesis and pathophysiology of FMS have confirmed the view that fibromyalgia is not a psychosomatic disorder. Although no laboratory tests can confirm fibromyalgia, endocrinologic and neurologic findings have suggested that disturbances in the autonomic and endocrine stress response system may contribute to the etiology of fibromyalgia. One endocrinologic and neurologic finding includes three times more than normal levels of cerebrospinal fluid substance P in FMS patients (Millea & Holloway, 2000). Fibromyalgia patients are also not more likely to report depressive symptoms or receive a lifetime psychiatric diagnosis of major depression (Kiramayer, Robbins, & Kapusta, 1988). In fact Kiramayer and colleagues (1988) found that only 1 in 5 FM patients ever had an episode of major depression, with most cases long before the onset of FMS compared to the general population rate, which is 15.3% (Keller, 2001). FMS patients also do not meet the DSM requirement for a somatization disorder. Kiramayer and colleagues (1988) suggest viewing somatization as more of a process of illness behavior than the cause of FM.

Research indicates that FM does not seem to be related to hysterical or factitious illnesses. Patients with hysterical or factitious illnesses will exhibit symptoms such as having a paralyzed arm. However, during hypnosis, these patients will gain feeling back in their arm, where FM patients under hypnosis will not "lose" FM symptoms.

Furthermore, symptoms of fibromyalgia are specific and stable whereas symptoms of factitious illnesses are changeable and sometimes bizarre (Hudson et al, 1985).

Generalized Hypervigilance

Generalized hypervigilance is similar to altered nociception and sensory amplification, which will be discussed in biological theories, but concentrates more on cognitive factors. The Hypervigilance Model of Pain Perception states that chronic pain patients have a " heightened sensitivity to pain (low threshold and tolerance) because of increased attention to external stimulation and a preoccupation with pain sensations" (McDermid, Rollman, & McCain, 1996). Hypervigilance could possibly be mediated by cognitive factors that reflect the influence of past experiences on present mental processes. Chronic pain patients may thus pay more attention to particular events (situations that increase pain levels) or form a cognitive schema that leads those patients to search for somatic cues (pain or other FM symptoms), which may reinforce their schema. This means FM patients may be more likely to monitor bodily sensations and become preoccupied about perceptual experiences (McDermid et al, 1996). Lautenbacher and Rollman (1997) believe that persons who are inclined to amplify painful experiences and who are hypervigilant to painful events are at risk for developing FM.

Studies have shown that hypervigilance of perceptual thresholds in FM patients does not only involve the perception of pain (Dohrenbusch, Sodhi, Lamprecht, & Genth, 1997). Non-symptomatic hearing loss was found in FM patients along with higher perceptual thresholds for everyday acoustic experience. FM patients also evaluate noise intensity and everyday noise more negatively than healthy normals (Dohrenbusch et al, 1997).

Biological Theories

Physical and Metabolic Findings

Many physical and metabolic findings in the study of FM patients have supported the theory that fibromyalgia is due to a biological pathogenesis. Muscle biopsies have shown that FM patients have metabolic and fiber abnormalities. The fibers tend to have a "moth-eaten" appearance as well as abnormal mitochondrial appearance (Baumstrak & Buckelew, 1992). Baumstrak and Buckelew (1992) also found that FM patients' quadriceps muscles have an abnormal rubber band-like fiber.

A chemical finding that supports the biological theory regarding FM's pathology is the finding of altered substance P levels in FM patients. Substance P is a neurotransmitter found only in the spinal fluid and transmits the signal of nociception from the spinal cord to the brain. Substance P is stored and released within the afferent nociceptive fibers (De Stefano, Villanova, Frati, Mangenilli, Francheschini, Biasi, Marcolongo, 2000). High levels of substance P will send pain messages to the brain even without an injury to the peripheral part of the body and cause chronic inflammation and pain (Stahl, 1999). In some fibromyalgia patients, levels of substance P has been found to be three times the amount of healthy people (Kossoff, 1999). Dopamine, norepinephrine, and serotonin regulate levels of substance P. These three chemicals appear to be abnormally low in FM patients. Women tend to have lower levels of serotonin than men, which may help to explain why FMS is more prevalent in women than compared to men (Kossoff, 1999).

Substance P is also known to play a role in the regulation of emotions, vomiting, asthma, depression, migraines, anxiety, and the cardiovascular response to stress.

However increasing Substance P receptor antagonists, such as dopamine, norepinephrine, and serotonin, all symptoms above can be alleviated except pain (Stahl, 1999).

Other physical and metabolic findings seen in a subset of FM patients include functional abnormalities in the hypothalamic-pituitary-adrenal (HPA) axis, which may represent a neuroendocrine dysfunction (Rau & Russell, 2000). HPA activity and sex may interact to evoke hyperarousal. Research in rats have found that aversive stimuli and stressful tasks are more likely to evoke sympathetic nervous system and HPA axis responses in females rats compared to male rats. Greater psychological responses in female rats were also reported (Winfield, 2000).

Despite the presence of these physical and metabolic abnormalities, it is again unknown if these abnormalities are due to etiological factors or caused by long-term chronic pain.

Genetic

If there is a genetic component to FM, the relationship still remains unclear. Twin studies do indicate that identical twins are more likely to develop FM than fraternal twins (vanWhy, 1997). This gives support that FM is at least partially caused or influenced by biology. Kossoff (1999) states FM does seem to run in families, so this vulnerability could be set off by genetic factors as well as a traumatic event, viral infection, or a learned way of coping with stress. Other biological variables may include gender since lower levels of serotonin have been found in women, which regulate substance P.

Sleep Disorder

Sleep disturbances in FM patients has been researched extensively, yet its role as a cause or effect of FMS still remains unclear. Research to investigate the patterns of

alpha electroencephalographic sleep and their association with pain in FM patients has provided valuable information (Rozienblatt, Moldofsky, Benedito-Silva, & Tufik, 2001). Rozienblatt and colleagues (2001) discovered three distinct patterns of alpha sleep activity in FM patients. Phasic alpha sleep, which is simultaneous with delta activity, was reported in 50% of FM patients. These patients reported pain worsening after sleep, longer duration of pain, less total sleep time, lower sleep efficiency, and less slow-wave sleep. Slow wave sleep is when the body restores itself and has also shown to prevent oncoming illness. Tonic alpha sleep that was continuous throughout non-REM sleep was reported in 20% of FM patients, and low alpha sleep was reported in 30% of patients. Phasic alpha sleep patients reported the highest increase in tender points after sleep, followed by low alpha sleep and tonic alpha activity. Rozienblatt and colleagues (2001) concluded that the clinical manifestations of FM correlate the best with phasic sleep activity. In other words, more disturbances during restorative, slow wave sleep causes increases in FM symptoms and severity. In addition, other factors such as fatigue, emotional disturbance, tension, and stress can contribute to sleep disturbances and be worsened by it (Baumstrak & Buckelew, 1992).

Growth hormone secretion may also be disrupted due to poor quality of sleep in FM patients (Maurizio & Rogers, 1997). Growth hormone is usually only secreted during stage 4 sleep. Maurizio and Rogers (1997) suggest that sleep is important for tissue restoration, bodily renewal, and regulating many neuroendocrine and immune functions. Deficiency in secretion of growth hormones and the sleep disturbances may help to account for the pain and neuroendocrine and immune abnormalities found in FM patients. However, the causal relationship between sleep disturbances and fibromyalgia remains

unclear-whether sleep disturbances cause fibromyalgia or only if these disturbances worsen the symptoms.

Serotonin Deficiency

The Serotonin Deficiency Model hypothesizes that a deficiency in serum serotonin and higher than normal levels of re-uptake sites for serotonin may account for pain and depressive symptoms, as well as slow-wave sleep disturbances. Furthermore, this deficiency may account for dysfunction of immune cells, a system, which will be discussed later. During slow-wave sleep, serotonin provides an opportunity for physical restoration during NREM, stage 3 and stage 4 sleep. A consistent loss of stage 3 and 4 non-restorative sleep has been observed in other diseases associated with aching and stiffness. Normal individuals will develop FM-like symptoms if they are deprived of stage 4 sleep (Maurizio & Rogers, 1997). The disruption of stage 4 sleep that fibromyalgia patients exhibit is called alpha-delta sleep (Baumstrak & Buckelew, 1992).

Pain modulation is thought to be controlled by the midbrain and to be indirectly mediated by serotonin (Baumstrak & Buckelew, 1992). Support for this model is the demonstrated efficacy of anti-depressant medications, which increases re-uptake of serotonin (Baumstrak & Buckelew, 1992). Another link between serotonin deficiency and fibromyalgia is that serotonin is important in central and peripheral pain mechanisms (Maurizio & Rogers, 1997). However, what causes the serotonin deficiency remains unclear.

Immune System and Viral Infections

The immune system is the body's homeostatic defense mechanisms that protects the body from disease-causing entities by controlling susceptibility to allergens infection,

cancers, and autoimmune disease in which the system attacks its host's normal tissue (Baumstrak & Buckelew, 1992). Interleukin-1 is a product of the central nervous system that is important for proper immune functioning. Interleukin-1 promotes deep sleep in animals and reaches its maximum activity at slow-wave sleep onset in healthy subjects. Since IL-1 has been linked to viral infection, Baumstrak and Buckelew (1992) have speculated that FMS can possibly be a manifestation of a viral infection. Suspected agents responsible for a deficiency in IL-1 and thus creating susceptibility to FM have included Epstein-Barr, cytomegalovirus, human herpes virus 6, enteroviruses, and HIV (Maurizio & Rogers, 1997). These agents might lead FM-prone people into an altered immune system state of "reduced natural killer cell function and increasing interferon activity" (Maurizio & Rogers, 1997). Furthermore, a deficiency in IL-1 may be caused in FM patients by sleep disturbances, thus damaging the immune system (Baumstrak & Buckelew, 1992). Other supporting evidence that FM is an abnormality in the immunologic system is that there are differences in immunoglobulin, cytotkine, tissue metabolism capillary permeability, and increases in CNS substance P (Maurizio & Rogers, 1997). Immunoglobulin and cytotkine are antibodies that protect the body from infection or tissue damage, but also causes hay fever, asthma and hives when these antibodies interact with mast cells to protect against the invading parasites (Parkin & Cohen, 2001). However, fibromyalgia, immune system dysfunction, and viruses remain only as association, since not all people develop FM after a virus infection or having an immune system dysfunction disorder.

Paraoxonase

Many researchers believe that FM is a variation of a functional somatic syndrome (Wessely, Nimuan, Sharpe, 1999). These researchers define a functional somatic syndrome for symptoms that a patient experiences that a doctor cannot medically explain after appropriate medical assessment and that cannot be explained in terms of a conventionally defined medical disease. Other illnesses such as Chronic Fatigue Syndrome and Gulf War Syndrome have similar symptoms as fibromyalgia. Some theories support the notion that all these illnesses can be classified under one name, Complex Widespread Syndrome. One speculative cause of these types of illnesses is paraoxonase polymorphism. Paraoxonase is a mammal enzyme that protects against chlorpyrifos and other organophosphates (Rowat, 1999). Chlorpyrifos are typically the active ingredient in insecticides to protect again disease-bearing pests, and organophosphates are usually used as an insecticide for food. Protection against chlorpyrifos and organophosphates is dependent on which form of the enzyme is inherited. Paraoxonase polymorphism has been suggested to be responsible for the body's ability to resist damage from pesticides. Pesticides can damage the central nervous system and damage the immune system. Supporting evidence for paraoxonase includes heightened reactions to low levels of toxicants in Multiple Chemical Sensitivity Disorder, Chronic Fatigue Syndrome, Fibromyalgia, and Gulf War Syndrome patients (Rowat, 1999). Further research on paraoxonase needs to be investigated since this could help solve the debate of whether these illnesses have a psychological or biological origin. Paraoxonase polymorphism could explain why only certain people develop fibromyalgia after a virus infection, bacterial infection, or exposure to toxicants.

Temporary Hypoxia

New research has shown that local pain in FM patients may be related to temporary hypoxia (Jeschonneck, Grohmann, Hein, & Sprott, 2000). Jeschonneck and colleagues reported abnormal oxygen pressure at the muscle surface above trigger points. Other findings include a reduction in blood flow in the skin above tender points. Pain at tender point locations is correlated with measured reduction in local blood flow. This imbalance between oxygen supply and demand may result in local hypoxia and a decrease in the concentration of high-energy phosphates. High-energy phosphates, such as adenosine triphosphate, are used in the body for energy (muscle movement) and metabolism. There are several morphological findings that support the hypothesis that local pain in fibromyalgia is due to hypoxia. First, the before mentioned "moth-eaten" fibers in trapezius muscle biopsies may be a result of hypoxia. Second, decreased levels of adenosine triphosphate (ATP), adenosine diphosphate (ADP), and phosphoryl creatine (PC) and increased levels of monophosphate and creatine have been correlated with decreased muscle blood flow. This correlation has also been found in other diseases including rheumatoid arthritis (Jeschonneck et al, 2000). Other supporting evidence includes studies done on the efficacy of connective tissue massage. Connective tissue massage has shown to increase blood flow and decrease pain at tender points (Sprott, Jeschonneck, Grohmann, & Hein, 2000). However, what causes the temporary hypoxia in FM patients to initially occur still remains unclear.

Sensory Amplification Syndrome

Another biological theory states that FM is a sensory amplification syndrome. According to this theory, the pain that patients experience is not caused by inflammation in the periphery, but associated with a central defect in pain processing (Clauw, 2000).

Studies done with chronic peripheral pain such as patients with low back pain or arthritis also have dysfunctional central mechanisms similar to FM patients that exacerbate the pain. This is also seen in patients with visceral pain such as patients with irritable bowel syndrome, interstitial cystitis, or chronic pelvic pain. More evidence suggesting that FM may be associated with a central defect in pain processing is that central pain responds differently than peripheral pain to treatment interventions (Clauw, 2000). However, researchers do not know yet what causes such sensory amplification. Furthermore, sensory amplification may cause the experience of chronic pain, but may not cause FM itself. Patients who have chronic pain may also become more attentive to their body and notice pain and changes in the body's homeostasis.

Qualitatively Altered Nociception

The theory regarding qualitatively altered nociception in FM patients is similar to the theory of sensory amplification. Both theories support the idea that FM pain is due to aberrant central pain mechanisms, not peripheral damage or injury. Nociception refers to "the physiologic process of transmitting a noxious stimulus from the periphery to the cerebral cortex where its consequences are interpreted" (Rau & Russell, 2000). Spinal dorsal horn neurons receive input from deep muscle tissues (Bendsten, Norregaard, Jensen, & Olsen, 1997). Stimuli of noxious intensities stimulate high-threshold mechanosensitive (HTM) neurons, while innocuous stimuli activate low-threshold mechanosensitive (LTM) neurons. This means LTM neurons usually do not mediate pain to spinal dorsal neurons. It is hypothesized that FM pain is caused by strong input from peripheral nociceptors, which has remodeled the circuitry of the dorsal horn. Bendsten and colleagues (1997) suggested that the strong input activates previously ineffective

synapses and forms new synaptic contacts between LTM afferents and dorsal horn neurons that normally receive input from HTM afferents. This would actually lead to the perception of pain from even innocuous stimuli. Supporting this theory is that FM patients exhibit allodynia, or perceive pain that is caused by a stimulus that should not normally be painful (Rau & Russell, 2000). If altered nociception is the pathology of FM pain, it still remains unclear what causes the remodeling of the dorsal horn circuitry. Altered nociception could in turn make the FM-prone patient to be hypervigilant, or pay more attention to bodily events (Quinter & Cohen, 1999).

Following altered nociception is the concept of neuroplasticity with chronic pain. Neuroplasticity assumes that pain can spread from injured to noninjured tissue by means of central neural activation. The distant pain can become both widespread and chronic, and even persisting long after resolution of an initial injury (White, Carette, Harth, Teasell, 2000). Studies conducted on animals have proved to show a spread of pain from injured to noninjured tissue through the activation of central neural pathways. White and colleagues (2000) believe that the spread of excitability (central sensitization) to adjacent neuron populations in the dorsal horn may result in a subjective sensation of spreading/radiating pain.

Summary of Theories

Fibromyalgia seems to stem from a biopsychosocial origin, which is a relatively new concept for understanding of disease and illness (Gardner, 2000). Although evidence strongly supports that psychopathology does not cause fibromyalgia, there is still positive correlation for some fibromyalgia patients (Baumstrak & Buckelew, 1992). Whether the pathogenesis of fibromyalgia is due to biological factors or psychological factors, the

diathesis-stress model indicates that all types of factors play an important role in chronic pain. Biology, mood states, and previous experiences and past coping strategies all contribute to the perception of chronic pain.

TREATMENT

Treatment for fibromyalgia should be a multifaceted approach since there is a need to improve symptoms in several different areas. In addition, treatment should provide a specific protocol for each individual since these patients differ in their symptoms and severity of symptoms (Maurizio & Rogers, 1997). The aim of treatment is to reduce pain, fatigue, stress, depression, and to increase self- efficacy and stage 3 and stage 4 sleep. However, since there is still no known "cure" for fibromyalgia syndrome, the main goal of all treatment should be aimed at adapting to chronic pain.

Pharmaceutical Interventions

Anti-inflammatory medication has been shown to help with pain and some stimulants may help improve fatigue (Ser Vaas, 2000). The three classes of antidepressants that have been successful in treating fibromyalgia symptoms are S-adenosylmenthionin, selective serotonin reuptake inhibitors, and tricyclic antidepressants. Tricyclic antidepressants are administered to increase non-REM stage 4 sleep by increasing serotonin levels (Maurizio & Rogers, 1997). Antidepressants have also shown to help depression, increase overall sense of well being, and improve some patients' pain control (Ser Vaas, 2000). Research suggests that fibromyalgia patients taking antidepressants are four times more likely to experience significant relief from symptoms than patients treated with other therapy or no therapy (Ser Vaas, 2000). Growth hormone

therapy to improve sleep disturbances and pain symptoms has also been used with some success.

Exercise

Many studies have demonstrated the role of cardiovascular fitness in improving and preventing FMS symptoms. Baumstrak and Buckelew (1992) discovered that aerobic conditioning might improve sleep in some patients. Exercise may be beneficial to fibromyalgia patients in relieving stress and reducing pain since exercise is a natural therapeutic technique for pain management and stress relief (Secord, 1998). Exercise may help reduce pain levels through a production of endorphins. These morphine-like endorphins may inhibit the release of substance P (Baumstrak & Buckelew, 1992). Exercise can also increase blood flow to affected regions, which may help reduce temporary hypoxia (Secord, 1998). However, most FM patients cannot exert previous levels of exertion and endurance after the onset of fibromyalgia. Finding the right exercise program is specific to each individual's pain toleration. Exercise programs should be started gradually and built up slowly. Stretching, strengthening, aerobic conditioning, fast walking, bicycling, Tai Chi, Yoga, and pool exercises in a heated pool for fibromyalgia patients have also been reported as being helpful (Secord, 1998).

Acupuncture and Massage

Another way to increase the production of endorphins is through acupuncture. Acupuncture is a Chinese therapeutic technique that involves solid needles inserted into the skin at different depths to raise endorphin levels (Starlanyl & Copeland, 1996). In normal acupuncture, the needles are inserted on the body's meridians, or sites where suggested energy paths help signal the brain to send healing to the area of pain. For

fibromyalgia patients, not only are body meridians used, but also actual tender point sites are used.

Studies have shown that FM patients demonstrate a reduction in regional blood flow above tender points compared to healthy controls (Sprott, Jeschonneck, Grohmann, & Hein, 2000). This reduction in blood flow may cause pain and a lower pain threshold. Acupuncture has shown to increase blood flow above tender points and increase pain threshold levels. While there has been mixed reviews on acupuncture's effectiveness, Sprott and colleagues (2000) found that the number of tender points reported by patients were lower after acupuncture.

Connective tissue massage therapy has been proven effective for some FMS patients. Connective tissue refers to the tissues that hold the body together and surround the organs. This type of massage breaks up scar tissue and releases toxins found in the connective tissue (Starlanyl & Copeland, 1996). Brattberg (1999) found that after a series of fifteen treatments of connective tissue massage therapy, patients reported a 37% pain reduction and a reduction in depression and use of analgesics. In addition, patients reported a positive effect on quality of life. However, three months after the fifteen treatments, patients reported their relief diminished; after six months, patients reported 90% of pain back to the reported basic value (Brattberg, 199). Connective tissue massage does not "cure" FM symptoms, only relieve or reduce the symptoms temporarily. The massage does not guarantee that the tender points will not resurface. As with most FM interventions, connective tissue massage therapy might be beneficial to some patients, but may need to be continually implemented and maintained.

Another therapy focusing on reducing pain at tender points is tender (or trigger) point injections. Tender point injections are considered a useful and safe adjunct for therapy for FMS (Reddy, Yunus, Inanici, & Aldag, 2000). Injections consist of procaine in an isotonic saline without epinephrine and are injected directly into the tender point site. Reddy and colleagues (2000) found that injections reduce pain in the tender points and generalized pain. However, Starlanyl and Copeland (1996) have found that FM patients with Myofascial Pain Syndrome (MPS) do not respond to trigger point injections effectively.

Self Care Strategies and Stress Reduction Techniques

Any treatment modality to reduce stress is beneficial for FM patients. The person's underlying stress often exacerbates FM flare-ups and stress itself can induce flare-ups in FM patients. Self-care strategies are implemented as an effective technique of treatment for FM patients to reduce levels of stress, depression, and anxiety. Reduction of these emotional experiences also reduces pain and flare-ups. Self-care strategies include rest, paging, prioritizing, and self-talk.

Rest as a strategy includes mind and body rest throughout the day. Types of rest that Hulme (1998) recommends are meditation, breathing awareness, skeletal muscle release, and internal organ quieting. Paging means organizing a FM patient's day into multiple work rest play sections. For example, tasks on jobs should be divided into two or three components. This coping skill strategy has also been termed "activity pacing" (Sandstrom, 1998). Prioritizing involves choosing which work is more important, delegating work to others, and scheduling work times during the week. Self-talk can help lessen negative thoughts. Hulme (1998) recommends FMS patients to become aware of

mind talk and replace negative thoughts with positive, hopeful statements. This cognitive behavioral technique has been shown to be effective in improving self-esteem and self-efficacy not only in FM patients, but also in the normal population. Another way to become aware of mind talk is to keep a daily journal about thought and feelings and to review the journal for negative thoughts and feelings (Hulme, 1998).

Other self-care strategies often recommended to FM patients are relaxation techniques such as hypnosis, guided imagery, and body awareness (Sandstrom, 1998), as well as problem-solving techniques, support groups, heat therapy, and proper nutrition (Maurizio & Rogers, 1997; Millea & Holloway, 2000). Educating family, friends, and coworkers is also seen as important since the common population is not informed about FM and fibromyalgia patients do not exhibit visible signs and symptoms of their disorder (Maurizio & Rogers, 1997). Since people affected with fibromyalgia look like normal people, others may not believe they are sick or understand the severity of the symptoms. Social support has been shown to significantly help reduce symptoms and increase adaptation to any chronic illness.

Melatonin

Some studies have investigated whether if melatonin treatment improves disturbed sleeping, fatigue, and pain symptoms in FM patients. Melatonin was thought to be a possible effective treatment for FM patients since other studies have shown melatonin supplementation to be beneficial in initiating and maintaining sleep in elderly patients suffering from insomnia alone or associated with symptoms of depression. Melatonin, a serotonin derivative, is synthesized by the pineal gland. One important physiological function of melatonin and the pineal gland is to "convey information

concerning the daily cycle of light and darkness to the body's physiology" (Citera, Arias, Maldonado-Cocca, Lazaro, Rosemffet, Scheines, & Cardinalli, 2000). Melatonin indicates the length of light by patterns of secretion. The daily secretions of melatonin constitute the organization of the circadian sleep-wake cycle by being an efferent hormonal signal. Citera and colleagues (2000) reported that a 3-mg/day-melatonin dose at bedtime could improve sleep in FM patients and also lessened reported tender point counts.

Tropisetron

Another study for treatment of FMS has looked at intravenous application of tropisetron (Stratz, Farber, Varga, Ulrike, Baumgartner, & Muller, 2000). Stratz and colleagues (2000) conducted a double-blind study to discover if intravenous application of tropisetron can reduce pain in fibromyalgia patients. Tropisetron plays a role in serotonin metabolism, which seems to be part of the pathophysiology of FM. Lowered levels of tryptophan in plasma and serotonin in serum and plasma have been found in FM patients. Lower levels of serotonin may be due to serotonin binding to 5-hydroxytryptamine-3 receptor. This binding releases substance P. Tropisetron is a 5-HT receptor antagonist that inhibits the release of substance P. It assumed that blocking the release of substance P via tropisetron could reduce pain. This study discovered that tropisetron injections reduced FM patients' reported pain levels (Stratz et al, 2000).

TYPES OF ONSET

Unlike the theories of biological mechanisms, there is a general consensus in the research on what precipitating factors contribute to the onset of fibromyalgia. Many patients report that FM symptoms began to develop after a physical or emotional

traumatic event (Aaron, Bradley, Alarcon, Triana-Alexander, Alexander, Martin & Alberts, 1997). Other subsets of FM patients report an idiopathic onset, or an onset that did not follow a perceived traumatic event. Traumatic onset can be further classified into two subgroups: emotional trauma or physical trauma. Types of trauma that have been reported by FM patients include motor vehicle accidents with or without physical injury, surgery, viral illnesses, repetitious strain injury, childbearing, infectious diseases, and bacterial infections. Viral illnesses that have been associated with FM include human herpes virus 6, parvovirus, HIV, and many others. Bacterial infections that have been reported included Brucellosis and Lyme disease. Other reported physically traumatic events have included silicone implants and dental amalgam leaking from fillings (van Why, 1997). Emotional trauma that has been associated with the development of FM includes family death or illness, divorce, and separation from family and friends. The importance of identifying the two types of onset (traumatic vs. idiopathic) is important since the two present different clinical pictures and subgroups may respond to treatment differently (Turk, Farber, Starz, Sinclair, 1996b). By classifying FM patients into subgroups, treatment can become more cost efficient and effective.

Several differences have been found between the of FM onset subgroups. Patients where FM reportedly occurs after a traumatic event tend to be more impaired socially, financially, and functionally than patients that report idiopathic onset (Turk et al, 1996b). Turk and colleagues (1996b) also found that patients who report a traumatic onset of FMS are significantly more likely to take opioid medications, receive physical therapy, nerve block, and TENS treatment than patients who report an idiopathic onset. Traumatic

onset patients also report significantly greater levels of pain, interference of symptoms with regular activities, affective distress, and functional impairments.

Turk and colleagues (1996b) found that FMS patients could be empirically classified further into three categories using the Multidimensional Pain Inventory (MPI). The MPI is used to classify chronic pain patients into psychosocial subgroups. Those patients receiving "dysfunctional" classification tend to exhibit high degrees of pain, interference, functional limitation, and affective distress. Patients in the "interpersonally distressed" group are characterized by possessing low degrees of social support and high degrees of punishing responses from others. Low degrees of pain and affective distress and high degrees of internal locus of control characterize patients classified as "adaptive copers". This means these patients had more self-determination than the other patients had, and believed they had control over their thoughts and actions. Turk and colleagues (1996b) report that 87% of FMS patients can be classified into the three groups. Most post-traumatic FM patients were classified as dysfunctional or interpersonally distressed. In contrast, most idiopathic FM patients were classified as adaptive copers. Turk and colleagues (1996b) also found that traumatic onset "dysfunctional" patients did not respond well to typical FM treatment, while the other groups responded well to typical interdisciplinary treatment.

In comparing physical trauma to emotional trauma and idiopathic onset, characteristics of patients with physically traumatic onset were more likely to report being unemployed, to be receiving disability compensation, and to be engaging in levels of physical activity (Aaron et al, 1997). Indeed, Aaron and colleagues (1997) found that a physically traumatic onset of fibromyalgia is associated with a more-disabling disease

course. Patients with a physical traumatic onset are seen more in public hospital settings than in private practice and use tertiary-level health care. This may be due to these subgroup of patients receiving monetary compensation for more specialized services (Aaron et al, 1997). Patients with physical traumatic onset may have won a lawsuit or have insurance that covers the medical treatment since they had a physical injury that can be covered.

It has been estimated that 14% of patients who attend specialized rheumatology practices experience FMS symptoms after an emotionally traumatic event (Aaron et al, 1997). Patients who report FMS characterized by emotional onset also have been reported to visit a greater number of physicians in with a six months time period compared to patients with physical trauma onset or no trauma history. This applies as well to the general population and patients with rheumatic diseases. Perceived stressful life events and reports of emotional distress predict an increase in health care-seeking behavior (Aaron, 1997). The term for emotional distress to account for post accident symptoms has been called "accidental neurosis" (Kuch, Evan, Watson, & Bubela, 1991). This suggests that accidents without an injury can still be psychologically traumatizing, with the individual's thought process becoming centered around fear of repetition of this accident or feeling that type of pain again (Kuch et al, 1991).

The differences between the two onsets are speculated to be due to factors other than physical abnormalities, (Turk et al, 1996a) much like other pain disorders. For example, pain severity and disability in chronic back pain patients cannot be determined merely on the basis of physiological pathology. Turk and colleagues (1996a) believe that the experience of a traumatic event may alter an individual's ability to evaluate sensory

information, consequently changing a person's pain perception and thresholds. Low pain tolerance has been reported in other pain patients following a traumatic event such as whiplash (Turk et al, 1996a). A traumatic onset causing a person to be hypervigilant may explain why patients with traumatic onset report fibromyalgia symptoms to be more severe. This supports the hypothesis that post-traumatic FMS is more problematic than idiopathic FMS (Aaron et al, 1996).

COPING STYLES, ANGER EXPRESSIONS, AND PAIN

The traditional biomedical model assumes that the perception of pain is always the result from an organic pathology (Turk & Okifuji, 1999). While the biomedical model may explain the relationship between pain and nociception for acute pain, it does not adequately explain chronic pain. The biopsychosocial model describes pain perception as being composed of several factors: nociception, an individual's mood states, and previous experience. Chronic pain patients do not report their perception of pain based solely on nociception, but rely on other factors such as cultural conditioning, expectations, social contingencies, mood states, and perception of control (Turk & Okifuji, 1999). Consequently, assessing and treating a person's pain may require a knowledge of the patient's current mood, way of coping with the symptoms, and the response of others toward the patient, including family members and the physician.

Maladaptation to a stress such as fibromyalgia is often explained using Lazarus and Folkmans' cognitive-phenomenological theory of stress (Bombardier, 1999). This theory posits that adaptation and maladaptation to a stress is mediated by two factors, appraisal and coping. Appraisal concerns the personal significance of the stressor and one's resources with dealing with it. Coping concerns the thoughts and behaviors used to

manage the demands of the stressor. Coping with chronic illness derives from appraisal processes. Judgments about the likelihood that fibromyalgia can limit one's adaptive resources may determine what forms of coping patterns will be emphasized (Bombardier, 1990). Coping strategies can be categorized into two functional domains, problem-focused and emotion-focused. A problem-focused strategy aims at managing the external aspects of the stressor, such as changing one's behavior and thoughts or environmental conditions. An emotion-focused strategy regulates the internal affective consequences of the stressor, such as wishful thinking that the pain will go away. In low back pain patients, emotion-focusing coping was negatively correlated with an averaged weekly pain rating (Bombardier, 1990). In chronic pain patients, emotion-focused coping has been reported to have a negative impact on adjustment while problem-focused coping has had a positive effect on adapting to chronic pain. Some studies on coping and its relation to anxiety and depression have found that anxiety and depression is positively correlated with emotion-focused coping (Bombardier, 1990). Appraisal of chronic illness as "holding one back" has seemed to be a particularly maladaptive way of perceiving one's medical condition. Bombardier (1990) found that a "holding back" appraisal predicted greater emotion-coping responses, poorer adjustment to illness, greater depressive symptoms, and poorer psychosocial adjustment. In contrast, optimistic and confronting types of coping strategies as well as problem-focused coping are the most effective in adapting to the stress of chronic pain (Hermann, Scholmerich, & Straub, 2000). Optimistic and confronting styles of coping mean the patient believes that treatment can reduce FM symptoms, and that the patient will address which stressors can be alleviated.

Anger has been found in almost all chronic pain patients. However, anger-based

coping styles among FM patients have not been researched extensively (Air, Neumann, Bor, Shir, Rubinow, & Buskila, 2000). Generally, chronic pain patients have been characterized by greater use of avoidant coping styles, more state anger, and more anger turned inward. Air and colleagues (2000) used the State-Anger Expression Inventory by Spielberger to discover if anger patterns in FM patients differed from other pain patients. They found that specific traits among FM patients were not different than rheumatoid arthritis patients, low back pain patients, and healthy controls. However, Air and colleagues did not identify if certain subgroups of FM patients have different anger patterns. Anger turned inward has been associated with maladaptation to chronic pain (Air et al, 2000). Distraction has been found to be an effective mechanism to cope with chronic pain when the pain is at low intensity. This may be an effective coping strategy for FM patients since it has been hypothesized that FM patients attend to somatic complaints such as pain more than normal people.

The goal of this thesis is to examine in onset subgroups of FM patients have similar coping patterns, appraisal techniques, and anger styles. Research has shown that adaptation to chronic illness may be affected by how patients appraise and cope with the stress of their illness (Bombardier, 1999). Past research on coping strategies in FM patients has only described the entire FM population as an undifferentiated whole. FMS patients have reported more daily hassles compared to other chronic disease patients (Air, Neumann, Bor, Shir, Rubinow & Buskila, 2000). Reported perceived stressful life events in childhood and adolescence appear to be very common among FM patients (Anderberg, Marteinweottir, Theorell, von Knorring, 2000). Anderberg and colleagues (2000) also found that FM patients report life events as experienced more negatively than healthy

controls. The worse mental health among FM patients has been associated with more hassles, more emotion coping, and less satisfaction with social support (Da Costa, Dobkin, Fitzcharles, Fortin, Beaulieu, Zummer, Senecal, Goulet, Rich, Choquette, & Clark, 2000). Anderberg and colleagues (2000) suggest that the diathesis-stress model might account for muscle ischemia secondary to muscular reactivity to emotional stressors in FM patients. This implies the maladaptive coping strategies can cause body responses to increase levels of pain. Since type of onset of FM has shown to respond to different treatments, it may be beneficial to discover if the different types of onset subgroups have different coping patterns and anger styles. By identifying coping patterns and anger styles, one can predict how a patient will adapt to the chronic illness.

HYPOTHESES

The primary objective of this thesis is to discover if subgroups of fibromyalgia patients have different coping styles, appraisal techniques, and anger traits. In an attempt to test group differences, five hypotheses were developed for this thesis. Hypothesis One predicts that Emotional Trauma Onset and Idiopathic Trauma Onset subgroups will have maladaptive appraisal techniques (higher scores on appraisal) than Physical Trauma Onset subgroups. Hypothesis Two predicts that the Emotional Trauma Onset and Idiopathic Onset subgroups will have lower problem-focused strategies than the Physical Trauma Onset subgroup. Hypothesis Three predicts that the Physical Trauma Onset subgroup will have significantly lower emotional-focused strategies than the other two subgroups. To examine anger expressions, Hypothesis Four predicts that the Emotional Trauma Onset and Idiopathic Onset subgroups will have higher anger-in scores than the Physical Trauma subgroup. Hypothesis Five predicts that the Emotional Trauma Onset

and Idiopathic Onset subgroups will score higher than the Physical Trauma Onset subgroup on anger control. Hypothesis Six predicts that the Emotional Trauma Onset subgroups and Idiopathic Onset subgroups will have higher State and Trait anger.

CHAPTER TWO

METHOD

The purpose of this chapter is to discuss the methodology used in the collection of data and the analysis of the data. The three major sections are subjects, materials, and procedures.

SUBJECTS

Subjects were recruited from three sites in New Braunfels, Texas: a rheumatology clinic, a massage therapy clinic specializing in massage for fibromyalgia patients, and a support group at McKenna Rehabilitation for fibromyalgia patients. Subjects were either receiving treatment for fibromyalgia or attending a support group meeting. Subjects picked up the survey battery that were left for them and mailed them to the investigator. Exclusion criteria included male; fibromyalgia patients are typically female, so the study focused on females. Ethnicity, social economic status, or having other medical disorders was not identified or used as exclusion criteria. Participants consisted of thirty females who had all been previously diagnosed with fibromyalgia by a health care professional such as a rheumatologist, pain management doctor, or family practitioner. The subjects had a mean age of 57 with a SD = 12.28.

MATERIALS

Survey

A battery of surveys along with a consent form was distributed to each subject. The survey battery consisted on four sections: demographics, appraisal techniques, coping styles, and anger traits. The survey battery along with the consent form can be found in Appendix A. All materials were submitted and approved by Southwest Texas State University IRB.

The one hundred and twenty-five-item survey took about thirty-five minutes to complete. Sections of the battery are as follows:

Demographic Section

Five items on the survey asked for demographical information. Fill in the blank questions included age, sex, duration of diagnosis (in years), and types of health care physician who made the diagnosis. The last demographical question asked was type of onset. This included physical trauma onset (PTO): virus, illness, motor vehicle accident, allergies, injury, or other. Emotional trauma onset (ETO) was defined as loss of a family member or close friend, divorce, depression, separation from family/friends, and other. Idiopathic onset (IO) was defined as: I don't know what caused the onset, nothing, or other. Participants read a short description of type of onset before circling one or more of type of onset.

Appraisal Section

The battery assessed appraisal techniques using the Appraisal Dimensions Scale (ADS), which is a self-report measure with Likert response options. The ADS assesses

thoughts and behaviors associated with six types of appraisal techniques: control, salience, novelty, duration, causality, and predictability. This scale contains six 4-items scales to make a 24-item test. The Likert scale was from 1 to 5, ranging from "strongly disagree" to "strongly agree". According to Lazarus and Folkman's cognitive-phenomenological theory of stress, appraisal is defined as "personal significance of the stressor and one's resources dealing with it" (Bombardier, et al, 1990). Combining all six subscales made a total appraisal score. Because the ADS is still in an experimental stage, no psychometric data is available. Reliability for the ADS in this study was fairly low, with an alpha of .38.

Coping Styles Section

Coping strategies were measured in this battery using the Ways of Coping Checklist (WCCL) by Folkman and Lazarus. This test is also a self-report measure with Likert response options. This scale includes 57 items with a Likert scale from 1 to 4, ranging from "never used" to "regularly used". The WCCL identifies the thoughts and actions an individual has used to cope with a specific stressful encounter. The test identifies eight types of cognitive coping strategies: problem-focused coping, wishful thinking, seeking social supports, avoidance, self blame, blame of others, counting of one's blessings and religiosity. According to Lazarus and Folkman's cognitive-phenomenological theory of stress, coping is defined as the "thoughts and behaviors used to manage the demands of the stressor" (Bombardier, et al, 1990). Reliability for the WCCL in this thesis was found to be .364.

Anger Traits Section

Anger expressions and experience were measured in this battery by using the State-Trait Anger Expression Inventory (STAXI) by Spielberger. This test is designed to measure the experience and expression of anger. This scale is a 44-item self-rating questionnaire with Likert response options. The scale is divided into three sections: 1) How I feel right now, 2) How I generally feel, and 3) When I am angry or furious. "How I feel right now" has nine questions with responses on a 4-point scale, ranging from "not at all" to "very much so." The section on "How I generally feel" contains ten questions with responses on a 4-point scale, ranging from "almost never" to "almost always." The last section titled "When I am angry or furious" contains twenty-four questions with a Likert scale from 1 to 4, ranging from "almost never" to "almost always." The STAXI consists of six scales and two subscales, but only two scales were used in the analysis: Anger-In and Anger Control. Anger-in describes angry temperament. Anger-In measures if the patient "turns anger inside" or suppresses anger. Angry Reaction is described by Anger-Control. Anger Control measures the frequency with which the patient tries to control expressions of anger. State Anger (how the subject feels right now) and Trait Anger (how the subject usually feels) were also addressed. Reliability for the STAXI in this study was found to be .68 after recoding anger control.

PROCEDURE

Contact was made with three collaborating sites to receive written consent to administer the survey. The proposal, along with the written permission from each site, was approved by the Southwest Texas State University Graduate Office and the IRB.

Surveys were administered to fibromyalgia patients at three sites in New Braunfels, Texas: 1) a support group for FM at McKenna Rehabilitation, 2) a

rheumatology clinic, and 3) a massage therapy clinic. A therapist at the massage therapy clinic specialized in massage for fibromyalgia. Survey packets were left at each site. Fifteen packets were left at the massage clinic, 30 at the rheumatologist, and 42 were mailed to support group members. Each packet included two consent forms (one to keep and one to return), the battery of surveys, and a self-addressed stamped envelope to return the survey. All survey packets were distributed to potential subjects at each site, but only thirty were returned to the investigator. All thirty surveys returned were used for this thesis.

STATISTICAL DESIGN

Statistical analysis was completed on SPSS. Descriptive statistics were completed to describe the demographical information reported by the participants. Five separate one-way ANOVAs were conducted to compare group differences on appraisal, problem-focused, emotion-focused, anger in, and anger control scores. Two additional one-way ANOVAs were conducted on State Anger and Trait Anger. Correlations were conducted on the dependent variables.

CHAPTER THREE

RESULTS

SAMPLE DEMOGRAPHICS

Results should be interpreted with caution due to missing data. Table One shows the sample demographics for this survey. Thirty surveys were returned from the three collaborating sites. All thirty subjects were female and had a mean age of 57 (SD = 12.28), with two participants not responding to age. The mean length of diagnosis for this sample was 7.9 (SD = 4.23), with three participants not filling in the length of their diagnosis.

When asked what type of health care professional diagnosed them with fibromyalgia, the majority of subjects reported that a rheumatologist gave the diagnosis. Specifically, eighteen or 82% of subjects reported that a rheumatologist diagnosed them with the disorder. Other professionals that reportedly made the diagnosis included 2 (9.1%) family practitioners, 1 (4.5%) nurse practitioner, and 1 (4.5%) pain management physician. Eight subjects did not specify what health care professional made the fibromyalgia diagnosis. This means that eight of the subjects in this study may have not even been diagnosed with fibromyalgia, but were actively seeking help for FM symptoms.

Table 1
Demographic
Characteristics

	N	Missing	Mean	SD
Age	28	2	57.0	12.2777
Diagnosis	27	3	7.9259	4.261

	Frequency	Valid Percent
Physician		
Rheumatologist	18	81.8%
Family practitioner	2	9.1%
Nurse practitioner	1	4.5%
Pain management doctor	1	4.5%
Missing	8	
Type of Onset		
Physical	11	36.7%
Emotional	11	36.7%
Idiopathic	8	26.7%

Type of onset was distributed fairly evenly. Eleven (36.7%) participants reported physical trauma onset, 11 (36.7%) reported Emotional Trauma Onset, and 8 (26.1%) reported Idiopathic Onset.

Table 2
Dependent Variable
Correlations and
Descriptive Statistics

	Mean	SD	N
Appraisal	85.7	6.28	30
Problem-focused	45.13	5.94	30
Emotion-focused	115.5	186	30
Anger-in	17.03	5.28	30
Anger control	24.93	6.28	30
State anger			
Trait anger			

	APP	PF	EF	AI	AC	SA	TA
Appraisal (APP)	1.00	.295	.342	.442*	-.081	.396*	.255
Problem-focused (PF)	.295	1.00	.168	.083	-.016	.298	.134
Emotion-focused (EF)	.342	.168	1.00	-.073	-.117	.373*	.329
Anger-in (AI)	.442*	.083	-.073	1.00	-.160	.424*	.455*
Anger control (AC)	-.081	-.016	-.117	-.160	1.00	.014	-.613**
State Anger (SA)	.396	.298	.373*	.424*	.014	1.00	.125
Trait Anger (TA)	.255	.134	.329	.445*	-.613**	.125	1.00

Note:
*. Correlation is significant at the 0.05 level (2-tailed).
**. Correlation is significant at the 0.01 level (2-tailed).

CORRELATIONS OF THE DEPENDENT VARIABLES

Table Two shows the descriptive statistics and the correlations between the seven dependent variables: appraisal, problem-focused, emotion-focused, anger-in, anger control, state anger, and trait anger. Significant correlations were found at the 0.05 level for appraisal and anger-in (r = .442), appraisal and state anger (r = .396), emotion-focused and state anger (r = .373), anger-in and state anger (r = .424), and anger-in and trait anger (r = .455). The only correlation significant at the 0.01 level was anger control and trait anger (-.613).

TESTING OF THE RESEARCH HYPOTHESES

Table 3 shows the five one-way ANOVAs that were conducted to compare type of onset group differences on appraisal techniques, coping styles, and anger expressions.

Hypothesis One predicted that Emotional Trauma Onset and Idiopathic Onset groups would report higher "maladaptive" appraisal techniques than the Physical Trauma Onset group. A one-way ANOVA was conducted to find group differences in appraisal scores. There was no statistically significant difference (F = .714, alpha = .498), indicating that type of onset did not impact appraisal techniques.

Hypothesis Two predicted that the Physical Trauma Onset group would report higher problem-solving strategies than the Emotional Trauma Onset and Idiopathic Onset groups. A one-way ANOVA was conducted to find group differences in problem-focused scores. There was no statistically significant difference in problem-focused scores (F = .2.399, alpha = .110), indicating that type of onset groups did not differ in problem-focusing strategies.

Hypothesis Three predicted that the Emotional Trauma Onset and Idiopathic Onset groups would report higher emotion-focused strategies than the Physical Trauma

Table 3
One-way ANOVAs

	SS	df	Mean Squares	F	Sig.
Emotion-focused					
Between groups	84496	2	42248	1.249	.303
Within groups	913457	27	33832		
Total	997953	29			
Anger-in					
Between groups	58.739	2	29.370	1.057	.361
Within groups	750.227	27	27.786		
Total	808.967	29			
Anger control					
Between groups	30.776	2	15.388	.826	.449
Within groups	503.091	27	18.633		
Total	533.867	29			
Appraisal					
Between groups	57.516	2	28.758	.714	.498
Within groups	1086.784	27	40.251		
Total	1144.300	29			
Problem-focused					
Between groups	155.648	2	77.824	2.399	.110
Within groups	875.818	27	32.43		
Total	1031.467	29			
State Anger					
Between groups	16.310	2	8.155	.277	.760
Within groups	795.057	27	29.447		
Total	811.367	29			
Trait Anger					
Between groups	.201	2	.100	.006	.994
Within groups	431.966	27	15.999		
Total	432.167	29			

Onset group. A one-way ANOVA was conducted to find group differences in emotion-focused scores. There was no statistically significant difference (F = 1.249, alpha = .361), indicating that type of onset does not impact emotion-focused strategies.

Hypothesis Four predicted that Emotional Trauma Onset and Idiopathic Onset groups would report turning anger inward more than the Physical Trauma Onset group. A one-way ANOVA was conducted to find group differences in anger-in scores. There was no statistically significant difference in anger-in scores (F = 1.057, alpha = .361), indicating that type of onset does not impact the tendency to turn anger inward.

Hypothesis Five predicted that Emotional Trauma Onset and Idiopathic Onset groups would report more anger control than Idiopathic Onset. A one-way ANOVA was conducted to find group differences in anger control scores. There was no statistically significant difference in anger control scores (F = .826, alpha = .449), indicating that type of onset groups do not differ in anger control.

Hypothesis Six suggested that Emotional Trauma Onset and Idiopathic Onset would report more State and Trait Anger. Two one-way ANOVAs were conducted to find group differences in State and Trait Anger. There was no statistically significant different in State and Trait Anger scores (F = .006, alpha = .994).

CHAPTER FOUR

DISCUSSION

Chapter four discusses the results and conclusions drawn from the statistical analysis. The chapter is divided into two major sections. The first section presents a discussion of the findings and offers possible explanations of the results. The second section describes weaknesses of the study, and explores areas of further research.

CONCLUSIONS

Research Results

Research Sample

The sample used in this thesis was small (n=30), but was deemed representative of the fibromyalgia population. Consistent with the research findings that 90% of fibromyalgia patients are female (van Why, 1997), all the participants in this study were female. Fibromyalgia has also been found to be one of the most common disorders seen in outpatient rheumatology clinics (Turk et al, 1996). In this study, 82% of subjects who responded reported that a rheumatologist diagnosed them with fibromyalgia. Although FM has been reported to manifest in childhood and adolescence, FM patients generally do no receive a diagnosis until after eight years or more of suffering from FM symptoms. Consistent with this research, most of the FM subjects in this study were largely middle-

aged adults. In this study, the mean age for the sample was 57 (SD = 12.28) with two people not responding to age. Despite the small sample size for this study, it was representative of the FM population.

Significant Research Correlations

Five of the correlations were significant at a 0.05 level. The correlation between appraisal and anger-in, (r = .442), suggests that subjects who have "maladaptive" appraisal techniques, such as feeling out of control and not knowing the course of their illness, tend to suppress anger. The correlation between appraisal and state anger (r = .396) suggests that subjects who have maladaptive appraisal techniques also reported being more angry during the survey. The correlation between emotion-focused and state anger (r = .373) suggests that subjects using emotion-focused strategies also were angrier while filling out the survey. Anger-in was correlated with state anger (r = .424) and trait anger (.455). This suggests that subjects who tend to suppress anger also tended to be angrier during the survey and report being angrier in general. The highest correlation found was between anger control and trait anger, significant at a 0.01 level (r = -.613). This finding suggests that subjects who tend to be angry also try to control their anger more. So, while the subjects may feel a great deal of anger regarding their situation, they nonetheless appear to find it important to control the expression of such negative emotion. This may be due to past difficulties getting affirmation and/or social support for their pain and impairment. This may also point to a common personality trait among FM patients. The reasons behind this common finding are as of yet unclear.

Research Hypotheses and Findings

None of the hypotheses predicted were proven in this study. This study predicted that type of onset (IO, TO) would be associated with different appraisal techniques. No group differences were found, indicating that onset type did not impact appraisal in this study. This result may have been due to the actual scale used to assess appraisal techniques, which may not have been sensitive enough to detect differences. For example, two of the subscales (novelty and duration) had statements that all subgroups would answer similarly. Novelty items, or statements that assessed how familiar FM was to the subjects, generally received high scores. The mean of length of diagnosis was 7.9 years, with only one patient reporting being diagnosed for one year. Since it takes an average of eight years for a patient to receive a diagnosis, all study subjects were obviously familiar with symptoms of FM. Duration, or how long FM will last, is another appraisal subscale that FM subjects should answer similarly. Since FM patients are informed that FM is a lifelong pain disorder without a known cure, the study participants were all informed about how long the illness would last. FM patients would consequently have similar scores on duration. Based on these shared qualities, this test may have not been a valid instrument to detect FM subgroup differences on appraisal techniques. This may also explain the paucity of research data on appraisal in FM subjects.

In addition to different appraisal techniques, this study predicted that different type of onset would yield different coping techniques. Specifically, this study suggested that Emotional Trauma Onset and Idiopathic Onset would report higher scores on emotion-focused strategies than FM patients with Physical Trauma Onset. This study also hypothesized that the Physical Trauma Onset subgroup would report higher problem-focused strategies than Emotional Trauma Onset and Idiopathic Onset subgroups.

However, there was no difference found between the three subgroups in either of the coping strategies. The lack of differences could have been due to selection bias. All participants came from three distinct treatment sites: a massage clinic, a rheumatologist, and a support group. As all these subjects were actively looking for relief from fibromyalgia symptoms, they may all have similar active, problem-focused coping strategies. The method of data gathering and recruiting could also have resulted in selection bias. People who voluntarily returned the survey and who also actively seek outside help may represent only a part of the fibromyalgia patient population. Those who are more passive copers, or who may see alternative treatment to massage, rheumatology, or support groups will consequently not be included or assessed by this study.

This study predicted that type of onset would impact quality of anger expression. Emotional Trauma Onset and Idiopathic Onset subgroups were expected to report higher scores on anger-in and anger control than the Physical Trauma Onset subgroup. Specifically, this investigator believed that the Physical Trauma Onset group would have lower anger-in and anger control scores because they had a specific, physical event to link to the onset of their fibromyalgia. However, this may not make a difference. As no group differences were found. Physical trauma that was reported to be associated with FM onset included motor vehicle accidents, surgeries, viruses, and injuries. These types of physical trauma are fairly common, and most people do not develop lifelong, widespread body pain after these events. Consequently, having a physical event to link to fibromyalgia may not have any impact on anger expressions.

This also supports Air and colleagues' (2000) research on chronic pain patients' anger traits compared to healthy controls. The results found all chronic pain patients to

have similar anger traits (more state anger and more anger turned inward) compared to healthy controls. These differences indicate that it may not be the specific disease (or from this study, not the type of onset) that determines anger patterns, but the mere fact that the patients are suffering from chronic pain. Living in a constant state of pain may predict anger expressions such as turning anger inward and controlling anger better than type of onset.

Weaknesses of this Thesis and Areas of Future Research

The major weakness of this study is the method of recruitment and data collection. Since the sample size was so small, each subject should have been interviewed. This would have significantly reduced the amount of missing data. Not all subjects answered questions about their age, length of diagnosis, and what type of health care professional made the diagnosis. Type of onset could have also been further explored. It would have been interesting to examine whether any subjects felt that two or more events were linked with the onset of their fibromyalgia, perhaps revealing combinations of both emotional and physical trauma onset. As has been discussed previously, recruiting patients from the three sites only allowed for a representation of FM patients that actively seek out help. Subjects could have also been interviewed to explore what type of help they seek in addition to the three sites. This would have been helpful to see if subjects who seek several areas of treatment adapt to FM more effectively than patients who seek less treatment.

Another weakness of this study was that the survey distributed to the patients may have been too long and may have caused fatigue. The survey took about thirty-five minutes to complete and consisted of one hundred and twenty-five-items. This may have

been too long for a chronic pain patient to maintain an interest in the study. The survey could have been shorter, for example, by focusing on one or two of the topics instead of all three. Having subjects report their current level of pain and typical level of pain could also have been reported to see if high levels of pain would make a difference in scores. Research has shown that current mood states such as anxiety, depression, and anger, are relative to pain levels (Turk & Okifuji, 1999). Subjects with higher levels of pain might have reported more anger turned inward and more anger control.

Another weakness of this study was sample size. The total size of this sample was 30, and each type of onset group had only 8 to 11 participants. A larger sample size might have yielded different results for some of the hypotheses. This sample also only consisted of females. Although fibromyalgia disproportionately affects females more than men, similar issues regarding appraisal, coping techniques and anger traits may also apply for male fibromyalgia patients. Group differences based on gender could also be explored.

Some researchers on coping have found that the current instruments do not have much predictive power, nor do they provide consistent results from study to study. Furthermore, there has yet to be developed a coping theory that improves adherence to preventative and treatment behaviors (Baum, Revenson, & Singer, 2001). This may be due to the reality that a general coping model cannot be supplemented for all types of people. For example, avoidance as an emotion-coping technique is usually considered a "maladaptive" strategy in coping and believed to cause poorer adjustment to illness (Bombardier, et al, 1990). However, other researchers believe avoidance in chronic pain patients is an effective strategy since pain in chronic pain patients will always persist. Air and colleagues (2000), for example, found avoidance or "distraction" to be an effective

coping mechanism when chronic pain is at low intensity. If a person must live with chronic pain, not all problem-focused coping techniques will be adaptive since the problem, pain, can never be truly resolved. Confronting the problem would always end in no solution and may increase frustration, since the pain would never completely be eradicated. Emotional coping techniques such as avoidance may actually be a good way to adapt of adapting to chronic illness, because it can help to reduce pain perception and make life more bearable.

Therefore, research should be concentrated on which coping mechanisms, whether they are emotion-focused or problem-focused, are effective in reducing pain and promoting positive adaptation and function in fibromyalgia patients. After identifying effective coping mechanisms for fibromyalgia, then research can be done to see if type of onset groups differ in regards to these effective coping mechanisms. Appraisal techniques could also be examined in chronic pain patients, since appraisal techniques may differ from a healthy person's techniques. With chronic pain patients, any appraisal must include the realization that their pain will persist. Therefore, research on adaptation to chronic illness through appraisal techniques and coping mechanisms should be further explored.

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APPENDICES

APPENDIX A

The following is the survey that was distributed to three sites in New Braunfels Texas: a rheumatology clinic, massage therapy clinic, and a support group.

Coping Styles, Anger Styles, and Classification of Fibromyalgia Patients

You are invited to participate in a study of anger and coping styles in people who have been diagnosed with fibromyalgia. I am a graduate student at Southwest Texas State University at San Marcos, Psychology Department. This study is part of Masters thesis. You were selected as a possible participant in this study because you have been diagnosed with fibromyalgia. You will be one of about forty subjects chosen to participate in this study.

If you decide to participate, you will be asked to complete a survey about coping styles and anger styles that will take about twenty minutes to complete.

Any information that is obtained in connection with this study and that can be identified with you will remain confidential and will be disclosed only with your permission. The thesis will be available at the Southwest Texas library, but subject identities will not be included.

Your decision whether or not to participate will not prejudice your future relations with Southwest Texas State University or where you are taking this survey (massage therapy clinic or support group). If you decide to participate, you are free to discontinue participation at any time without prejudice.

If you have any questions, please ask me. If you have any additional questions at a later time, Dr. Leticia Flores, my committee chair will be happy to answer them. Contact with Dr. Flores can be made through the psychology department at Southwest Texas State University. (lf11@swt.edu)

You will be offered a copy of this form to keep.

You are making a decision whether or not to participate. Your signature indicates that you have read the information provided above and have decided to participate. You may withdraw at any time without prejudice after signing this form should you choose to discontinue participation in this study.

_____ _____

Signature of Participant Date

_____ _____

Signature of Investigator Date

Please fill in this information before completing the test.

AGE: _____
SEX:_____
DURATION OF DIAGNOSIS (in years): _____
TYPE OF HEALH CARE PHYSICIAN WHO MADE THE DIAGNOSIS:

TYPE OF ONSET:

Popular theory believes that certain stressors trigger the onset of fibromyalgia. This means trauma does not **cause** fibromyalgia, but finally triggers the symptoms to be noticeable (like your first flare-up).

Traumatic onset means that fibromyalgia symptoms became present after a physical or emotional traumatic event. Idiopathic onset means the onset of fibromyalgia occurred without any stressful factors.

I would like you to circle what you perceived as the onset of fibromyalgia. I would also like to remind you that this information will be kept confidential. No names will be given, which means only numbers in the type of onsets will be recorded and discussed.

Please circle **one** or **more**:

Physical onset:
Virus
Illness
Motor vehicle accident
Allergies
Injury
Other: _____

Emotional Onset:
Loss of a family member or close friend

Divorce
Depression
Separation from family/friends
Other: _____

Idiopathic Onset:
I do not know what caused the onset
Nothing
Other: _____

Rate the degree to which you agree/disagree with the following statements describing the major problem listed below.	strongly disagree	disagree	mixed feelings	agree	strongly agree
PROBLEM: DEALING WITH FIBROMYALGIA					
1. I believe my problem is controllable.	1	2	3	4	5
2. My problem is very important to me.	1	2	3	4	5
3. I am quite familiar with these kinds of problems.	1	2	3	4	5
4. I believe my problem is only temporary.	1	2	3	4	5
5. I have not played a part in my problem.	1	2	3	4	5
6. I know the course my problem will follow.	1	2	3	4	5
7. My problem is nothing to be concerned about.	1	2	3	4	5
8. My problem is something new to me.	1	2	3	4	5
9. My problem will not go away.	1	2	3	4	5
10.My problem is a result of my own doing.	1	2	3	4	5
11.I am not certain how my problem will proceed.	1	2	3	4	5
12.I believe my problem is out of control.	1	2	3	4	5
13.My problem is of serious concern to me.	1	2	3	4	5
14.My problem is a new kind of experience for me.	1	2	3	4	5
15.This is just a short lived problem.	1	2	3	4	5
16.There is something that can be done about my problem.	1	2	3	4	5
17.My problem is predictable.	1	2	3	4	5
18.My problem is not the result of my own behavior.	1	2	3	4	5
19.My problem is not a big deal.	1	2	3	4	5
20.I have experienced this type of problem before.	1	2	3	4	5
21.My problem will probably last a long time.	1	2	3	4	5
22.My actions have contributed to my problem.	1	2	3	4	5
23.Little can be done to change my problem for the better.	1	2	3	4	5
24.I don't know what's going to happen next with my problem.	1	2	3	4	5

The items below represent ways that you may have dealt with the stressor (dealing with fibromyalgia). We are interested in the degree to which you have used each of the thoughts or behaviors represented in these items in order to deal with the stressor. Please circle the appropriate column if the thought/behavior was: never used; rarely used; sometimes used; or regularly used (at least 4 ot 5 times per week)	never used	Rarely used	Sometimes used	Regularly used
THOUGHTS/BEHAVIORS				
1. Bargained or compromised to get something positive from the situation.	1	2	3	4
2. Count my blessings.	1	2	3	4
3. Blamed yourself.	1	2	3	4
4. Concentrated on something good that could come out of the whole thing.	1	2	3	4
5. Kept my feelings to myself.	1	2	3	4
6. Figured out who to blame.	1	2	3	4
7. Hoped a miracle would happen.	1	2	3	4
8. Asked someone I respected for advice and followed it.	1	2	3	4
9. Prayed about it.	1	2	3	4
10.Talked to someone how I was feeling.	1	2	3	4
11Stood my ground and fought for what I wanted.	1	2	3	4
12.Refused to believe that it happened.	1	2	3	4
13.Criticized or lectured yourself.	1	2	3	4
14.Took it out on others.	1	2	3	4
15.Came up with a couple of different solutions to the problem.	1	2	3	4
16.Wished I were a stronger person--more optimistic and forceful.	1	2	3	4
17.Accepted my strong feelings, but didn't let them interfere with other things too much.	1	2	3	4
18.Focused on the good things in my life.	1	2	3	4
19.Wished that I could change the way I felt.	1	2	3	4
20.Changed something about myself so that I could deal with the situation better.	1	2	3	4
21.Accepted sympathy and understanding from someone.	1	2	3	4
22.Got mad at the people or things that caused the problem.	1	2	3	4
23.Slept more than usual	1	2	3	4
24.Spoke to my clergyman about it.	1	2	3	4
25.Realized you brought the problem on yourself.	1	2	3	4
26.Felt bad that I couldn't avoid the problem.	1	2	3	4
27.I knew what had to be done, so I doubled my efforts and tried harder to make things work.	1	2	3	4
28.Thought that others were unfair to me.	1	2	3	4

29.Daydreamed or imagined a better time or place than the one I was in.	1	2	3	4
30.Tried to forget the whole thing.	1	2	3	4
31.Got professional help and did what they recommended.	1	2	3	4
32.Changed or grew as a person in a good way.	1	2	3	4
33.Blamed others.	1	2	3	4
34.Went on as if nothing had happened.	1	2	3	4
35.Accepted the next best thing to what I wanted.	1	2	3	4
36.Told myself things could be worse.	1	2	3	4
37.Talked to someone who could do something concrete about the problem.	1	2	3	4
38.Tried to make myself feel better by eating, drinking, smoking, taking medications, etc.	1	2	3	4
39.Tried not to act too hastily or follow my own hunch.	1	2	3	4
40.Changed something so things would turn out right.	1	2	3	4
41.Avoid being with people in general.	1	2	3	4
42.Thought how much better off I am than others.	1	2	3	4
43.Had fantasies or wishes about how things might turn out.	1	2	3	4
44.Just took things one step at a time.	1	2	3	4
45.Wished the situation would go away or somehow be finished.	1	2	3	4
46.Kept others from knowing how bad things were.	1	2	3	4
47.Found out what other person was responsible.	1	2	3	4
48.Thought about fantastic or unreal things (like the perfect revenge or finding a million dollars) that made me feel better.	1	2	3	4
49.Came out of the experience better than I went in.	1	2	3	4
50.Told myself how much I have already accomplished.	1	2	3	4
51.Wished that I could change what had happened.	1	2	3	4
52.Made a plan of action and followed it.	1	2	3	4
53.Talked to someone to find out about the situation.	1	2	3	4
54.Avoided my problem.	1	2	3	4
55.Relied on my faith to get my through.	1	2	3	4
56.Compared myself to others who are less fortunate.	1	2	3	4
57.Tried not to burn my bridges behind me, but left things open somewhat.	1	2	3	4

A number of statements that people use to describe themselves are given below. Read each statement and then fill in the circle with the number which indicates how you feel rightnow. Remember that there are no right or wrong answers. Do not spend too much time on any one statement, but give the answer which seems to best describe you present feelings.	not at all	Somewhat	Moderately So	Very Much So
HOW I FEEL RIGHT NOW				
1. I am furious.	1	2	3	4
2. I feel irritated.	1	2	3	4
3. I feel angry.	1	2	3	4
4. I feel like yelling at somebody.	1	2	3	4
5. I feel like breaking things.	1	2	3	4
6. I am mad.	1	2	3	4
7. I feel like banging on the table.	1	2	3	4
8. I am burned up.	1	2	3	4
9. I feel like swearing.	1	2	3	4
A number of statements that people use to describe themselves are given below. Read each statement and then fill in the circle with the number which indicates how you generally feel. Remember that there are no right or wrong answers which seems to best describe how you generally feel.	Almost never	Sometimes	Often	Almost Always
HOW I GENERALLY FEEL				
11. I am quick tempered.	1	2	3	4
12. I have a fiery temper.	1	2	3	4

13. I am a hotheaded person.	1	2	3	4
14. I get angry when I'm slowed down by others' mistakes.	1	2	3	4
15. I feel annoyed when I am not given recognition for doing good work.	1	2	3	4
16. I fly off the handle.	1	2	3	4
17. When I get mad, I say nasty things.	1	2	3	4
18. It makes me furious when I am criticized in front of others.	1	2	3	4
19. When I get frustrated, I feel like hitting someone.	1	2	3	4
20. I feel infuriated when I do a good job and get a poor evaluation.	1	2	3	4
Everyone feels angry or furious from time to time, but people differ in the ways that they react when they are angry. A number of statements are listed below which people use to describe their reactions when they feel angry or furious. Read each statement and then fill in the circle with the number which indicates how often you generally react or behave in the manner described when you are feeling angry or furious. Remember that there are no right or wrong answers. Do not spend too much time on any one statement.	Almost never	Sometimes	Often	Almost Always
WHEN I AM ANGRY OR FURIOUS.....				
21. I control my temper.	1	2	3	4
22. I express my anger.	1	2	3	4
23. I keep things in.	1	2	3	4
24. I am patient with others.	1	2	3	4
25. I pout or sulk.	1	2	3	4
26. I withdraw from people.	1	2	3	4
27. I make sarcastic remarks to others.	1	2	3	4
28. I keep my cool.	1	2	3	4
29. I do things like slam doors.	1	2	3	4
30. I boil inside, but I don't show it.	1	2	3	4
31. I control my behavior.	1	2	3	4
32. I argue with others.	1	2	3	4
33. I tend to harbor grudges that I don't tell anyone about.	1	2	3	4

34. I strike out at whatever infuriates me.	1	2	3	4
35. I can stop myself from losing my temper.	1	2	3	4
36. I am secretly quite critical of others.	1	2	3	4
37. I am angrier than I am willing to admit.	1	2	3	4
38. I calm down faster than most people.	1	2	3	4
39. I say nasty things.	1	2	3	4
40. I try to be tolerant and understanding.	1	2	3	4
41. I'm irritated a great deal more than people are aware of.	1	2	3	4
42. I lose my temper.	1	2	3	4
43. If someone annoys me, I'm apt to tell him or her how I feel.	1	2	3	4
44. I control my angry feelings.	1	2	3	4

VITA

Suzanne Kneuper was born in Oletha, Kansas, on January 8, 1977. She graduated high school with honors from New Braunfels High School in New Braunfels, Texas. Suzanne received her Bachelors of Science in Psychology and minor in Biology at Southwest Texas State University. She graduated cum laude. During graduate school, Suzanne was an Instructional Assistant in the Psychology Department and taught competitive gymnastics. In December 2001, Suzanne Kneuper will receive her Masters of Arts in Health Psychology from Southwest Texas State University.

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