CHRONIC PAIN AND SHORT-TERM MEMORY:

IS A LONGER DURATION OF CHRONIC PAIN IN FIBROMYALGIA PATIENTS ASSOCIATED WITH A GREATER IMPAIRMENT OF SHORT-TERM MEMORY FUNCTION?

THESIS

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Michael J. Olds, B.S.

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CHAPTER 1

INTRODUCTION

Fibromyalgia is a disease both controversial and complicated. It has no known etiology, many ancillary manifestations, rather subjective diagnoses, and no definitive treatment (Hubbard, 1999). Research into etiology and treatments are often found to be contradictory or profoundly different from previous or current research in the field, fueling the fires of pre-existing controversies in this area. Areas that research has indicated could be possible etiologies include sleep disturbances, muscle metabolism, and muscle deconditioning (Anch, Lue, MacLean, & Moldofsky, 1991; Bennett, 1989). There are even some professionals who debate the existence of fibromyalgia as it is defined, claiming it to be manifestations of other problems, including but not limited to problems of a psychological nature such as depression.

Fibromyalgia is considered a form of nonarticular rheumatism that typically presents as chronic, diffuse, aching pain in the muscles (Hubbard, 1999). In 1990 the American College of Rheumatology outlined what it determined to be the diagnostic criteria for fibromyalgia. These diagnostic criteria are fulfilled when the patient has eleven out of eighteen specified tender points and widespread pain in all four quadrants of their body for a minimum of three months (Wolfe, Smythe, & Yunis, 1990). These tender points are areas that are extremely tender to the touch and are located at specific, predictable

anatomical sites (see Figure 1), with a lack of tenderness at corresponding control sites (Hubbard, 1999).

In addition to these diagnostic criteria, there are a number of secondary symptoms associated with fibromyalgia. These include, but are not limited to, cognitive and memory impairments, depression, sleep disturbance, fatigue, post-exertion malaise, irritable bowel, chronic headaches, peripheral vascular instability resembling Raynaud's phenomenon (excessive sensitivity to cold), parasthesias, a subjective sensation of peripheral swelling without objective edema, and fluctuation of symptoms in response to changes in weather or stress level (Hubbard, 1999; Smythe, Bennett, & Wolfe, 1993). Because so many of these symptoms overlap with the symptomology other disease processes such as rheumatoid arthritis and Lyme disease, the presence of the aforementioned tender points remains the only reliable basis for diagnosis with fibromyalgia (Goldenberg, 1989).

Although descriptions of symptoms resembling fibromyalgia date back to biblical days, it was not recognized as a distinct syndrome until after Gowers' introduction of it under the name "fibrositis" in 1904 (Smythe, 1989). Since that time there have been many research studies seeking to answer questions concerning fibromyalgia. The results of these studies have been inconclusive for the most part, leaving the scientific community with no universally agreed upon etiology, treatment, or understanding of the processes of fibromyalgia.

While the physical features of fibromyalgia have received a good deal of attention, the psychological factors involved have received considerable scrutiny as well. In the 1950s and 1960s fibromyalgia was often considered a manifestation of hysteria and was

frequently equated with psychogenic rheumatism (Goldenberg, 1987). More recently fibromyalgia patients have been found to demonstrate elevated scores for depression, hypochondriasis, and hysteria on the Minnesota Multiphasic Personality Inventory(MMPI) and similar scales (Goldenberg, 1989; Payne, Leavitt, & Garron, 1982). The neuropsychological complaints of fibromyalgia patients reveal some other areas that should be examined. Common complaints include depression, as well as difficulty remembering and general forgetfulness (Hubbard, 1999). The complaints involving memory functioning touch on an area in which the research literature is sadly lacking. The few studies examining this area tend to be inconclusive and often contradictory (Hubbard, 1999; Kaplan, Meadows, Vincent, Logigian, & Steere, 1992; Landro, Stiles & Sletvold, 1997; Sletvold, Stiles & Landro, 1995).

Statement of Problem

Although there is a good deal of research available on psychological symptoms and comorbidities related to fibromyalgia, a relatively small portion of it deals with the patients' self-reported psychological complaints. While depression is generally recognized as a component of fibromyalgia, the self-reported complaints of difficulty remembering and general forgetfulness have only been specifically examined in four studies to date (Hubbard, 1999; Kaplan et al., 1992; Landro et al., 1997; Sletvold et al., 1995). The results of these four studies have been mixed, inconclusive, and at times contradictory.

Hubbard, (1999) found that the cognitive functioning of the fibromyalgia patients within her study was significantly better than their self-reported complaints of

concentration and memory difficulties. However, it was found that the subjects' scores on the Brown Adult Attention-Deficit Disorder Scale did correlate significantly with both their self-reported complaints of memory impairments and their elevated scores on the Symptom Checklist-90-R (SCL-90-R) and the Centre for Epidemiological Studies in Depression Scale (Hubbard, 1999). These clinically elevated scores on the Brown Adult ADD scale did not, however, correlate with their objective memory performance. As with much of the literature on fibromyalgia, these subjects were found to have a greater number of psychological symptoms than the general populace, particularly more symptoms of depression (Hubbard, 1999).

Kaplan et al., (1992) examined the relationships between fibromyalgia, depression and memory function and reported no significant differences between fibromyalgia patients and depressives. They suggested that these two groups' subjective memory complaints had no organic basis and were instead due psychological factors such as distress and difficulty in attending to information (Kaplan et al., 1992). This study's findings suggest that the subjective memory complaints so often reported by fibromyalgia patients could be solely attributable to psychological factors.

Landro et al. (1997) compared memory functioning in patients with primary fibromyalgia, major depression and healthy controls. They found that both the depressed group and the fibromyalgia group had significant impairment on long-term memory tasks requiring effortful processing, but when the depressive status of the fibromyalgia patients was controlled for, only the subsample with a history of lifetime major depressive disorder showed a significant memory impairment (Landro et al., 1997). This study reiterated findings from a previous study of theirs which also examined information

processing in fibromyalgia patients. Both of these studies found that fibromyalgia patients showed a deficiency in psychomotor speed, independent of a history of major depressive disorder (Landro et al., 1997; Sletvold et al., 1995). While major depressives also show deficits in psychomotor speed, in these studies current depressive status and history of depression are controlled for. The earlier of the two studies also found that fibromyalgia patients without a history of lifetime major depressive disorder had a significant non-specific deficit in information processing ability (Sletvold et al., 1995). While both of these studies are exploratory in nature, they speculate that the reported memory impairments of fibromyalgia patients could at least partially result from a slowed rate of information processing or slowed psychomotor speed (Landro et al., 1997; Sletvold et al., 1995).

Due to its high rate of comorbidity with fibromyalgia, chronic fatigue syndrome (CFS) must also be examined in research concerning fibromyalgia. Based upon the standard classification criteria, 50% to 70% of fibromyalgia patients either have a current or past diagnosis of CFS (Goldenberg, 1999). Chronic fatigue syndrome shares many of the neuropsychological symptoms of fibromyalgia that intuitively seem to contribute to impaired cognitive functioning. These symptoms include, but are not limited to, depression, muscle pain, sleep disturbance and fatigue (Barrows, 1994). As with research into fibromyalgia, the studies on CFS have had inconclusive and sometimes contradictory results (Hubbard, 1999).

One such study found CFS patients to have significant memory deficits in several areas including memory consolidation, vulnerability to interference, and slow or uncertain decision making (Sandman, Barron, Nackoul, Goldstein, & Fidler, 1993).

Another concluded that self-reported complaints of cognitive impairment were due to depression after finding no significant deficits attributable to CFS itself (Schmaling, DiClementi, Cullum, & Jones, 1994). Yet another study concluded that there was no evidence of any significant cognitive impairments due to CFS, and once again implicated depression as the culprit in these types of self-reported complaints (Krupp, Sliwinski, Masur, Freidberg, & Coyle, 1994).

The neuropsychological complaints of fibromyalgia patients are clearly an area that needs more scrutiny. Very few studies have been conducted that specifically examine self-reported cognitive impairments in fibromyalgia patients, although such studies are needed and have been recommended to the professional community (Landro et al., 1997). The studies in this area that have been conducted have had relatively small sample sizes, and have yielded mixed and sometimes contradictory results. Hubbard's (1999) research used a sample size of 34, while other studies into possible cognitive impairments of fibromyalgia patients had sample sizes ranging from 11 to 25 participants (Hubbard, 1999). Whether fibromyalgia patients suffer from cognitive impairments or not is still a question that has not been truly answered. But even if there were a definitive positive answer to that question, it is unknown whether these impairments are due to biological processes of fibromyalgia, or are merely manifestations of psychological comorbidities such as depression.

This study will focus on one of the neuropsychological complaints most commonly reported by fibromyalgia patients – deficits of working memory. Working memory describes an individual's capacity to process information, and is an update of what many laymen and professionals of the past would call short-term memory. Working memory is

considered a key component of learning and the most active part of the information processing system (Wechsler, 1997). Working memory uses prior knowledge, associations, and the placing of information into categorical groups to facilitate the encoding of new information into long-term memories. A deficit of the working memory system could easily account for fibromyalgia patients' self-reported problems with difficulty in remembering things and general forgetfulness.

Previous studies have suggested that fibromyalgia patients had deficits of working memory attributable to difficulties in concentrating and sustaining attention (Hubbard, 1999). Others have explained the cognitive deficits reported by fibromyalgia patients as being due to depressive symptoms (Kaplan et al., 1992; Landro et al., 1997; Sletvold et al., 1995). While these hypotheses could certainly have credence, there are yet other processes that could also serve to explain in part these widespread complaints of memory dysfunction.

For this study it is hypothesized that fibromyalgia can lead to cognitive impairments through a complex biological chain of events. In any pain response the spinal cord sends a signal to the sympathetic nervous system, which causes it to enter a state of arousal and release an increased amount of glucocorticoids (Bonica, 1990). Individuals dealing with chronic pain for a long period of time would be expected to show neuroendocrine activity similar to patients with other conditions resulting in sympathetic arousal over a long period of time. One such condition that has been examined at length is posttraumatic stress disorder (PTSD).

PTSD shares a number of features with fibromyalgia. In both conditions the sympathetic nervous system stays aroused on an almost continuous basis and the

individual's limbic system becomes hypersensitized to noxious stimuli (Ray, 2002). Shalev, Peri, & Brandes (2000) found that patients with PTSD had an increased sensitivity to sound, while the same results were found in fibromyalgia patients by McDermid, Rollman, & McCain (1996). This increased sensitivity to sound is indicative of hypersensitization of the somatic pain response, which leads to the sympathetic nervous system staying in a state of near constant arousal.

Studies have repeatedly shown that the higher levels of glucocorticoids involved with prolonged sympathetic arousal in PTSD result in memory deficits and possible damage to the hippocampus (McEwen, 1992; Bremner & Narayan, 1998; Golier & Yehuda, 1998; Sapolsky, 1999). The hippocampus is a structure of the brain that is considered intimately involved with the formation of memories, and lesions of the hippocampus have been shown to interfere with working memory functions (Zigmond et al., 1999).

This exploratory study will follow in others' footsteps by trying to determine objectively whether or not fibromyalgia patients suffer from cognitive deficits, particularly in the area of working memory. If these deficits are found, this study hopes to show if they are in part due to the length of time that the system has been subjected to the heightened levels of glucocorticoids involved with prolonged sympathetic arousal. As with previous works, this study will also examine the relationship between depression and memory impairments in fibromyalgia patients.

Statement of Purpose

Although the medical community has known of the existence of fibromyalgia for some time now, there are still more questions than answers concerning this disorder.

There still is no definitive treatment that shows consistent long-term benefits, although many different treatments are implemented on a regular basis. Some of these medical treatments include tricyclic antidepressants, seretonin reuptake inhibitors, tranquilizers, muscle relaxants, painkillers, anti-epileptics, anti-inflammatory drugs, physical therapy, stretching, and tender point steroid injections (Wolfe, 1997). It is suggested that many of the treatments used for fibromyalgia could also possibly promote a reliance on medication and quite a few of them have little or no research support (Wolfe, 1997).

Wolfe (1997) also suggested that only through developing a greater understanding of the psychological status of fibromyalgia patients can we come to a true understanding of fibromyalgia itself. He notes that for many fibromyalgia patients, psychological factors such as depression, anxiety, psychosocial issues, inappropriate pain behavior and somatization are more problematic than the physical issues relating to their fibromyalgia (Wolfe, 1997).

While examining the psychological status of these patients is important in developing an understanding of fibromyalgia, examining their neuropsychological status can also be important in its treatment. Those treating fibromyalgia patients need to know if their patients are having problems in one or more areas of information processing just as badly as they need to know whether or not their patients are clinically depressed. Memory deficits could prove to be problematic in any group that is commonly prescribed psychotropic medications. Poor memory could lead to increased odds of developing an unhealthy addiction to medication, or even possible overdoses. If the medical community is aware of these dangers, it can serve to avoid possible complications of prescribed

medications. Medication could possibly be administered in a more controlled fashion, and doctors could be more wary of over-medication.

The primary purpose of this exploratory study is to contribute to the literature on fibromyalgia and the neuropsychological complaints that often accompany it. While memory problems are a common complaint among fibromyalgia patients, the professional community is still divided on whether these memory deficits actually exist, and if so, what they are attributable to. This study hypothesizes that memory deficits do exist in fibromyalgia patients, and that these deficits are in part due to duration of sympathetic arousal from chronic pain. This study will be unable to determine definitively whether memory deficits are due to heightened levels of glucocorticoids as it will not be measuring glucocorticoid levels in the blood. However, it will hopefully pave the way for other studies to examine this from a medical and biological standpoint.

If this study does find memory deficits in fibromyalgia patients based on duration of pain, it hopes to contribute to better ways of understanding and treating it in the future. If there are significant impairments of memory, physicians should be aware of this when prescribing psychotropic medications that could have serious consequences when taken too frequently. If this study's hypotheses are supported, they could also point out the importance of addressing the issue of sympathetic arousal due to chronic pain in regards to long-term neurological damage. If this study does find memory impairments attributable to duration of pain, then physicians should take any complaints of chronic pain more seriously, whether they find an organic basis or not.

CHAPTER 2

REVIEW OF LITERATURE

History of Fibromyalgia

While fibromyalgia syndrome has only been recognized as a distinct process relatively recently, there is some evidence from historical accounts that it may in fact be nothing new to us. There are some historical accounts taken from the bible that seem to fit the description of fibromyalgia, as well as descriptions of notable Victorians such as Charles Darwin and Florence Nightingale (Smythe, 1989). Darwin and Nightingale were both lifelong invalids, yet they both lived to quite old ages for the times. There are also personal accounts from the mid 1800s that appear to be a perfect match with what we now know about fibromyalgia (Goldenberg, 1987). In 1824 Balfour described tender points associated with rheumatism, and in 1841 Valleix also described tender points in his treatise on neuralgia (Goldenberg, 1987). There is no way to determine for certain if any of these historical figures did in fact have fibromyalgia, but they seem to show that the symptomology of fibromyalgia is not something that is specific only to the past century or to any particular country.

The modern conception of fibromyalgia really only began in 1904, when William Gowers first introduced the term "fibrositis" based on the marked tenderness he had found to be associated with some regional pain syndromes (Smythe, 1989). Gowers also

noted the presence of fatigue and sleep disturbances, an absence of systemic or localized inflammation, and an extreme sensitivity to touch (Goldenberg, 1989). In the 1930s a study was conducted by Lewis and Kellgren, which described referred pain due to injections of a hypertonic saline solution into deep muscle tissue (Smythe, 1989). Referred pain is pain in which the source of the pain is not the same part of the body where the pain is experienced. Referred pain is believed to be mediated by the central nervous system, and examples could include phantom limb pain and the pain sometimes felt in the arms when one is having a heart attack. Lewis and Kellgren found that the distribution of pain referral patterns was uniform and differed from dermatomal patterns (Smythe, 1989). These patterns helped contribute to the development of using fixed trigger points as part of the diagnostic criteria of fibromyalgia (Goldenberg, 1989).

Even after Gowers coined the term fibrositis and described its symptoms, there were many professionals who did not accept it as a valid syndrome. For over half of the twentieth century, fibromyalgia was considered by many to be a form of "tension rheumatism" or psychogenic rheumatism (Smythe, 1989). The absence of any biological markers led most of the rheumatology community to consider it to be entirely psychogenic, in effect only existing in the patients' minds (Goldenberg, 1989). For those who did not consider it to be entirely psychogenic, the term "fibrositis" became a catch-all diagnosis for patients with vague symptoms that did not fit the diagnostic criteria for rheumatoid arthritis, osteoarthritis, muscle strains, bursitis, tendonitis, degenerative arthritis, or systemic lupus erythematosus (Goldenberg, 1989).

In 1977, the term "fibromyalgia" was introduced by Hench, which replaced the term "fibrositis" for most professionals dealing with this syndrome (Bruckle, Nenndorf, &

Muller, 1992). The term "fibrositis" helped contribute to the controversies concerning the existence of this syndrome, as the term itself actually means "inflammation of the fibrous tissue". Clinical examinations typically did not find any true inflammation, making the term in essence a misnomer. "Fibromyalgia", on the other hand, means "pain in the fibrous tissue". Fibromyalgia is currently believed to have a physical basis, although all of the physical processes involved with it are not identified and/or fully understood. Although there are certainly exceptions, for the most part fibromyalgia patients are no longer dismissed as having a problem that is "all in their head".

Epidemiology

Fibromyalgia has been found to exist in all industrialized countries, and affects around 2% of the adult population, including 3.9% of women ages 20-40, and 5.8% of women between the ages of 40 and 60 (Bruckle et al., 1992; Wolfe, 1997). Other estimates have ranged between 2.1% of patients in family practice clinics, 5.7% of patients in general medical clinics, 5.8% of patients in hospital settings, and 12% of patients seen by rheumatologists (Goldenberg, 1989; Smythe et al., 1993). Although the mean age of onset is in the late 40s, both children and the elderly can be affected as well (Smythe et al., 1993).

Eighty to ninety percent of patients are women, and fibromyalgia is considered the third most prevalent rheumatologic disorder after rheumatoid arthritis and osteoarthritis (Smythe, 1991). Fibromyalgia is estimated to affect 2% of the general population, but it is estimated to affect 3.4% of women and only 0.5% of men (Goldenberg, 1999). While it is uncommon for men to be diagnosed with fibromyalgia, when they are there is usually a slightly earlier age of onset (Hubbard, 1999). Fibromyalgia seems to hit middle-aged

Caucasian women the most, but other groups are affected as well (Felson, 1989). In the American College of Rheumatology's 1990 criteria study, 89% of patients were female, 93% were Caucasian, 5% were Hispanic and 1% were African-American (Wolfe et al., 1990).

American College of Rheumatology (1990) Diagnostic Criteria

In 1990 the American College of Rheumatology laid down its diagnostic criteria for fibromyalgia. These criteria were developed through a study comparing 293 fibromyalgia patients with 265 control patients with regional chronic musculoskeletal pain or a systemic rheumatic disease (Goldenberg, 1999; Wolfe et al., 1990). For these criteria to be met, the patient must have:

A) At least eleven of the eighteen specified tender points (see Figure 1)

B) Widespead pain in all four quadrants of the body for a minimum of three months (Wolfe et al., 1990).

Based on the symptoms of widespread pain and sensitivity in at least 11 of 18 tender points, this study found a sensitivity of 88% and specificity of 81% in distinguishing fibromyalgia from other causes of chronic musculoskeletal pain (Goldenberg, 1999; Wolfe et al., 1990). These tender points are found in predictable locations as nine bilateral pairs in the following anatomical locations (see Figure B-1):

1. Where the back of the head meets the neck.
2. Halfway down the side of the neck.
3. The middle of the trapezius (the muscle that stretches from the back of the neck to the shoulder.
4. About halfway down the inside border of the shoulder blade.

5. Between the 2nd and 3rd rib about one inch from the breastbone.
6. Just past the outer prominence of the elbow.
7. Just behind the edge of the outer hip.
8. The mid portion of the buttocks.
9. Just above the inside of the knee (Csillag, 1992; Fritz, Paholsky, & Grosenbach, 1999).

Patients can also be diagnosed with fibromyalgia without the eleven of eighteen tender points if they have widespread pain in all four quadrants of the body and many of the secondary symptoms commonly associated with it. Some of the more common of these secondary symptoms are listed as fatigue, cognitive or memory impairment, jaw pain, post exertion malaise and muscle pain, difficulties sleeping, morning stiffness, skin sensitivities, numbness and tingling sensations, irritable bowel, chronic headaches, menstrual cramping and PMS, and dizziness or impaired coordination (Smythe et al., 1993).

Clinical Features of Fibromyalgia

As previously noted, the main feature of fibromyagia is diffuse chronic pain through all four quadrants of the body. This pain typically starts in one central location such as the neck, back or shoulder, then becomes more diffuse and generalized as time goes on (Goldenberg, 1989). The most common complaint from fibromyalgia patients is that they "hurt all over" (Hubbard, 1999). This contrasts with many other chronic pain conditions in that they tend to produce pain that is relatively localized, while fibromyalgia patients often have difficulty locating the exact location where they hurt (Hubbard, 1999). Upon

examination, patients will also have severe pain elicited by palpitation of tender points at specific anatomical sites, with a lack of tenderness at corresponding control sites (Wolfe et al., 1990).

Another of the most common complaints of fibromyalgia patients is that they are almost always fatigued and usually wake feeling unrefreshed, regardless of the amount of time that they slept (Goldenberg, 1999). Studies have repeatedly found that sleep disturbances are reported by approximately 75-95% of fibromyalgia patients (Anch, Lue, MacLean, & Moldofsky, 1991; Goldenberg, 1989). These sleep disturbances typically consist of disruption of delta sleep by the intrusion of alpha brain waves (Thorson, 1994). Alpha waves are brain waves that are generally associated with a state of being awake and conscious, while delta waves are the brain wave patterns typically associated with very deep non-REM sleep.

Yunus, Masi and Aldag (1989) studied the clinical features of 113 patients with primary fibromyalgia as compared to 77 patients with rheumatoid arthritis and 67 healthy controls. They asked these subjects to report what symptoms they experienced to a moderate or severe degree and received the following results from the primary fibromyalgia group; 94% reported pain, 85% reported general fatigue, 79% reported morning fatigue, 76% reported general stiffness, 72% reported anxiety, 62% reported poor sleep, 60% reported that they "hurt all over", 60% reported mental stress, 40% reported having a "swelling feeling", 37% reported depression and 36% reported having parasthesias (Yunus et al., 1989).

Cognitive dysfunction is another area in which fibromyalgia patients often have complaints. These self-reported neuropsychological complaints tend to revolve around

difficulties with memory, attention and concentration (Hubbard, 1999; Krupp et al., 1994; Landro et al., 1997; Schmaling et al., 1994; Sletvold et al., 1995). However, objective studies of these neuropsychological complaints have yielded mixed and inconclusive results. Although the research on this area is inconclusive, these complaints of cognitive impairment often lead to costly and invasive neurological testing (Simms, Goldenberg, Felson, & Mason, 1988).

Fibromyalgia patients typically have a long list of comorbid problems and ancillary manifestations. Some of these include chronic fatigue syndrome, irritable bowel syndrome, temporomandibular joint syndrome (TMJ), depression, anxiety, sleep disturbances, post exertion malaise and muscle pain, numbness and tingling sensations, skin sensitivities, Raynaud's-like symptoms (excessive sensitivity to cold), severe PMS and menstrual cramping, dizziness, stiffness, parasthesias, headaches, impaired coordination and cognitive or memory impairments (Hubbard, 1999; Smythe et al., 1993).

The severity of fibromyalgia symptoms does not tend to remain static, but rather seems to fluctuate in response to environmental stressors. The factors most commonly implicated in exacerbating fibromyalgia symptoms are emotional stress, inactivity, poor sleep and changes in weather conditions (Goldenberg, 1989). Symptoms also seem to show a circadian fluctuation in which symptoms are most severe in the early morning and late in the day (Goldenberg, 1989).

Fibromyalgia Etiology

Although fibromyalgia is now thought to have a physical basis, there is still no widely accepted explanation of what causes this syndrome. Pain is usually associated with inflammation of some sort, yet researchers have failed to find the presence of inflammation to explain the symptoms experienced by fibromyalgia patients. There have not been any consistent pathological lesions or laboratory abnormalities found in fibromyalgia patients to explain their symptoms either (Hudson, Hudson, Pliner, Goldenberg, & Pope, 1985). Biopsies have been performed on fibromyalgia patients' tender points, yet they have not showed any histologic abnormalities as compared to healthy controls (Yunis & Kalyan-Raman, 1989). Laboratory investigations have shown several abnormalities within fibromyalgia patients, but these have not been consistent and still do not explain the etiology of this syndrome. Some of these abnormalities include low cerebral spinal fluid (CSF) levels of serotonin, norepinephrine and tryptophan, EEG abnormalities, low levels of adenosine triphosphate (ATP), elevated levels of CSF substance P, abnormally low exercising muscle blood flow, low natural killer cell activity, and accelerated bone metabolism (Goldenberg, 1999; Thorson, 1994).

Fibromyalgia patients' sleep disturbances are an area that has received some attention as a possible etiology for the syndrome. Studies have repeatedly found that sleep disturbances are found in approximately 75-95% of fibromyalgia patients (Anch et al., 1991; Goldenberg, 1989). The most common sleep disturbance in fibromyalgia involves the intrusion of alpha brain waves into deep delta sleep (Moldofsky, Scarisbrick, England, & Smythe, 1975; Moldofsky & Scarisbrick, 1976; Thorson, 1994). Moldofsky & Scarisbrick (1976) were also able to induce fibromyalgia-like symptoms of

musculoskeletal pain in healthy volunteers by disrupting delta sleep. Bennet (1989) suggests that sleep disturbances could lead to fibromyalgia symptoms through a cycle initiated by the sleep disturbances, rather than some direct biological process. In his formulation slow wave sleep disturbances lead to fatigue, which leads to inactivity, which in turn leads to unfit muscles. These unfit muscles are then more prone to microtrauma, which leads to increased pain, which causes increased disruptions of delta wave sleep (Bennet, 1989). However, these sleep disturbances cannot be the sole causative factor in fibromyalgia as not all fibromyalgia patients experience them. In addition, many fibromyalgia patients report that they experienced musculoskeletal pain before the onset of sleep disturbances.

The connection between fibromyalgia and depression is another area that has received some scrutiny as a possible explanation of the etiology of fibromyalgia. There have been some who have suggested that fibromyalgia could be a symptom of depression, and while there is some evidence to support this hypothesis, there is also evidence to refute it (Goldenberg, 1989). Hudson & Pope (1989) suggest that possibly fibromyalgia is a form of "affective spectrum disorder" in which both depression and fibromyalgia are caused by the same underlying physiologic abnormality, however this proposed underlying process has yet to be discovered. Due to its known association with depression, serotonin levels have also been examined in fibromyalgia patients, but with mixed results. Some studies have found fibromyalgia patients to have lower levels of serotonin in the bloodstream, but others have found little differences between fibromyalgia patients and healthy controls (Russell, Fletcher, Michalek, McBroom, & Hester, 1991).

There simply is no evidence for a single causal factor in fibromyalgia, while there appear to be many factors that contribute to some degree or another. Self-reports have indicated that the onset of fibromyalgia can be precipitated by physical trauma, emotional trauma, or in some cases even viral infections (Goldenberg, 1999). Some studies have found there to be a link to Lyme disease, with 10% to 25% of patients with established Lyme disease developing fibromyalgia that persists for years after the successful treatment of the Lyme disease (Goldenberg, 1999). Other inquiries have found the development of fibromyalgia in 21% of people with cervical spine injuries (Goldenberg, 1999). While research has given us many clues and hints, the theory has yet to be developed that ties them all together into something that makes predictive sense.

While these proposed etiologies could account for some cases of fibromyalgia, none of them can account for all cases. Not all fibromyalgia patients have sleep disturbances, and not all fibromyalgia patients evidence symptoms of depression. Due to the lack of a single etiologic factor, Goldenberg (1999) suggested that fibromyalgia is best thought of as a disorder of pain perception. He also suggested that this disorder of pain perception in all likelihood involved neurohormonal dysregulation (Goldenberg, 1999).

Psychological Features of Fibromyalgia

There are more factors contributing to the dismissal of fibromyalgia as psychogenic than merely the absence of objective physical explanations for patients' complaints. Fibromyalgia patients tend to exhibit a greater amount of psychological symptoms than normal controls, but there does not seem to be a predominant personality type within the fibromyalgia population (Goldenberg, 1999). In particular, fibromyalgia patients tend to

both report and exhibit the symptoms of depression indicative of a depressive disorder (Goldenberg, 1999).

The connection between fibromyalgia and depression has been examined by a number of researchers to date. General estimates of depression among fibromyalgia patients tend to range between 25% and 50% (Hubbard, 1999). The highest estimate found was from a study in which six out of seven fibromyalgia patients were reported to have a current or previous history of depression (Tariot, Yocum, & Kalin, 1986). Another study found 71% of fibromyalgia patients to have a history of major depression, as opposed to 31% of arthritis patients (Hudson et al., 1985). Kirmayer, Robbins, & Kapusta (1988) found 20% of their patients had experienced an episode of major depression at some time, but in most cases the depression had occurred before the onset of fibromyalgia symptoms. Families of fibromyalgia patients have also been shown to have a greater history of depression than families of rheumatoid arthritis patients or healthy controls (Goldenberg, 1989; Hudson et al., 1985).

While examining fibromyalgia symptoms to determine diagnostic criteria, the American College of Rheumatology also found it to be associated with multiple psychological symptoms. This study's 293 fibromyalgia patients were given psychiatric clinical interviews, and 22% were found to meet DSM-III-R criteria for current major depression (Wolfe et al., 1990). Of these patients with current major depression, 27% had a lifetime history of major depression and 28% met the criteria for a lifetime history of panic disorders (Wolfe et al., 1990).

While there clearly seems to be some relationship between fibromyalgia and depression, the nature of this relationship is still not really understood. Goldenberg

(1989) points out that there are three predominant explanations of this relationship, but two out of the three have evidence to both support and refute them. The third is a matter of theoretical conjecture at this time, and it remains to be seen whether or not it will be found to be credible.

The first explanation is that fibromyalgia is actually a symptom of depression. This is supported by the positive results of treating fibromyalgia patients with antidepressants, fibromyalgia patients' family histories of depression, and the finding that there tends to be a history of depression predating the onset of fibromyalgia. However, depression is not seen in all fibromyalgia patients, and the antidepressant response could be secondary to improving the quality of sleep (Goldenberg, 1989).

The second explanation is that the depression commonly associated with fibromyalgia is in fact caused by the symptoms of fibromyalgia. It makes intuitive sense that people who experience chronic pain with an unknown etiology and an uncertain prognosis would tend to be depressed. However, there is evidence to refute this hypothesis as well. Fibromyalgia patients' history of depression usually predates their fibromyalgia symptoms, and their family histories of depression have been documented as well (Goldenberg, 1989).

The third hypothesis is that there is an underlying pathophysiologic abnormality common to both fibromyalgia and depression. Hudson and Pope (1989) suggest that fibromyalgia is a form of "affective spectrum disorder". They propose that there is an underlying affective process that can manifest as a number of different problems. It is suggested that fibromyalgia, major depression, bulimia, panic disorder, obsessive compulsive disorder, attention deficit disorder with hyperactivity, cataplexy, migraines

and irritable bowel syndrome could all be manifestations of an "affective spectrum disorder" (Hudson & Pope, 1989).

Depression is not the only area in which fibromyalgia patients seem to exhibit elevated levels of psychopathology. As with other areas of inquiry into fibromyalgia, the results concerning the relation between fibromyalgia and psychological symptoms have been mixed and inconclusive. One study found no significant differences in psychological symptoms between fibromyalgia patients and controls (Clark, Campbell, Forehand, Tindall, & Bennett, 1985). However, both the fibromyalgia patients and the controls from this study were taken from a general medical outpatient population, and 41% of the controls reported experiencing moderate to severe pain (Clarke et al., 1985).

Payne et al. (1982) found that fibromyalgia patients' MMPI profiles were higher and more variable than MMPI profiles from patients with mixed arthritis and rheumatoid arthritis. However, only the hysteria and hypochondriacal scales were in the clinical range, while there were no significant differences found on the depression subscale. Ahles, Khan, Yunis, Spiegel, & Masi (1991) also found fibromyalgia patients to have higher MMPI profiles than rheumatoid arthritis patients and normal controls. While still within the normal range, they found fibromyalgia patients to score highest on the subscales for hypochondriasis, depression and hysteria (Ahles et al., 1991). Another study examining fibromyalgia patients' MMPI profiles also found them to score highest on the hypochondriasis and hysteria scales, but not significantly higher on the depression subscale (Wolfe, Cathey, & Kleinhenksel, 1984).

Care must be taken when interpreting the results from these MMPI profiles. Smythe (1984) suggested that there is a 40% probability of labeling a chronic pain patient as

"neurotic" based on elevated scores on the depression, hysteria and hypochondriasis scales of the MMPI. However, the MMPI does not differentiate between organic and non-organic somatic symptoms. Any chronic pain patient will be likely to present elevated scores on these subscales based solely on the physical pain they are experiencing (Hubbard, 1999). As the MMPI does not differentiate between physical and psychological symptoms, fibromyalgia patients' elevated scores could very well be related to physical pain rather than psychopathology (Birnie, Knipping, Van Rijswijk, De Blecourt, & De Voogd, 1991; Goldenberg, 1989; Hudson et al., 1985).

Psychological Features of Patients with Chronic Pain

Many of the descriptions of fibromyalgia patients are similar to descriptions of other patients with chronic pain and the two groups share many psychological features (Birnie et al., 1991). Many chronic pain patients have undergone a series of diagnostic procedures, therapies, surgeries and countless medical and paramedical visits with little or no lasting benefits. In addition to complaints about pain, these patients often have to contend with disturbed psychosocial functioning, diminished physical functioning, fear of damaging the body through physical activity, dependency on medication, dependency on various forms of physiotherapy, an increased sense of helplessness and hopelessness, withdrawal from psychosocial activities, emotional conflicts, negative emotional and affective changes, and receiving or attempting to receive assistance from social service agencies (Birnie et al., 1991).

These characteristics, however, describe the psychological condition of both fibromyalgia patients and patients with other chronic pain conditions (Birnie et al., 1991).

These findings serve to support the suggestion that the psychological symptoms of fibromyalgia could be secondary to the pain or disability associated with it (Hudson et al., 1985). Chronic pain is likely to exacerbate the stress and adverse psychological reactions in any disorder, and fibromyalgia is certainly no exception.

Block (1993) suggests a working framework for understanding the interaction between chronic pain, stress, muscle fatigue and sleep. In this model persistent pain causes muscle tension and loss of sleep, which increases levels of psychological stress. Loss of sleep and muscle tension lead to muscle fatigue, which in turn leads to a continuation of persistent pain. This self-perpetuating pain cycle is exacerbated by the psychological stress inherent to it and any co-existing psychological or physical stressors (Block, 1993). This cycle of chronic pain is supported in fibromyalgia by the large proportion of patients who recall that their symptoms started with pain and stiffness, followed by sleep disturbances, then subsequently followed by hyper-irritability and anhedonia (Goldenberg, 1989).

Cognitive Functioning in Fibromyalgia and Related Conditions

While complaints of cognitive impairments are common with fibromyalgia patients, few studies to date have specifically examined their neuropsychological status (Hubbard, 1999; Landro et al., 1997; Kaplan et al., 1992; Sletvold et al., 1995). Possibly examining the neuropsychological status of other chronic pain populations can be helpful in shedding some light on the cognitive impairments so frequently reported by fibromyalgia patients (Dufton, 1989; Hubbard, 1999; Kaplan et al., 1992; Krupp et al., 1994; Sandman et al., 1993; Schmaling et al., 1994). Many of these studies seem to indicate that

depression and other manifestations of emotional distress could be intimately intertwined with self-reported problems with concentration and memory (Hubbard, 1999).

Landro et al. (1996) examined memory functioning in 25 fibromyalgia patients, 18 healthy controls and 22 patients with current major depression. They found that both the fibromyalgia patients and the patients with major depression showed significant impairment on long-term memory tasks requiring effortful processing. However, when the depressive status of the fibromyalgia patients was accounted for, only those with a history of major depressive disorder showed significant memory impairment as compared to the healthy controls.

Hubbard (1999) used a battery of objective neuropsychological measures and subjective measures to examine the cognitive and psychological functioning of 34 fibromyalgia patients. Although all of the subjects complained of problems with concentration and memory, objective measures found them to be within the average range of functioning. However, scores on the Brown Adult Attention-Deficit Disorder Scale did correlate significantly with the subjects' self-reported complaints of memory impairments. The Brown Adult ADD scale also correlated significantly with the subjects' elevated scores of depression and psychopathology on the Symptom Checklist-90-R and the Centre for Epidemiological Studies in Depression Scale, but did not correlate significantly with objective memory tasks.

Another study examined complaints of memory problems in patients with fibromyalgia, lyme encephalopathy and major depression, but found that only the group with lyme encephalopathy showed quantifiable memory deficits on objective measures (Kaplan et al., 1992). They found no significant differences between fibromyalgia

patients and depressives on objective memory tasks, and concluded that both groups' self-reported memory impairments were primarily due to psychological distress.

Sletvold et al. (1995) found objective evidence of psychomotor slowing in fibromyalgia patients both with and without a lifetime history of major depression. Their results indicated that those fibromyalgia patients who had never met the DSM-III-R criteria for depression were significantly different from healthy controls in their ability to process information and in the speed at which they were able to hold and manipulate information in working memory.

Krupp et al. (1994) examined cognitive impairments and depression in patients with chronic fatigue syndrome (CFS) and multiple sclerosis (MS). They found that CFS patients had more depressive symptoms than MS patients, but the MS patients showed higher levels of cognitive impairment. However, they did find that the CFS patients performed worse than healthy controls on tests of visuomotor search and on the Logical Memory Test of the Wechsler Memory Scale-Revised. They concluded that the CFS patients' poor performance on the Logical Memory Test appeared to be related to depression, but their visuomotor deficits had no relation to depression at all.

Another study comparing cognitive performance between CFS patients, depressed patients, and healthy controls found the CFS group to have significant deficits in memory consolidation, higher vulnerability to interference, and slow or uncertain decision making (Sandman et al., 1993). However, a similar study conducted by Schmaling et al. (1994) found the cognitive functioning of CFS patients to be within the normal range, yet it did not differ significantly from the cognitive functioning of depressed patients.

Grigsby, Rosenberg, & Busenbark (1995) compared 19 patients with chronic pain to 25 patients with minor head trauma on information processing and motor subtests of the Human Performance Measurement System. They found that both groups performed below the normal range on all measures except visual digit span, and the chronic pain patients actually scored lower than the head trauma patients on 2 out of 6 measures of central processing speed.

Jamison, Sbrocco, & Parris (1988) also examined cognitive impairment in a chronic pain population. They divided 363 chronic pain patients into two groups based on how much self-reported difficulty they experienced with concentration and memory. Both groups were then given physician ratings of their depression and anxiety, a pain evaluation questionnaire, and an SCL-90. Their results indicated that concentration and memory problems were significantly related to ratings of depression and anxiety, poor family support, and the pain's interference with sexual and recreational activities. They found that concentration and memory impairment were not significantly related to pain intensity, pain duration or demographic factors.

Cognitive Functioning in Depressed Patients

A relationship between depression and cognitive impairment has been shown to exist repeatedly in the research literature (Danion et al., 1991; Landro et al., 1997; Schmaling et al., 1994; Sletvold et al., 1994). Much of this research indicates that depressed patients' cognitive impairments tend to revolve around difficulties with effort-demanding cognitive processes (Danion et al., 1991). Depressed patients tend to have difficulties learning information that requires sustained effort and the use of elaborate cognitive

operations (Danion et al., 1991). Depressed patients have also been shown to have deficits in information processing and in episodic and semantic memory (Landro et al., 1997; Sletvold et al., 1995).

There is research to support some investigators' allegations that psychiatric patients' complaints of memory impairments do not always correlate with objective measures of memory function. A study conducted by Wells (1979) found that depressed patients were much more likely to complain about cognitive deficits than were demented patients, although the demented patients in fact showed higher objective levels of impairment. However, numerous studies using objective measures have found that cognitive impairments in depressed patients do exist.

Finlayson and Bird's (1991) review of studies concerning neuropsychological functioning in depressed patients suggests that most of this population's difficulties involve visuo-spatial abilities, attention and concentration. Research conducted by Martin, Oren & Boone (1991) indicates that depressed patients suffer from deficiencies of information processing capacity and higher order conceptual reasoning. Other studies have found depressed patients to have deficiencies in intellectual speed, mental speed, and psychomotor speed (Blackburn, 1975; Miller, 1975). Miller (1975) suggested that the reduced intellectual and psychomotor speed of depressives was not due specifically to depression, but rather, were an index of the severity of any psychiatric illness. This hypothesis is supported by research finding that bipolar depressives had higher levels of cognitive impairment than unipolar depressives (Blackburn, 1975).

Patients with major depression have also been found to have impairments on a number of verbal memory tasks, although these deficiencies do not resemble the storage and

coding deficits found in amnesiacs (Danion et al., 1991; Hart, Kwentus, Taylor, & Harkins, 1987; King, Caine, Conwell, & Cox, 1991; Kopelman, 1986; Weingartner, Cohen, Murphy, Martello, & Gerdt, 1981; Weingartner & Silberman, 1982; Wolfe, Granholm, Butters, Saunders, & Janowsky, 1987). While depressed patients perform below the normal range on verbal recall tasks, they tend to perform within the normal range on verbal recall tasks using recognition measures (Dunbar & Lishman, 1984; Frith et al., 1983; King et al., 1991; Silberman, Weingartner, Laraia, Byrnes, & Post, 1983; Wolfe et al., 1987).

While there are many components of memory and information processing systems that could contribute to the problem, in general depressed patients seem to have difficulties learning and remembering information that requires elaborate cognitive processing or sustained effort (Danion et al., 1991; Landro, 1997). Patients with major depression are also much more likely to have impaired memory performance on free-recall tests than on recognition tests and delayed-recall tests (Danion et al., 1991).

Chronic Fatigue Syndrome (CFS)

No discussion of fibromyalgia would be complete without mentioning the relation to chronic fatigue syndrome (CFS). The two conditions are commonly comorbid, and there is quite a bit of symptom overlap between the two when looked at independently. Just as with fibromyalgia, CFS patients commonly complain of sleep disturbances, generalized achiness, headaches, depression, anxiety, parasthesias and post-exertion intensification of symptoms (Demitrack, 1998). Patients with CFS have even been shown to have tender points similar to those found in fibromyalgia patients (Demitrack, 1998). The main

difference between the two is that fibromyalgia patients present chronic diffuse pain as their main symptom, while CFS patients present chronic fatigue as their primary symptom. However, there is considerable overlap in secondary symptoms between the two.

More than 90% of CFS patients also report having generalized cognitive impairments that tend to revolve around memory deficits and problems with attention and concentration (Krupp et al., 1994). Within this group, the severity of cognitive dysfunction remains unknown and it remains unclear whether CFS patients experience cognitive deficits due primarily to CFS, or whether neuropsychological complaints are due to associated factors such as depression (Krupp et al., 1994).

Although there has been a large school of thought that attributed the cause of CFS to chronic Epstein-Barr virus infection, laboratory findings have not universally supported this (Goldenberg, 1999). As with fibromyalgia, CFS has come to be diagnosed based solely upon symptoms and clinical signs. In 1988 the Center for Disease Control laid down its diagnostic criteria for chronic fatigue syndrome. In order to meet these criteria the patient must fulfill both of the major criteria and at least 8 out of 11 of the symptom criteria, or both of the major criteria, 6 or more of the 11 symptom criteria, and 2 or more of the symptoms listed under physical criteria (Hubbard, 2000). These criteria are as follows:

Major criteria

1. The onset of persistent or relapsing, debilitating fatigue that does not resolve with bed rest and that reduces activity levels by 50% for at least 6 months

2. Other plausible disorders of fatigue are ruled out by evaluation based on history, examination, and laboratory findings

Symptom criteria

1. Neurospsychological complaints
2. Low-grade fever
3. Sore throat
4. Painful nodes
5. Unexplained generalized muscle weakness
6. Headaches
7. Myalgia
8. Prolonged fatigue after levels of exercise that would have been easily tolerated before onset
9. Unexplained generalized muscle stiffness
10. Sleep disturbance
11. Main symptom complex developing over a few hours to a few days

Physical criteria

1. Low grade fever between 37.6-38.6 Celsius
2. Non-exudative pharyngitis
3. Palpable anterior or posterior cervical or axillary lymph nodes (Hubbard, 2000).

While the relation between the two is still not clearly understood, a relationship definitely seems to exist between chronic fatigue syndrome and fibromyalgia. Based on standard classification criteria, 50% to 70% of fibromyalgia patients also have a current

or past diagnosis with chronic fatigue syndrome (Goldenberg, 1999). The two disorders also have similar symptoms, chronicity, demographics and therapies (Goldenberg, 1999). To date, both also have unknown etiologies and are considered incurable, although they are treatable to a degree. However, there is still no treatment that has consistent results across the population, and fibromyalgia patients often spend large amounts of money trying various treatments that have no effect.

Effects of Sympathetic Arousal on Memory

Any pain response will in part consist of arousal of the sympathetic nervous system. As the body prepares for fight or flight in response to the pain stimulus, it releases higher levels of corticotropin and other various neurohormones. In fibromyalgia patients, research has shown there to be an exaggerated corticotropin response to corticotropin-releasing hormone and other variable disturbances of the sympathetic nervous system (Goldenberg, 1999). There is also evidence of a sympathetic-parasympathetic imbalance occurring in fibromyalgia, which is considered to be related to neurally mediated hypotension (Goldenberg, 1999).

Research has repeatedly indicated that the heightened levels of glucocorticoids seen in the stress response can result in damage to the hippocampus, which is a brain area involved in learning and memory (Bremner & Narayan, 1998; McEwen, 1992). Cognitive impairments related to heightened levels of glucocorticoids have been examined extensively in posttraumatic stress disorder (PTSD). These studies have consistently found that the persistent sympathetic response in PTSD leads to significant reductions in hippocampal volume, as well as significant deficiencies of memory function

(Bremner et al., 1997; Bremner & Narayan, 1998; Golier & Yehuda, 1998; Jenkins, Langlais, Delis, & Cohen, 1998; Sapolsky, 1999; Southwick, Morgan, Nicolaou, & Charney, 1997).

It is possible that the memory problems experienced by fibromyalgia patients are similar in nature to those experienced by PTSD patients. While PTSD patients experience chronic psychogenic stress for extended periods of time, fibromyalgia patients experience chronic pain and the stress associated with it. However, the nervous system anomalies sometimes found in fibromyalgia serve to complicate this issue. Some research has found that fibromyalgia patients actually have a reduced level of cortisol secretion (Bombadier & Buchwald, 1996). However, this study hypothesizes that the pain and stress of fibromyalgia leads to elevated levels of glucocorticoids for an extended period of time. If this is indeed the case, fibromyalgia patients should show a greater level of memory impairment with a longer duration of symptoms.

Treatment

Quite a few treatment modalities have been tried with fibromyalgia, with varying degrees of success. While no treatment has been found to "cure" fibromyalgia, several have been found to help alleviate some of the symptoms associated with it. Research seems to indicate that a multimodal approach to treating fibromyalgia is the best approach (Baumstark & Buckelew, 1992; Hawley & Cathey, 1985; Masi & Yunus, 1990; Nies, 1992; Secord, 1998). Some of the main components suggested for this multimodal approach include patient education, basic sleep hygiene, stretching and aerobic exercise,

medication to help improve the quality of sleep, and psychological intervention to help manage stress and coping strategies (Secord, 1998).

Treatment with medication has led to meaningful improvement in 30% to 50% of patients, most often with the use of serotonin reuptake inhibiting anti-depressants (Goldenberg, 1999). These anti-depressants work to alleviate the sleep disturbances so commonly reported by fibromyalgia patients, which also helps reduce the severity of pain and other symptoms (Secord, 1998). Some of the other medication treatments reported to cause modest improvements include muscle relaxers, analgesics and tranquilizers (Goldenberg, 1999; Secord, 1998). However, not all reports claim there to be beneficial effects from these medications. There are also a number of medications or medical treatments that have been used which have yielded no positive results. Some of these include non-steroidal anti-inflammatory drugs and corticosteroids, magnesium and malic acid, full spectrum light exposure, S-adenosyl-L-methionine, and topical hydrochloride in sphenopalatine blocks (Goldenberg, 1999).

Non-medical treatments have been tried with varying success rates as well. Cognitive-behavioral treatments have been somewhat helpful in alleviating stress and promoting appropriate beliefs and coping strategies (Goldenberg, 1999; Secord, 1998). As stress commonly exacerbates the symptoms of fibromyalgia, alleviating stress can also help reduce the severity of pain symptoms. Quite a few other treatments have also been used for fibromyalgia patients, including cardiovascular fitness training, regional sympathetic blocks, electromyographic biofeedback, hypnotherapy, and electroacupuncture (Goldenberg, 1999). However, the most effective treatment remains a

multi-modal approach incorporating medication, stress management and lifestyle changes designed to improve fitness, relaxation therapy and recreation (Bennett, 1996).

Prognosis, Outcome, and Disability

While the symptoms of fibromyalgia tend to remain fairly stable over time, it is a chronic condition with relatively persistent pain, which can often be rather debilitating (Goldenberg, 1999; Hudson et al., 1985). Studies of the long-term effects of fibromyalgia indicate that it's symptoms influence all aspects of daily life and have a significant impact on work, family life, and leisure activities (Henriksson, 1994). A longitudinal study of 1604 fibromyalgia patients found that 27% of them received at least one form of social security or other disability payments (Goldenberg, 1999). It is not at all uncommon for fibromyalgia patients to report significant work loss, need for work modifications, and work disability (Smythe et al., 1993). Patients who had concurrent diagnoses of fibromyalgia and chronic fatigue syndrome have been found to have particularly high rates of unemployment, with some estimates reaching as high as 51% (Goldenberg, 1999).

A study conducted in 1996 found the mean yearly per patient costs for fibromyalgia to be similar to costs for the treatment of osteoarthritis, about $2274 annually (Goldenberg, 1999). Fibromyalgia patients are admitted to hospitals 3.4 times more often than age-matched controls, and they are 3.5 times more likely to undergo surgery (Bruckle et al., 1992). Disability costs attributable to fibromyalgia in America add up to millions of dollars each year. In Canada, fibromyalgia was found to be responsible for 9% of the disability payments at a major Canadian life insurance company, which translated into

approximately $200,000,000 per year (Smythe et al., 1993). In addition to the other problems faced by fibromyalgia patients, their treatment can in many cases be quite expensive.

Summary

Fibromyalgia is a syndrome with chronic pain that affects millions of people worldwide. After having been dismissed as a psychogenic disorder for years, it has come to be widely accepted as having a physiological basis by both physicians and psychologists. Thus far there is no known etiology, and laboratory findings have been inconclusive or contradictory to each other. It is commonly accompanied by symptoms other than just chronic pain, including chronic fatigue, sleep disturbances, depression, muscle stiffness, memory impairment and many others. While many treatments have shown modest improvements in symptom severity, there is no known cure for fibromyalgia. In most cases, fibromyalgia causes major disruptions of normal daily life due to persistent pain, chronic fatigue, depression and deficits of attention and memory.

Although the vast majority of fibromyalgia patients complain of having problems with their memory, very few studies to date have specifically examined this area. The four studies that have looked into fibromyalgia patients' reports of cognitive dysfunction have had inconclusive and often contradictory results (Hubbard, 1999; Kaplan et al., 1985; Landro, 1997; Sletvold et al., 1994). Some of these studies have indicated that depression might account for the cognitive deficits reported by fibromyalgia patients, but this has not been conclusive.

The human body's pain response involves activation of the sympathetic nervous

system, which then releases higher levels of glucocorticoids. Research on persistent stress in posttraumatic stress disorder has shown that elevated levels of glucocorticoids can lead to reductions in the volume of the hippocampus, a structure that is part of the brain's memory system (Bremner et al., 1997; Bremner & Narayan, 1998; Golier & Yehuda, 1998; Jenkins et al., 1998; Sapolsky, 1999; Southwick et al., 1997). While the exact nature of fibromyagia patients' self-reports of memory dysfunction is unclear, perhaps heightened levels of glucocorticoids could contribute to the problem. The persistent pain and stress of fibromyalgia could lead to hippocampal deterioration similar to that found in PTSD. If so, a longer duration of pain and stress should lead to greater levels of memory impairment.

CHAPTER 3

METHODOLOGY

Subject Selection Procedures

The subjects making up the sample of this study will be recruited from fibromyalgia support groups in the Texas towns of Wimberley and Georgetown. All subjects will sign a consent form (see Appendix C), and will only be considered for inclusion in this study if they have fulfilled the diagnostic criteria for fibromyalgia, based on the subjects' self-report of having been diagnosed with fibromyalgia by a rheumatologist or MD.

Instruments Used

Demographic questionnaire

All subjects will complete a questionnaire (see Appendix D) with information concerning age, marital status, children, level of education, date of diagnosis with fibromyalgia, who made the diagnosis (rheumatologist, MD, etc.), which if any prescription medications are currently being taken, how many times per week they engage in any form of exercise, and whether they have ever been diagnosed with depression or any other psychiatric disorder. This questionnaire will also ask if they believe their short-term memory functions as well as it did before the onset of

fibromyalgia. Measures of pain severity, depressive status and working memory function will also be assessed using objective measures.

Measures of pain severity, depressive status and working memory function

All subjects will complete the BDI-II (Beck Depression Inventory – Short form) to screen for current depressive status. The BDI-II is not meant to be used as a diagnostic tool for depression, but rather is a self-report analysis of depressive symptoms (Sundberg, 1987). The test consists of 21 items, on which people are asked to report feelings consistent with their own for the past two weeks. Each of these items assess these feelings on a Likert scale of 0-3, except for items 16 and 18 which are scaled as 0, 1a, 1b, 2a, 2b, 3a and 3b in order to reflect any directional changes in eating and sleeping patterns. Interpretation of scores is based on criterion-referenced procedures using the following interpretive ranges: 0-13 – minimal depression; 14-19 – mild depression; 20-28 – moderate depression; and 29-63 – severe depression (Beck, Brown, & Steer, 1996).

The severity of current pain will be assessed using the Short-form McGill Pain Questionnaire. This test is a self-report measure that lists 15 descriptive words that are meant to describe the pain subjects have experienced for the past 48 hours. Subjects rank each of these words as either none, mild, moderate, or severe. Each of these ranking categories is assigned a numerical value ranging from 0 for none to 3 for severe. These numerical values are summed to provide a quantitative estimate of pain severity over the previous 2 days (Melzack, 1987).

Working memory will be assessed using the working memory subscale of the Wechsler Adult Intelligence Scale. This subscale consists of the digit span, mental arithmetic, and letter-number sequencing subtests. These three subtests are combined to

provide a working memory index, which will be used as the measure of working memory function.

Analysis

As this is an exploratory study, the primary method of analysis will be looking for significant correlations between variables. Scores on the working memory subscale of the WAIS will be compared to population norms using a one sample T-test. Correlation matrices will then be run on all variables to see if performance on the memory task is associated with variance in any of the independent variables. Part and partial correlations may also be run to see if relationships exist between variables with the variance of possible confounds statistically removed.

If a sufficient sample size is obtained, more advanced statistical procedures may be employed. Using multivariate regression analysis, working memory will serve as the dependent variable, with length of time since diagnosis with fibromyalgia serving as the independent variable. Other information will be controlled for including depressive status, amount of medication currently being taken, marital status, children, education, history of psychiatric diagnoses, and whether or not the subject engages in exercise. After statistically removing the variance attributable to other variables, this study hopes to find an impairment of working memory function attributable to the length of time since diagnosis with fibromyalgia.

CHAPTER 4

RESULTS

Demographics

The results of the demographic questionnaire are presented in Table A-1. The subjects' ages ranged from 25 to 87 years old, with the mean age being 49.29. The sample consisted 100% of women, with 61.9% (n = 13) reporting that they are currently married. Only 2 of the subjects had no children, and the average number of children was 2.14. All of the subjects had at least a high school education, and 71.4% (n = 15) had at least 2 years of college.

Only 14.3% (n = 3) of the sample were taking no prescribed medications at all, and on average the subjects were taking around 4 medications a day (M = 3.95). The highest number of medications taken was 13 (n = 2). On average, it had been almost 5 years (M = 4.81) since the subjects had been diagnosed with fibromyalgia, but they had been experiencing chronic pain for 14.71 years on average. In 33.3% (n = 7) of the subjects the diagnosis of fibromyalgia was made by a rheumatologist, 19% (n = 4) were diagnosed by family practitioners, 14.3% (n = 3) were diagnosed by neurologists, 9.52% (n = 2) were diagnosed by internists, and 23.81% (n = 5) did not specify what type of physician made the diagnosis. Of the 21 subjects, 33.3% (n = 7) had never had a psychiatric diagnosis, 57.2% (n = 12) had been diagnosed with depression in the past, and 14.3% (n = 2) had experienced a psychiatric diagnosis other than depression.

Results of Working Memory Index

The results of the Working Memory Index (WMI) percentile rankings from the Wechsler Adult Intelligence Scale indicate that the sample performed significantly lower than the general population $t(20) = -2.53$, $p < .05$. The average score on the WMI was 92.90, which yielded an average percentile ranking of 36.52. With the WMI being a standardized index score, the population mean is set at 100 and the population percentile rank mean is 50. The frequency distribution of WMI scores can be found in Table A-5.

The WMI results did not correlate significantly with any of the other variables. While not statistically significant, the strongest correlation was with scores on the McGill Pain Questionnaire $r = -0.31$, $p > .05$. The correlations were particularly low between WMI scores and the independent variables being examined in this study. The correlation between WMI scores and the length of time since diagnosis was $r = -0.024$, $p > .05$ and the correlation between WMI scores and the length of time that they had experienced pain (see Table A-2) was $r = 0.139$, $p > .05$.

Results of BDI-II

The subjects in this study ranged from being not depressed at all to being severely depressed (see Table A-3). Of them 19.05% ($n = 4$) scored in the not depressed range, 23.81% ($n = 5$) scored as mildly depressed, 28.57% ($n = 6$) were moderately depressed, and 28.57% ($n = 6$) scored as having severe depression. As a whole, the sample scored on the lower end of the range for moderate depression ($M = 22.57$), which ranges from 20 to 28.

While scores on the BDI did not correlate significantly with the dependent measure, WMI index ($r = -0.133$, $p > .05$), a few other correlations did come out that were both

significant and interesting. A significant negative correlation was found between BDI scores and age ($r = -0.65$, $p < .01$). After creating a standardized residual of BDI scores with the variance of age removed, there was no significant correlation between the residual and scores on the WMI index ($r = -0.29$, $p > .05$). There was also a significant correlation between scores on the BDI and scores on the McGill Pain Questionnaire ($r = 0.52$, $p < .05$). For scores on the McGill Pain Questionnaire, see Table A-4. There was also a relatively high, albeit non-significant, negative correlation between BDI scores and the amount of time that they had been experiencing pain ($r = -0.42$, $p > .05$).

Other observed relationships

A significant correlation was found between the subjects' past history of psychiatric diagnoses (most notably depression) and the number of medications being currently taken ($r = 0.53$, $p < .05$). While not significant, a relatively high negative correlation was found between the amount of time the subjects had experienced pain and their scores on the McGill Pain Questionnaire ($r = -0.37$, $p > .05$).

All of these results are somewhat limited due to the small sample size of this study. With the number of variables involved, a much larger sample size would be needed to perform a multiple regression analysis with the variance of possible confounds removed. Due to the number of possible confounds, this approach would be needed to learn whether or not the independent variables being scrutinized do in fact have an effect on working memory. Even with the variables that did have a statistically significant relationship with each other or standardized norms, the ability to generalize these results is limited due to the small sample size.

CHAPTER 5

DISCUSSION

The results of this study dictate the acceptance of the null hypothesis. No relationship was found between the duration of pain fibromyalgia patients had experienced and the functioning of working memory. However, the results did indicate that the working memory of the subjects was significantly lower than that of the general population. This is a notable finding as there is still considerable debate as to whether or not fibromyalgia patients' self-reported complaints of memory problems are in fact valid. While fibromyalgia patients' memory complaints have been examined from a number of different angles, this study suggests that perhaps closer scrutiny should be given to the functioning of working memory in particular. Unfortunately, this study does not shed any light onto what the possible causes of these memory deficits could be.

The results of prior studies examining fibromyalgia and memory have tended to yield results that were either inconclusive or contradictory to other studies (Hubbard, 1999; Kaplan et al., 1992; Landro et al., 1997; Sletvold et al., 1995). The results of this study add one more to the ranks of those studies asserting the validity of fibromyalgia patients' self-reported complaints of memory dysfunction. This study also suggests that perhaps closer scrutiny should be given to the functioning of working memory in particular.

There are a number of limitations of this study that could contribute to the lack of findings in regards to relationships between working memory and other variables. The

first and largest limitation is the small sample size of this study. With the number of possible confounding variables, a relatively large sample size is going to be needed in order to extract the variance of different confounds. Variables that might contribute to memory deficits in fibromyalgia patients could include age, severity of pain, duration of pain, depression, medications, and level of mental activity, as well as other possible factors that have not been identified at this time. A larger sample size is also needed in order to find significance in what might be relatively weak relationships.

Another limitation of this study is the source of subjects. In this study subjects were recruited solely from fibromyalgia support groups. The demographics of fibromyalgia patients attending support groups might not be representative of the larger population of fibromyalgia patients in general. The population of fibromyalgia patients attending support groups could differ from the population of fibromyalgia patients in any of a number of ways. There could possibly be differences in behaviors related to seeking assistance from others, differences in levels of social support, and differences in the severity of current pain or depression. One notable characteristic of this sample is that they appear to have a relatively high level of education, with the average level of education for the sample being two and a half years of college.

This study certainly helps to identify areas to focus on in future research of fibromyalgia and memory. The most important finding of this research is that a statistically significant difference in the functioning of working memory does exist between fibromyalgia patients and the general public. What could be causing this memory deficit is a subject that is still open to speculation. It could be attributable to the biological processes of pain, biological processes of pain over a period of time, the

effects of the depression, the effects of some unrecognized process of fibromyalgia, or a cumulative effect of multiple factors. Studies examining the functioning of memory in other acute and chronic pain conditions could possibly rule out some of these possibilities and studies similar to this one could help identify the specific factors related to memory deficits in fibromyalgia patients.

If subsequent studies find these self-reported complaints of memory impairment to be in fact valid, there are a number of implications for the treatment and management of fibromyalgia. However, some of the possible changes to treatment would be dependent upon learning more about which factors contribute the most to these memory impairments. If they are found to be related to depression, a more active treatment of depression in fibromyalgia patients could help alleviate these memory problems. If the factors contributing to these deficits had more to do with the biological processes of pain, perhaps some pharmaceutical approach could be employed. This would also have profound implications in the treatment of other conditions involving acute or chronic pain. However, it is quite likely that a number of factors could contribute to these memory deficits, necessitating a more multi-disciplinary approach.

The presence of memory deficits in fibromyalgia patients also brings up some issues related to the safety and quality of life of fibromyalgia patients. With some of the medications commonly prescribed for fibromyalgia patients, perhaps more care should be taken to avoid the possibility of accidental overdoses based on forgetting whether or not the medication had already been taken. There are a number of behavioral approaches that could help alleviate this possible problem, such as using some sort of organizer or pillbox to keep track of when the medication has been taken. Fibromyalgia patients might also

be encouraged to use mediums such as a daily planner in order to minimize the effects of impaired memory on their day-to-day life.

If this study were to be replicated, there are a number of ways in which it could be improved upon. First and foremost would be obtaining a larger sample size. With the number of possible confounding variables that exist, a sample two or three times the size of this one would be necessary to properly control for extraneous variance. It could also be advantageous to use a matched control group rather than relying solely upon within group variance. There are also a few other variables that should be examined, such as levels of mental and physical activity.

In conclusion, there appears to be some validity to fibromyalgia patients' complaints of having problems with their memory. While the nature of these memory complaints and their etiology is still unknown, this is an area that should certainly be researched further. There are more implications to impaired memory in fibromyalgia patients than just validation of their subjective experiences. The recognition of memory deficits existing in fibromyalgia patients could point to possible changes that could be made in the treatment of fibromyalgia. A better understanding of the etiology of this memory impairment could lead to much better methods of controlling and managing it, as well. If more light can be shed on what aspect of fibromyalgia contributes most to this memory impairment, perhaps one day steps can be taken to avoid this impairment of memory altogether.

Appendix A

Demographic Information

Table A-1

Age & Sex	Value	N
Female	100%	21
Age	49.29 (mean)	
Marital Status		**N**
Currently married	61.90%	13
Single, divorced, or widowed	38.10%	8
Diagnosed By:	**% of Sample**	
Rheumatologist	33.33	7
Family Practitioner	23.81	5
Neurologist	14.29	3
Other or type of MD not specified	28.57	6
Number of children	**% of Sample**	**Frequency**
0	9.52	2
1	19.05	4
2	38.10	8
3	19.05	4
4	9.52	2
5	4.76	1

Demographic Information (continued)

Table A-1 (continued)

Number of Medications taken	**% of Sample**	Frequency
0	14.29	3
1	19.05	4
2	9.52	2
3	9.52	2
4	4.76	1
5	19.05	4
6	9.52	2
7	4.76	1
13	9.52	2
Education		
Completed High School	28.57	6
Some College	38.10	8
Completed College	19.05	4
Master's Degree	14.29	3

Time with Pain

Table A-2

Time since diagnosis	**% of Sample**	N
One year or less	19.05	4
2-5 years	52.38	11
6-10 years	19.05	4
More than 10 years	9.52	2
Time since onset of pain		
One year or less	4.76	1
2-5 years	23.81	5
6-10 years	19.05	4
11-15 years	23.91	5
16-25 years	9.52	2
More than 25 years	19.05	4

Results of BDI-II Short Form

Table A-3

Level of Depression	**% of Sample**	n
Not depressed (0-13)	19.05	4
Mild depression (14-19)	23.81	5
Moderate depression (20-28)	28.57	6
Severe depression (29-63)	28.57	6

Results of McGill Pain Questionnaire

Table A-4

Score	% of Sample	n
7	4.76	1
11	4.76	1
15	4.76	1
16	4.76	1
18	4.76	1
19	14.29	3
20	4.76	1
23	4.76	1
24	4.76	1
26	4.76	1
27	9.52	2
28	9.52	2
32	4.76	1
33	4.76	1
36	4.76	1
37	4.76	1
38	4.76	1

Results of Working Memory Index

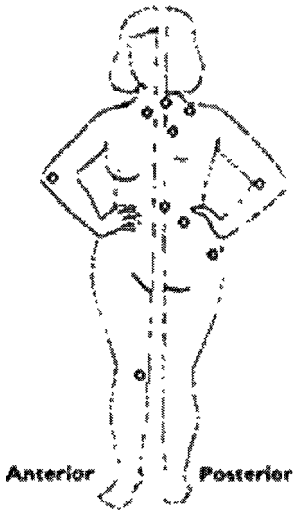
Table A-5

Score	Percentile Rank	% of Sample	n
71	3	4.76	1
73	4	9.52	2
75	5	4.76	1
84	14	9.52	2
88	21	4.76	1
92	30	9.52	2
94	34	9.52	2
97	42	14.29	3
99	47	4.76	1
102	55	4.76	1
104	61	4.76	1
106	66	14.29	3
117	87	4.76	1

Appendix B

Figure B-1. The locations of the nine bilateral pairs of tender points. These are located where the back of the head meets the neck, halfway down the side of the neck, the middle of the trapezius, halfway down the inside border of the shoulder blade, between the 2nd and 3rd rib about one inch from the breastbone, just past the outer prominence of the elbow, just behind the outer edge of the hip, in the mid-portion of the buttocks, and just above the inside of the knee.

Figure B1



Appendix C

Consent Form

Chronic pain and short-term memory:

Is a longer duration of chronic pain in fibromyalgia patients associated with a greater impairment of short-term memory function?

You are invited to participate in a study of the effects of chronic pain on short-term memory in fibromyalgia patients. I am a graduate student in the psychology department at Southwest Texas State University at San Marcos. This study is being conducted as the thesis of a Masters degree in health psychology. This study is designed to discover if the duration of experiencing chronic pain has an effect on short-term memory in fibromyalgia patients.

You were selected as a possible participant in this study based on having been diagnosed with fibromyalgia. If you decide to participate, you will be given several short questionnaires and asked to complete a brief test of short-term memory function. This test includes repeating series of numbers and letters, as well as a small portion on mental arithmetic. I will only need to meet with you once, and the entire process should take no more than about thirty minutes. All of this information is gathered to rule out factors that could affect short-term memory function other than the duration of chronic pain.

Any information that is obtained in connection with this study and that can be identified with you will remain 100% confidential and would only be disclosed with your written permission.

Your decision whether or not to participate will in no way prejudice your future relations with Southwest Texas State University. If you decide to participate, you are free to

discontinue participation at any time. If you have any questions, please feel free to ask me either in person or at 512-836-6000. If you have any additional questions or concerns, please feel free to contact Dr. Randall Osborne at 512-245-2526.

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. If you find the process stressful in any way, you are welcome to withdraw from the study, or reschedule for a later date. Should you choose to discontinue participation in this study after signing this form, you may withdraw at any time without prejudice.

You will be offered a copy of this form to keep if you so choose.

Signature of participant_____ Date___/___/___

Signature of investigator_____ Date___/___/___

Appendix D

Research Subject Information Questionnaire

1. Date of birth: _____
2. Date of diagnosis with fibromyalgia: _____
3. How long did you experience the pain associate with fibromyalgia prior to diagnosis? _____
4. Who made the diagnosis of fibromyalgia? _____
5. Would you say your short-term memory functions as well today as it did before the onset of fibromyalgia? (yes/no)
6. What prescribed medications are you currently taking, and how much do you take per day or week?

   _____

   _____

7. How many times per week on average do you exercise, and what type of exercise is it?

   _____

   _____

8. What is your marital status? _____
9. Do you have children, and if so how many? _____
10. How many years of education have you had? _____
11. Have you ever been diagnosed with depression or any other psychiatric disorder? (if yes, please give specifics of diagnosis)_____

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Vita

Michael Jerry Olds was born in Trenton, Georgia, on October 17, 1972, the son of Judy Floyd Olds and Edward Eugene Olds. After graduating from Southwood High School in May 1991, he enrolled at Louisiana State University in Shreveport. In May of 1995 he graduated from Louisiana State University in Shreveport with a Bachelor of Science degree in psychology. In the fall of 1999 he entered the Graduate School of Southwest Texas State University in San Marcos, Texas.

Permanent address: 12324 Danny Drive
Austin, Texas 78759

This thesis was typed by Michael Jerry Olds.