Training Manual for Providers of Psychoeducational Services to FMS Patients

George E. Vasquez Jr.

Loma Linda University

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LOMA LINDA UNIVERSITY
School of Behavioral Health
in conjunction with the
Department of Psychology

A Training Manual for Providers of
Psychoeducational Services to FMS Patients

by

George E. Vasquez Jr., M.A.

Project submitted in partial satisfaction of
the requirements for the degree of
Doctor of Psychology

June 2012
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<tr>
<td>ACR</td>
<td>American College of Rheumatology</td>
</tr>
<tr>
<td>ACS</td>
<td>American Cancer Society</td>
</tr>
<tr>
<td>ADA</td>
<td>American Dental Association</td>
</tr>
<tr>
<td>AGS</td>
<td>American Geriatric Society</td>
</tr>
<tr>
<td>AMA</td>
<td>American Medical Association</td>
</tr>
<tr>
<td>CBT</td>
<td>Cognitive-Behavioral Therapy</td>
</tr>
<tr>
<td>CDC</td>
<td>Centers for Disease Control and Prevention</td>
</tr>
<tr>
<td>CFS</td>
<td>Chronic Fatigue Syndrome</td>
</tr>
<tr>
<td>CNS</td>
<td>Central Nervous System</td>
</tr>
<tr>
<td>fMRI</td>
<td>Functional Magnetic Resonance Imaging</td>
</tr>
<tr>
<td>FMS</td>
<td>Fibromyalgia Syndrome</td>
</tr>
<tr>
<td>GAD</td>
<td>Generalized Anxiety Disorder</td>
</tr>
<tr>
<td>HCV</td>
<td>Hepatitis C Virus</td>
</tr>
<tr>
<td>HHV-6</td>
<td>Human Herpesvirus-6</td>
</tr>
<tr>
<td>HIV</td>
<td>Human Immunodeficiency Virus Type I</td>
</tr>
<tr>
<td>HPA</td>
<td>Hypothalamic-Pituitary-Adrenal</td>
</tr>
<tr>
<td>IBS</td>
<td>Irritable Bowel Syndrome</td>
</tr>
<tr>
<td>MCSS</td>
<td>Multiple Chemical Sensitivity Syndrome</td>
</tr>
<tr>
<td>NHLBI</td>
<td>National Heart, Lung, and Blood Institute</td>
</tr>
<tr>
<td>NIAMS</td>
<td>National Institute of Arthritis and Musculoskeletal and Skin Diseases</td>
</tr>
<tr>
<td>NIDCD</td>
<td>National Institute on Deafness and Other Communication Disorders</td>
</tr>
<tr>
<td>NIDCR</td>
<td>National Institute of Dental and Craniofacial Research</td>
</tr>
<tr>
<td>Abbreviation</td>
<td>Definition</td>
</tr>
<tr>
<td>--------------</td>
<td>------------</td>
</tr>
<tr>
<td>NIDDK</td>
<td>National Institute of Diabetes and Digestive and Kidney Diseases</td>
</tr>
<tr>
<td>NINDS</td>
<td>National Institute of Neurological Disorders and Stroke</td>
</tr>
<tr>
<td>NREM</td>
<td>Nonrapid Eye Movement</td>
</tr>
<tr>
<td>NRS</td>
<td>Nonrestorative Sleep</td>
</tr>
<tr>
<td>NSAID</td>
<td>Nonsteroidal Anti-Inflammatory Drug</td>
</tr>
<tr>
<td>OCD</td>
<td>Obsessive-Compulsive Disorder</td>
</tr>
<tr>
<td>OSA</td>
<td>Obstructive Sleep Apnea</td>
</tr>
<tr>
<td>PET</td>
<td>Positron Emission Tomography</td>
</tr>
<tr>
<td>PTSD</td>
<td>Post-Traumatic Stress Disorder</td>
</tr>
<tr>
<td>REM</td>
<td>Rapid Eye Movement</td>
</tr>
<tr>
<td>RLS</td>
<td>Restless Legs Syndrome</td>
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<tr>
<td>RP</td>
<td>Raynaud’s Phenomenon</td>
</tr>
<tr>
<td>SA</td>
<td>Sleep Apnea</td>
</tr>
<tr>
<td>SES</td>
<td>Socioeconomic Status</td>
</tr>
<tr>
<td>SNRI</td>
<td>Serotonin-Norepinephrine Reuptake Inhibitor</td>
</tr>
<tr>
<td>SS</td>
<td>Symptom Severity</td>
</tr>
<tr>
<td>SSRI</td>
<td>Selective Serotonin Reuptake Inhibitor</td>
</tr>
<tr>
<td>TCA</td>
<td>Tricyclic Antidepressant</td>
</tr>
<tr>
<td>TMJ</td>
<td>Temporomandibular Joint</td>
</tr>
<tr>
<td>UARS</td>
<td>Upper-airway resistance syndrome</td>
</tr>
<tr>
<td>WPI</td>
<td>Widespread Pain Index</td>
</tr>
</tbody>
</table>
ABSTRACT

A Training Manual for Providers of Psychoeducational Services to FMS Patients

by

George E. Vasquez Jr., M.A.

Doctor of Psychology, Graduate Program in Psychology
Loma Linda University, June 2012
Dr. Louis Jenkins, Chairperson

Fibromyalgia syndrome (FMS) is a chronic pain syndrome that is typically characterized by chronic and widespread pain; tenderness; sleep problems, fatigue, and muscle stiffness. Additional symptoms are many and can include cognitive impairment, numbness, and psychological distress such as anxiety and depression. FMS is typically diagnosed using criteria that were established by the American College of Rheumatology in 2010 and is currently experienced by approximately 5 million adults in the United States. A biopsychosocial conceptualization of the etiology of FMS currently exists, wherein the development of FMS is attributed to the interplay of particular predisposing, precipitating, and perpetuating factors. The management of fibromyalgia syndrome is diverse, often involving psychoeducation, certain pharmacological treatments, and particular nonpharmacological treatments. Among the common nonpharmacological treatment options is psychoeducational group therapy; several nuances of psychoeducational group therapy with fibromyalgia syndrome patients are discussed.
CHAPTER 1
INTRODUCTION

Fibromyalgia syndrome (FMS) is a complex chronic pain disorder that is currently prevalent among approximately 2% of the general U.S. adult population (Wolfe, Ross, Anderson, Russell, & Hebert, 1995). In other words, approximately 5,000,000 adults in the United States experience FMS (Lawrence et al., 2008). The symptomatology, etiology, and management of FMS are generally complex and variable, as is the prognosis for FMS patients (Sharpe & O’Malley, 2005). Regarding diagnosis, FMS is currently diagnosed using the 2010 or 1990 American College of Rheumatology (ACR) diagnostic criteria for fibromyalgia syndrome (Wolfe et al., 2011). Moreover, FMS tends to have a severely maladaptive impact on daily quality of life (Arnold et al., 2008; Bernard, Prince, & Edsall, 2000).

A recent review of the fibromyalgia literature revealed that there is currently no comprehensive review of fibromyalgia that is specifically written for mental health professionals. Given that the role of mental health professionals in the management of FMS has increased over the last decade (Sharpe & O’Malley, 2005), a comprehensive review of FMS that is specifically designed to train mental health professionals so that they can provide competent mental health services to FMS patients seems to be highly warranted. As such, the current training manual attempts to fill the above-stated void in the fibromyalgia syndrome literature. What follows is a comprehensive review of fibromyalgia syndrome, written by one mental health professional for the benefit of other mental health professionals.
CHAPTER 2

THE SYMPTOMATOLOGY OF FIBROMYALGIA SYNDROME

Fibromyalgia syndrome is characterized by the following primary symptoms:

chronic and widespread pain; tenderness; sleep difficulties; fatigue; and muscle stiffness

(Bennett, 2009; Wallace, 1997; see Table 1).

Table 1

*Lowest and highest prevalence rates of FMS symptoms among FMS study populations*

<table>
<thead>
<tr>
<th>FMS Symptom</th>
<th>Lowest Prevalence Rate</th>
<th>Highest prevalence rate</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pain</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Widespread pain</td>
<td>78% (Bengtsson et al., 1986a)</td>
<td>100% (Campbell et al., 1983)</td>
</tr>
<tr>
<td>Localized pain</td>
<td>62% (neck pain; Wolfe &amp; Cathey, 1983)</td>
<td>91% (neck/shoulder pain; Campbell et al., 1983)</td>
</tr>
<tr>
<td>Tenderness</td>
<td>65% (Wolfe et al., 1990)</td>
<td>69% (Wolfe &amp; Cathey, 1983)</td>
</tr>
<tr>
<td>Sleep difficulties</td>
<td></td>
<td></td>
</tr>
<tr>
<td>General sleep difficulty</td>
<td>56% (Yunus et al., 1981)</td>
<td>80% (Goldenberg, 1987)</td>
</tr>
<tr>
<td>Sleep initiation difficulty</td>
<td>36% (Campbell et al., 1983)</td>
<td>53% (Bengtsson et al., 1986a)</td>
</tr>
<tr>
<td>Sleep maintenance difficulty</td>
<td>62% (Wolfe et al., 1985)</td>
<td>71% (Bengtsson et al., 1986a)</td>
</tr>
<tr>
<td>Nonrestorative sleep</td>
<td>78% (Bengtsson et al., 1986a)</td>
<td>95% (Campbell et al, 1983)</td>
</tr>
<tr>
<td>Sleep apnea</td>
<td>9% (Shaver et al., 2006)</td>
<td>25% (Hamm et al., 1989)</td>
</tr>
<tr>
<td>Restless legs syndrome</td>
<td>20% (Shaver et al., 2006)</td>
<td>64% (Stehlik et al., 2009)</td>
</tr>
<tr>
<td>Upper-airway resistance syndrome</td>
<td>N/A</td>
<td>96% (Gold et al., 2003a)</td>
</tr>
<tr>
<td>Fatigue</td>
<td>55% (Wolfe et al., 1985)</td>
<td>100% (Campbell et al., 1983)</td>
</tr>
<tr>
<td>Muscle stiffness</td>
<td>40% (Bengtsson et al., 1986a)</td>
<td>84% (Yunus et al., 1981)</td>
</tr>
<tr>
<td>Condition</td>
<td>Percentage</td>
<td>Source</td>
</tr>
<tr>
<td>------------------------------------------</td>
<td>------------</td>
<td>-------------------------------------</td>
</tr>
<tr>
<td>Morning muscle stiffness</td>
<td>75%</td>
<td>Wolfe &amp; Cathey, 1983</td>
</tr>
<tr>
<td></td>
<td></td>
<td>91% (Campbell et al., 1983)</td>
</tr>
<tr>
<td>Attention deficits</td>
<td>N/A</td>
<td>100% (Dick et al., 2002)</td>
</tr>
<tr>
<td>Impaired selective visual attention ability</td>
<td>N/A</td>
<td>100% (Grace et al., 1999)</td>
</tr>
<tr>
<td>Poorly developed sustained auditory concentration ability</td>
<td>N/A</td>
<td>100% (Glass et al., 2004)</td>
</tr>
<tr>
<td>Divided attention (dual-tasking) difficulty</td>
<td>N/A</td>
<td></td>
</tr>
<tr>
<td>Memory deficits</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Impaired working memory</td>
<td>23%</td>
<td>(Leavitt &amp; Katz, 2006)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>83% (when distraction present; Leavitt &amp; Katz, 2006)</td>
</tr>
<tr>
<td>Impaired episodic memory</td>
<td>N/A</td>
<td>N/A</td>
</tr>
<tr>
<td>Impaired semantic memory</td>
<td>N/A</td>
<td>49% (Leavitt &amp; Katz, 2008)</td>
</tr>
<tr>
<td>Processing speed deficit – A selective naming speed deficit</td>
<td>N/A</td>
<td>49% (word reading deficit); 54% (color naming deficit; Leavitt &amp; Katz, 2008)</td>
</tr>
<tr>
<td>Executive functioning deficits</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Shifting deficit</td>
<td>N/A</td>
<td>N/A</td>
</tr>
<tr>
<td>Impaired temporal preparation ability</td>
<td>N/A</td>
<td>N/A</td>
</tr>
<tr>
<td>Inhibition deficit</td>
<td>N/A</td>
<td>N/A</td>
</tr>
<tr>
<td>Paresthesias</td>
<td>26%</td>
<td>(Yunus et al., 1981)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>84% (Simms &amp; Goldenberg, 1988)</td>
</tr>
<tr>
<td>Subjective joint and soft tissue swelling</td>
<td>32%</td>
<td>(Yunus et al., 1981)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>100% (Jacobsen et al., 1993)</td>
</tr>
<tr>
<td>Impaired balance</td>
<td>45%</td>
<td>(Bennett et al., 2007)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>68% (Katz et al., 2005)</td>
</tr>
<tr>
<td>Raynaud’s phenomenon</td>
<td>10%</td>
<td>(Bennett et al., 1993)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>53% (Vaerøy et al., 1988)</td>
</tr>
<tr>
<td>Oral and ocular symptoms</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sjögren’s syndrome</td>
<td>N/A</td>
<td>7% (Bonafede et al., 1995)</td>
</tr>
<tr>
<td>Xerostomia</td>
<td>12%</td>
<td>(Yunus et al., 1993)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>63% (Katz et al., 2007)</td>
</tr>
<tr>
<td>Keratoconjunctivitis sicca</td>
<td>N/A</td>
<td>38% (Günaydin et al., 1999)</td>
</tr>
<tr>
<td>TMJ dysfunction</td>
<td>29%</td>
<td>(Bennett et al., 2007)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>75% (Plesh et al., 1996)</td>
</tr>
<tr>
<td>Glossodynia</td>
<td>N/A</td>
<td>33% (Rhodus et al., 2003)</td>
</tr>
<tr>
<td>Dysphagia</td>
<td>37%</td>
<td>(Rhodus et al., 2003)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>42% (Katz et al., 2007)</td>
</tr>
<tr>
<td>Dysgeusia</td>
<td>N/A</td>
<td>34% (Rhodus et al., 2003)</td>
</tr>
<tr>
<td>Subjective dizziness</td>
<td>4%</td>
<td>(Rosenhall et al., 1996)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>61% (Katz et al., 2007)</td>
</tr>
<tr>
<td>Subjective vertigo</td>
<td>8%</td>
<td>(Bayazit et al., 2002)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>47% (Bengtsson et al., 1986a)</td>
</tr>
</tbody>
</table>
Chronic and Widespread Pain

Many fibromyalgia syndrome patients express that they experience “pain all over” (Wolfe et al., 1990, p. 170). They describe their pain as a gnawing/aching/burning sensation that originates at a given joint and/or muscle in one quadrant of the body (i.e., at a tender point site) and spreads throughout the rest of the body (Bennett, 2009; Hallberg & Carlsson, 2000; Sharpe & O’Malley, 2005; Wallace, 1997). Many fibromyalgia patients describe their pain as constantly present (Wallace, 1997) and the intensity of their pain as waxing and waning throughout the day (Bennett, 2009; Moldofsky, 1994; Yunus, Masi, Calabro, & Shah, 1982). To elaborate, the pain typically waxes (i.e., intensifies) from awakening to approximately 10:00 A.M., wanes (i.e.,

<table>
<thead>
<tr>
<th>Symptom</th>
<th>Rate</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Headaches</td>
<td>44% (Yunus et al., 1981)</td>
<td>76% (Marcus et al., 2005)</td>
</tr>
<tr>
<td>Increased sensory sensitivity</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Skin sensitivity and hives</td>
<td>N/A</td>
<td>43% (Katz et al., 2007)</td>
</tr>
<tr>
<td>Light sensitivity</td>
<td>N/A</td>
<td>69% (Katz et al., 2007)</td>
</tr>
<tr>
<td>Sun sensitivity</td>
<td>N/A</td>
<td>45% (Katz et al., 2007)</td>
</tr>
<tr>
<td>Sexual dysfunction</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Decreased sexual desire</td>
<td>N/A</td>
<td>63% (Aydin et al., 2006)</td>
</tr>
<tr>
<td>Decreased sexual arousal</td>
<td>N/A</td>
<td>54% (Aydin et al., 2006; Shaver et al., 2006)</td>
</tr>
<tr>
<td>Difficulty achieving orgasm</td>
<td>33% (Shaver et al., 2006)</td>
<td>56% (Aydin et al., 2006)</td>
</tr>
<tr>
<td>Decreased sexual satisfaction</td>
<td>N/A</td>
<td>48% (Aydin et al., 2006)</td>
</tr>
<tr>
<td>Psychological distress</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Anxiety</td>
<td>38% (Bennett et al., 2007)</td>
<td>76% (Katz et al., 2007)</td>
</tr>
<tr>
<td>An anxiety disorder</td>
<td>27% (Epstein et al., 1999)</td>
<td>60% (Arnold et al., 2006)</td>
</tr>
<tr>
<td>Depression</td>
<td>29% (Ahles et al., 1987)</td>
<td>80% (Martinez et al., 1995)</td>
</tr>
<tr>
<td>Any mood disorder</td>
<td>29% (Epstein et al., 1999)</td>
<td>35% (Thieme et al., 2004)</td>
</tr>
</tbody>
</table>
decreases) from approximately 10:00 A.M. to approximately 2:00 P.M., and waxes again from approximately 2:00 P.M. to bedtime (Bennett, 2009; Moldofsky, 1994; Yunus et al., 1982).

Flares are bouts of significantly intense pain that are experienced for indiscriminate amounts of time and can be triggered by any of the following factors: surgery, a soft tissue injury, significant physical exertion, extended inactivity, a long car trip, and psychological stress such as depression and/or anxiety (Bennett, 2009; Yunus et al., 1982). Furthermore, particular weather conditions have been described by fibromyalgia patients as factors that exacerbate pain, sometimes triggering flares (De Blécourt, Knipping, de Voogd, & van Rijswik, 1993; Hagglund, Deuser, Buckelew, Hewett, & Kay, 1994; Russell, 1989; Wolfe, Hawley, Cathey, Caro, & Russell, 1985; Yunus et al., 1982). Specifically, decreased temperature (i.e., cold weather; Strusberg, Mendelberg, Serra, & Strusberg, 2002) and high atmospheric pressure (Guedj & Weinberger, 1990; Strusberg et al., 2002) have been observed to exacerbate pain in FMS patients. In addition, widespread pain has been observed among 100 to 78% of fibromyalgia study patients (Bengtsson et al., 1986a; Campbell, Clark, Tindall, Forehand, & Bennett, 1983; Wolfe et al., 1990; Yunus, Masi, & Aldag, 1989), whereas localized pain (e.g., neck pain) has been observed among 91 to 62% of fibromyalgia study patients (Campbell et al., 1983; Wolfe & Cathey, 1983; Wolfe et al., 1985).

**Tenderness**

Tender skin is a common symptom of fibromyalgia syndrome (Bennett, 2009; Martínez-Lavin, López, Medina, & Nava, 2003; Reilly & Littlejohn, 1992; Sharpe &
O’Malley, 2005). Many fibromyalgia patients experience increased skin sensitivity to touch, especially at tender point sites, such that any form of contact between a patient’s skin and another object results in the experience of pain for the patient (Bennett, 2009; Martínez-Lavin et al., 2003; Reilly & Littlejohn, 1992; Wallace, 1997). Carli, Suman, Biasi, and Marcolongo (2002) observed a relationship between the number of tender point sites on a patient and the patient’s pressure pain threshold, in which an increase in the number of tender point sites on a patient tends to be accompanied by a reduction in the patient’s pressure pain threshold. In other words, FMS patients with a higher number of tender point sites (and therefore experiencing more widespread tenderness) are more likely than patients with a lower number of tender point sites (who consequently experience more localized tenderness) to have lower superficial and deep pressure pain thresholds (Carli et al., 2002). Thus, increased tenderness is associated with increased sensitivity to pain, such that pain is experienced at a level of skin and deep tissue (muscular) pressure that does not evoke a pain response in healthy people (i.e., people who are not diagnosed with FMS; Carli et al., 2002).

A review of the literature reveals a lack of research regarding the prevalence of tenderness among the members of the fibromyalgia population. Nevertheless, Wolfe and Cathey (1983) and Wolfe et al. (1990) observed tenderness (i.e., “skinfold tenderness”) among 69 and 65%, respectively, of their respective FMS study samples.

**Sleep Difficulties**

Many FMS patients describe their sleep as abnormal or disturbed (Affleck, Urrows, Tennen, Higgins, & Abeles, 1996; Shaver et al., 1997; Wolfe et al., 1990). They
report difficulty regarding the initiation as well as maintenance of sleep (i.e., they report
difficulty falling asleep as well as staying asleep; Bennett, 2009; Campbell et al., 1983).
Some patients report that any type of noise can awaken them from sleep and others
endorse being so restless during sleep that they are consciously aware of their thoughts
and dreams (and as such, do not experience restorative sleep; Harding, 1998). According
to Bennett (2009), nonrestorative sleep (NRS) is the most prominent sleep difficulty that
is experienced by FMS patients. Nonrestorative sleep refers to the experience of waking
up from one’s normal sleep duration feeling tired/unrefreshed (Stone, Taylor, McCrae,
Kalsekar, & Lichstein, 2008). Furthermore, nonrestorative sleep has been found to cause
the greatest impairment in daytime functioning (Ohayon, 2005). Nonrestorative sleep
significantly increases physical and intellectual fatigue as well as sensitivity to touch; it
impairs alertness and memory functioning; and it exacerbates anxiety and depression
(Ohayon, 2005). As such, it would seem that if a fibromyalgia patient’s symptomatology
included NRS, then the patient would experience exacerbations of his or her other
symptoms (i.e., the patient’s nonrestorative sleep would likely exacerbate his or her
tenderness, fatigue, cognitive difficulties, anxiety, and/or depressed mood; Ohayon,
2005). Sleep difficulties have been observed among 95 to 36% of FMS study patients
(Bengtsson et al., 1986a; Campbell et al., 1983; Goldenberg, 1987; Wolfe & Cathey,
1983; Wolfe et al., 1985; Wolfe et al., 1990; Yunus et al., 1989; Yunus, Masi, Calabro,

Moreover, the literature (e.g., Affleck et al., 1996; Schaefer, 1995) suggests that
there is a relationship between sleep quality and the experience of pain for the
fibromyalgia patient, where poor sleep tends to exacerbate the pain that is experienced
during the following day. To elaborate, there are two types of sleep: nonrapid eye
movement (NREM) sleep and rapid eye movement (REM) sleep (Berry, Geyer, &
Carney, 2005). Furthermore, there are four stages of NREM sleep (stages one, two, three,
and four; Berry et al., 2005). The first two stages of NREM sleep are commonly
identified as light NREM sleep whereas the last two stages are commonly identified as
deep NREM sleep, delta sleep, or slow-wave sleep (SWS; Bae & Foldvary-Schaefer,
2005). In general, adults experience five to seven sleep cycles each night (Bae &
Foldvary-Schaefer, 2005); each cycle is comprised of one segment of NREM sleep (i.e.,
the individual experiences all four stages of NREM sleep) followed by one segment of
REM sleep (Berry et al., 2005). Regarding the relationship between sleep quality and
pain, researchers (e.g., Moldofsky, Scarisbrick, England, and Smythe, 1975) have
indicated that when a fibromyalgia patient’s stage four NREM sleep is disturbed (due to
any sleep difficulty), disruptions in the metabolism of serotonin are likely to subsequently
occur (serotonin is a neurotransmitter that has been implicated in pain perception; Chase
& Murphy, 1973; Kundermann et al., 2009; Moldofsky & Warsh, 1978; Pickering,
Januel, Dubray, & Eschalier, 2003). Consequently, the patient tends to awaken the
following morning with a reduced pain threshold, which results in increased pain
perception throughout the remainder of the day (Moldofsky et al., 1975).

Some FMS patients also suffer from a primary sleep disorder such as sleep apnea,
restless legs syndrome, and/or upper-airway resistance syndrome (Bennett, 2009). Sleep
apnea (SA) is a disorder that occurs during sleep, in which an individual experiences
shallow breathing or one or more breathing pauses (National Heart, Lung, and Blood
Institute [NHLBI], 2010). The duration of a breathing pause can range from a few
seconds to a few minutes (NHLBI, 2010). According to the NHLBI (2010), OSA or obstructive sleep apnea is the most typical form of sleep apnea. Similar to SA, OSA occurs during sleep and produces shallow breathing or breathing pauses; in contrast to SA, OSA is characterized by airway blockage/collapse, which typically results in snoring (the movement of air through the blockage/collapse produces the snoring; NHLBI, 2010). Moreover, individuals with SA are at an increased risk for developing high blood pressure, diabetes, obesity, and/or an irregular heartbeat (NHLBI, 2010). They are also at an increased risk for experiencing a stroke, heart attack, and/or driving or work-related accident (NHLBI, 2010). Sleep apnea has been observed among 25 to 9% of fibromyalgia study patients (Hamm, Derman, & Russell, 1989; May, West, Baker, & Everett, 1993; Shaver, Wilbur, Robinson, Wang, & Buntin, 2006).

Restless legs syndrome (RLS) is a neurologically-based movement disorder that is characterized by the experience of significantly unpleasant sensations in the legs (often in the calf area of the legs; Mahowald, 2003; Stepanski, 2005). These unpleasant sensations tend to occur whenever an individual is forced to hold his or her legs still for an extended period of time, such as during sleep, a long performance, a car trip, and/or an airplane ride (Mahowald, 2003; Stepanski, 2005). Furthermore, when RLS is experienced during sleep, the symptoms (i.e., the unpleasant sensations) tend to be strongest between midnight and 4 A.M. (Hening et al., 1999). As such, RLS is one of the prominent causes of insomnia (Mahowald, 2003). According to Mahowald (2003), RLS patients tend to describe their significantly unpleasant sensations as “crawling,” “searing,” “boring,” “drawing,” or “pulling” sensations. Terms such as “tingling,” “pain,” or “numbness”
(terms that are typically used by an individual to describe an unpleasant sensation) are rarely used by RLS patients to describe their unpleasant sensations (Mahowald, 2003).

Moreover, stimulation/movement of the legs (e.g., stomping one’s feet, walking around one’s room, and/or rubbing/stroking one’s legs) is the typical method of obtaining relief from the unpleasant sensations of RLS; however, these relief efforts are only effective as they are being employed (Mahowald, 2003). Typically, after an RLS patient employs one of the relief efforts listed above, the patient returns to an inactive state (e.g., returns to bed) where his or her legs return to a stationary position; the unpleasant sensations subsequently return (Mahowald, 2003). In addition, restless legs syndrome has been observed among 64 to 20% of fibromyalgia syndrome study samples (Shaver et al., 2006; Stehlik, Arvidsson, & Ulfberg, 2009; Yunus & Aldag, 1996).

Upper-airway resistance syndrome (UARS) is a sleep disorder that is marked by a disturbed pattern of breathing (Gold, Dipalo, Gold, & Broderick, 2003a; Guilleminault, Strohs, Clerk, Cetel, & Maistros, 1993). To elaborate, for the individual suffering from UARS, there is a tendency for the individual’s airway to partially collapse during sleep, resulting in mildly restricted airflow, which in turn, results in increased breathing efforts and arousal from sleep (Gold et al., 2003a; Guilleminault et al., 1993). According to Gold, Marcus, Dipalo, and Gold (2002), the collapse of the upper airway in UARS is less severe than the collapse of the upper airway in OSA; however, this collapse of the upper airway is still more severe than that which is experienced by healthy control subjects during sleep. Furthermore, the common symptoms of UARS include chronic insomnia, nocturnal awakening with subsequent difficulty returning to sleep, daytime fatigue, cold hands and feet, and lightheadedness following abrupt standing or bending (Guilleminault
Other symptoms of UARS include headaches, irritable bowel syndrome (IBS), and sleep-onset insomnia (Gold, Dipalo, Gold, and O’Hearn, 2003b). Moreover, Gold et al. (2003a) observed UARS among 96% of their FMS study sample.

Fatigue

Fatigue is another primary symptom of fibromyalgia syndrome (Sharpe & O’Malley, 2005; Wallace, 1997; Yunus & Masi, 1993). Many fibromyalgia patients describe fatigue as a state of general weariness (i.e., a state of physical, cognitive, and emotional weariness) that impairs physical functioning and reduces life satisfaction/enjoyment (Arnold, 2008; Bennett, 2009). Physical weariness is low energy, reduced physical activity, tiredness, general weakness, reduced physical endurance, slowness/sluggishness, sleepiness, and increased effort in order to complete a physical task and/or overcome a state of physical inactivity (Arnold, 2008). Cognitive weariness denotes decreased attention and concentration, slowed thinking, and reduced mental endurance (Arnold, 2008). Emotional weariness refers to reduced initiation of behavior, decreased motivation, a lack of interest in other people and activities, a general avoidance of effort, and feeling bored, low, and/or overwhelmed (Arnold, 2008). Bennett (2009) indicated that nonrestorative sleep (as mentioned above) and depression (discussed below) tend to exacerbate fatigue. Fatigue has been observed among 100 to 55% of fibromyalgia study patients (Bengtsson et al., 1986a; Campbell et al., 1983; Goldenberg, 1987; Wolfe & Cathey, 1983; Wolfe et al., 1985; Wolfe et al., 1990; Yunus et al., 1989; Yunus et al., 1981).
Muscle Stiffness

According to Bennett (2009) and Yunus and Masi (1983), muscle stiffness is a highly common symptom of fibromyalgia syndrome. Muscle stiffness refers to an experience of tight and contracting sensations in one’s muscles when one is at a state of rest (J. C. Jones, 2007). Relaxed and supple sensations, which are typically experienced in one’s muscles when one is at a state of rest, are not experienced in the muscles of the FMS patient with muscle stiffness (such sensations are replaced by a general sensation of tension; J. C. Jones, 2007). Fibromyalgia patients tend to experience muscle stiffness in the morning (Yunus, Holt, Masi, & Aldag, 1988). The duration of this “morning” muscle stiffness tends to range from 5 to 240 minutes (Yunus et al., 1981). The literature (e.g., Bennett, 2009; Lakie & Robson, 1988) suggests that morning muscle stiffness is a consequence of the following thixotropic property of muscle tissue: muscle tissue tends to become stiff as the duration of sleep/rest increases. Thus, it appears that the duration of an FMS patient’s nightly sleep period, regardless of the presence of sleep disturbances, is long enough for the patient to awaken in the morning with stiff muscles. Moreover, general muscle stiffness has been observed among 84 to 40% of fibromyalgia syndrome study patients (Bengtsson et al., 1986a; Goldenberg, 1987; Wolfe et al., 1990; Yunus et al., 1989; Yunus et al., 1981) whereas morning muscle stiffness has been observed among 91 to 75% of fibromyalgia syndrome study patients (Bengtsson et al., 1986a; Campbell et al., 1983; Wolfe & Cathey, 1983; Wolfe et al., 1985).
Introduction to Additional Symptoms

A review of the literature (e.g., Bennett, 2009; Wallace, 1997; Wolfe et al., 1990) reveals that there are other symptoms associated with fibromyalgia syndrome. These other symptoms are generally less common than the primary symptoms discussed above (i.e., they are less characteristic of fibromyalgia than are the primary symptoms discussed above; see Table 1); however, the impact of any of these other symptoms on the daily functioning of a fibromyalgia patient can be just as significant as the impact of one of the primary symptoms discussed above (Bennett, 2009; Wallace, 1997). In other words, the following symptoms can impair the daily functioning of a fibromyalgia patient to a degree that is comparable to that of one of the primary symptoms discussed above.

Cognitive Impairment

The general cognitive functioning of a fibromyalgia syndrome patient is often impaired by attention/concentration and memory deficits (Glass, 2008). These attention/concentration and memory deficits are often referenced in the fibromyalgia literature (e.g., Sharpe & O’Malley, 2005; Wallace, 1997) as the “single” symptom of cognitive impairment. Cognitive impairment among fibromyalgia patients can also include a selective processing speed deficit (Leavitt & Katz, 2008) and executive functioning deficits (Glass, Park, Crofford, & Fougnie, 2006; Correa, Miró, Martínez, Sánchez, & Lupiáñez, 2011).
Attention/concentration deficits

Attention or concentration refers to “the ability to select some information for more detailed inspection, while ignoring other information” (Atkinson, Atkinson, Smith, Bem, & Nolen-Hoeksema, 2000, p. 691). The following attention/concentration deficits have been observed among some fibromyalgia syndrome patients: impaired selective visual attention (Dick, Eccleston, & Crombez, 2002); poorly developed sustained auditory concentration ability (Grace, Nielson, Hopkins, & Berg, 1999); and dual-tasking difficulty (Glass, Park, & Crofford, 2004). Selective visual attention refers to an individual’s ability to sort out visual information (i.e., to detect visual information that is relevant to the goal/task at hand while ignoring visual information that is irrelevant or distracting to the goal/task at hand; Robertson, Ward, Ridgeway, & Nimmo-Smith, 1994). Research (e.g., Dick et al., 2002) suggests that there is a subset of the fibromyalgia syndrome population that demonstrates impaired selective visual attention ability - these individuals are unable to sort out visual information (i.e., they are unable to ignore distracting visual information). To illustrate, Dick et al. (2002) observed impaired selective visual attention ability among 100% of the fibromyalgia patients in their study. For this subset of the fibromyalgia population, impaired selective visual attention can impact daily functioning. For example, a fibromyalgia patient demonstrating impaired selective visual attention ability will likely be unable to complete medical history forms in the lobby of a doctor’s office in a timely manner, for the patient will likely be unable to ignore the distracting visual stimuli of the lobby of the doctor’s office (e.g., the activities of other patients in the lobby). Consequently, the doctor may require the patient
to stay in the lobby after the appointment and complete the medical history forms, which may disrupt the patient’s plans for the rest of the day.

Sustained auditory concentration ability refers to an individual’s ability to maintain active concentration on a long-term auditory task or goal (Manly, Robertson, Anderson, & Nimmo-Smith, 1999). As such, individuals with poorly developed sustained auditory concentration ability (e.g., some fibromyalgia patients) are unable to sustain attention for an extended period of time on an auditory activity such as a conversation, regardless of their level of interest in the individual(s) with whom they are conversing, for concentrating on the speech of another individual for an extended period of time is quite challenging/difficult for them (Robertson et al., 1994). Consequently, these individuals are likely to prefer and exclusively engage in short conversations with other people to avoid the challenge of concentrating on the speech of other people for an extended period. Regarding the prevalence of impaired sustained auditory concentration ability among FMS patients, 100% of the FMS patients in Grace et al.’s (1999) study demonstrated significant sustained auditory concentration difficulty.

Divided attention or dual-tasking ability refers to an individual’s ability to engage in more than one activity at a time (e.g., engaging in a conversation with one’s spouse while simultaneously cooking dinner; Robertson et al., 1994). As such, individuals with divided attention difficulty (e.g., some FMS patients) tend to engage in only one activity at a time, for engagement in multiple activities simultaneously is quite difficult and distressing for them (Gross et al., 2004). For an FMS patient demonstrating impaired dual-tasking ability, work performance can be hindered by the inability to perform more than one activity at a time (many work responsibilities require the ability to dual task). As
the impairment progresses over time, the patient may request a reduction in her work hours in order to compensate for the impairment, be pressured to resign or quit by her employer on account of diminished productivity, or be fired from her place of employment altogether on account of unsatisfactory job performance; regardless of the subsequent course of action, the patient’s occupational functioning / financial situation (daily functioning) will be impacted (Arnold et al., 2008). Glass et al. (2004) observed dual-tasking difficulty among 100% of the fibromyalgia patients in their study.

**Memory Deficits**

Deficits in several cognitive processes (specifically in working memory, episodic memory, and semantic memory) have been observed among some fibromyalgia patients (Glass, 2008). In working memory, information is simultaneously stored and processed for approximately less than 30 seconds (Glass, 2008; Park, Glass, Minear, & Crofford, 2001). Researchers (e.g., Park et al., 2001) have suggested that working memory is indicative of “mental horsepower;” in other words, working memory refers to the cognitive energy / problem-solving capacity that an individual possesses and employs in his or her daily activities. Activities of daily living that involve working memory include dialing a phone number, writing an individual’s phone number on a piece of paper as the individual is announcing his or her phone number, taking written notes during a doctor’s appointment, and communicating with others (i.e., understanding others’ speech and producing speech in response; Lehmann & Schnider, 2008).

Individuals with impaired working memory (e.g., some FMS patients) are unable to simultaneously store and process information for approximately less than 30 seconds;
as such, performing activities of daily living can be quite challenging (Glass, 2008). For example, a fibromyalgia patient with impaired working memory will likely experience significant difficulty when communicating with a doctor. During a doctor’s appointment, it will likely be quite difficult for the patient to simultaneously store and process the information that is received from the doctor (e.g., recommendations regarding pain medication). Consequently, the patient will likely be less inclined to ask appropriate follow-up questions (e.g., questions regarding the side effects of a pain medication), for there will not have been enough time, within the context of the conversation, for the patient to process the new information (the recommendations) and develop an appropriate response (follow-up questions). Thus, impaired working memory could significantly interfere with a fibromyalgia patient’s pain management. Moreover, working memory deficits in fibromyalgia patients are consistent with working memory deficits in older adults who are not diagnosed with fibromyalgia (Park et al., 2001).

Deficits in working memory for fibromyalgia patients tend to be exacerbated when a source of distraction is present (Leavitt & Katz, 2006). For example, when a nurse interrupts a conversation between a fibromyalgia patient and the patient’s rheumatologist in order to ask the rheumatologist a question, the interruption interferes with the patient’s ability to retain in working memory the content of the conversation between the patient and the patient’s rheumatologist. The nurse’s interruption becomes the focus of the patient’s attention and subsequently, the focus of the patient’s working memory (i.e., simultaneously storing and processing information regarding the nurse and the nurse’s question supersedes simultaneously storing and processing the content of the recent conversation between the patient and the patient’s rheumatologist; the latter
information subsequently fades from awareness). Consequently, upon the nurse’s departure from the room, it will likely be quite difficult for the patient to resume the conversation with the rheumatologist, for the patient will have to refocus attention and working memory on to the content of the conversation (i.e., “catch up” with the conversation). Thus, distraction seems to have the capacity to seriously worsen the working memory ability of some FMS patients (Leavitt & Katz, 2006). In addition, Leavitt and Katz (2006) observed impaired working memory among 23% of the fibromyalgia syndrome patients in their study; the prevalence rate increased to 83% when distraction was introduced to the working memory task.

Episodic memory, a type of long-term memory, refers to an individual’s ability to recall specific episodes or events from the individual’s past, in relation to time and location (Brand & Markowitsch, 2003; Glass, 2009). An example of an episodic memory is an individual’s memory of the doctor’s appointment in which the individual received the diagnosis of fibromyalgia syndrome. There is a subset of the fibromyalgia syndrome population that experiences impaired episodic memory functioning - these individuals are unable to recall information regarding specific past events or episodes (Grace et al., 1999; Landrø, Stiles, & Sletvold, 1997; Park et al., 2001). For this subset of the FMS population, impairments in episodic memory can significantly impact daily functioning (Landrø et al., 1997; Rao et al., 1991).

To illustrate, imagine an FMS patient whose ability to recall information regarding specific past events has recently declined. Specifically, imagine a patient who reports (to a psychologist) recently reduced ability to recall the daily physical therapy techniques that were learned at a past consultation appointment with a physical therapist.
(the consultation appointment occurred approximately two months ago). The patient subsequently states that not being able to recall all of these physical therapy techniques has resulted in incomplete daily physical therapy and consequently, generally increased muscle pain. In response to the patient’s presenting problem, the psychologist suggests that the patient employ a compensatory strategy such as note-taking in important activities of daily living (e.g., future physical therapy appointments). Employment of such a compensatory strategy is intended to assist the patient in living as close to baseline as possible (i.e., it is intended to assist the patient in remembering important information such as information from a future physical therapy appointment). Overall, the presence of episodic memory deficits and the employment of commensurate coping skills can impact (change) the daily functioning of an FMS patient. In addition, Park et al. (2001) observed that for FMS patients demonstrating impaired episodic memory, they are likely to experience a level of difficulty in recalling specific past events and/or episodes that is consistent with the level of difficulty that is commonly experienced by older people undergoing normal cognitive (i.e., episodic memory) decline.

Semantic memory, another type of long-term memory, refers to an individual’s ability to recall general knowledge about the world (Farah & Grossman, 2003). An individual’s general knowledge about the world is comprised of facts as well as general information regarding the people, events, and objects (including words) of the individual’s world (Carlson, 2007; Farah & Grossman, 2003; Tulving, 1972). Examples of a semantic memory include the memory of the identity of the current president of the United States (people), the memory of the date on which the World Trade Center collapsed (events), and the memory of the definition of a word and/or the fact that
chronic and widespread pain is a primary characteristic of fibromyalgia syndrome (objects; Carlson, 2007; Galotti, 1999).

Research (e.g., Leavitt & Katz, 2008; Park et al., 2001) suggests that there is a subset of the fibromyalgia population that experiences impaired semantic memory functioning – these individuals are unable to access (recall) facts / general information about the world. To illustrate, Leavitt and Katz (2008) observed impaired semantic memory functioning in approximately 49% of the fibromyalgia patients in their study. In addition, Park et al. (2001) observed that for FMS patients demonstrating impaired semantic memory, they are likely to have trouble in recalling general information about the world in much the same manner as older people undergoing normal cognitive (i.e., semantic memory) decline. Moreover, for this subset of the FMS population, impairments in semantic memory can significantly impact daily functioning (Glass, 2009).

For example, researchers (e.g., Arnold et al., 2008) have observed word-finding difficulties (commonly referenced in the literature as fibro fog) in FMS patients who endorse memory dysfunction. According to one patient, “‘Yeah, normal ordinary words that you’re trying to say just don’t come to you’” (Arnold et al., 2008, p. 117). Given that word knowledge (which includes the definitions of words, the phonetic rules governing the pronunciation of words, and the grammatical rules governing the use of words in spoken sentences) is stored in semantic memory, if an FMS patient experiences word-finding difficulties then not only is impaired semantic memory functioning indicated, but it is highly likely that the patient will experience communication/social difficulties (Glass, 2009). In other words, the patient will likely be unable to orally articulate thoughts as well as respond to questions in a quick manner, for the patient will likely be
unable to access semantic memory and retrieve (recall/remember) the word(s) that are intended to be said (Arnold et al., 2008). Consequently, as the word-finding difficulties progress over time and communication with others becomes increasingly difficult and frustrating, it is likely that the patient will start abstaining from social interactions so as to avoid the challenge of communicating as well as feelings of guilt, frustration, and embarrassment over impaired cognitive (i.e., semantic memory) functioning (Arnold et al., 2008). For fibromyalgia patients such as the one in the example above, impaired semantic memory functioning has the potential to significantly weaken social/daily functioning (Arnold et al., 2008).

Processing Speed Deficit – A Selective Naming Speed Deficit

Processing speed refers to the amount of time that is required for an individual to process sensory information (Leavitt & Katz, 2008). It is comprised of many individual speed abilities, including naming speed (Roberts & Stankov, 1999). Naming speed is the amount of time that is required for an individual to verbally process visual stimuli (i.e., to read a written word or name/state the color of an object; Leavitt & Katz, 2008). Recent research (e.g., Leavitt & Katz, 2008) suggests that there is a subset of the FMS population that experiences a selective naming speed deficit – these individuals are markedly slow at reading singular words and naming colors. Specifically, Leavitt and Katz (2008) observed FMS patients demonstrating a selective naming speed that was (on average) 203 milliseconds slower than the selective naming speed of healthy controls. Moreover, Leavitt and Katz (2008) observed slow word reading speed among 49% of the
FMS patients in their study; slow color naming speed was observed among 54% of the FMS patients in their study.

The literature is currently void of research or theories regarding the impact of a selective naming speed deficit on the daily functioning of a fibromyalgia patient (Leavitt & Katz, 2008); however, given that any type of processing speed deficit can interfere with the functioning of more advanced cognitive functions, it is possible that a selective naming speed deficit will impact aspects of a fibromyalgia patient’s language functioning such as the patient’s basic oral communication skills (Lövdén, Rönnlund, & Nilsson, 2002). For example, it may become difficult for the patient to read an eye chart (i.e., state the letters that are on the chart) during a vision examination. Moreover, Leavitt and Katz (2008) did not observe any other processing speed deficits among the fibromyalgia patients in their study. Furthermore, a review of the literature reveals that in general, fibromyalgia patients tend to demonstrate average processing speed abilities (Grace et al., 1999; Park et al., 2001).

**Executive Functioning Deficits**

Executive functioning refers to the higher-order cognitive capacities that allow an individual to independently undertake a goal-directed activity or task such as writing a training manual for mental health professionals or (for a fibromyalgia patient) developing a weekly schedule that balances work, family/social responsibilities, medication management, doctors’ appointments, exercise behaviors, etc. (Gioia, Isquith, Guy, & Kenworthy, 2000; Lezak, 2003). In short, executive functions are responsible for an individual’s purposeful problem-solving behavior, especially when the problem is novel.
Examples of higher-order cognitive capacities include the capacity to plan and organize an approach to solving a problem / carrying out a task or activity (planning/organization capacity) and the capacity to start a task or activity as well as independently generate problem-solving strategies or ideas (initiation capacity; Gioia et al., 2000). Additional higher-order cognitive capacities include the capacity to smoothly transition from one task, activity, or aspect of a problem to another (shifting capacity; Gioia et al., 2000; Goldberg, 2009) and the capacity to resist or inhibit an impulse (inhibition capacity; Gioia et al., 2000).

Fibromyalgia syndrome patients experience deficits in the following capacities: shifting (Glass et al., 2006), planning/organization (specifically, temporal preparation ability; Correa et al., 2011; Gioia et al., 2000), and inhibition (Correa et al., 2011). Regarding shifting capacity, Glass et al. (2006) observed some fibromyalgia syndrome patients experiencing shifting difficulty in response to increased environmental demands; in other words, as the rules for a set of tasks became more complex, the patients faltered in their transition from one task to another, which led to declines in performance on all of the tasks. For a fibromyalgia syndrome patient experiencing shifting difficulties, daily living can be challenging/frustrating (Glass et al., 2006). The capacity to smoothly transition from one activity or task to another is required for optimal functioning in a complex environment such as the home or work environment, where multiple activities or tasks require attention at approximately the same time and consequently, an individual needs to be able to smoothly transition from one activity or task to another in order to complete all of the activities or tasks on time (Glass et al., 2006). For the fibromyalgia
patient with deficient shifting skills, transitioning from one home or work task to another will likely be cumbersome and slow, resulting not only in time delays for the completion of each task, but in several tasks remaining incomplete at the end of the day (Glass et al., 2006).

Planning/organization capacity is dependent on the functioning of multiple abilities including temporal preparation ability (Gioia et al., 2000). In order for an individual to plan and organize an approach to solving a problem / carrying out a task or activity, the individual’s temporal preparation ability needs to be intact (Gioia et al., 2000). Temporal preparation ability refers to the ability to foresee an upcoming event and consequently develop well-organized responses for the event (Correa et al., 2011). The individual is not only able to predict the timing as well as content (details) of the event, but the individual can also prepare effective responses for the event (Nobre, Correa, & Coull, 2007). The individual’s planned responses are based on similar responses that were effective in the past, when employed during similar events (Nobre et al., 2007).

Research (e.g., Arnold et al., 2008; Correa et al., 2011) suggests that there is a subset of the fibromyalgia population that experiences impaired temporal preparation ability – these individuals are unable to anticipate or prepare a well-organized response for an upcoming event. Correa et al. (2011) proposed the idea that for these particular fibromyalgia patients, the primary symptoms of the syndrome (e.g., chronic pain, fatigue, and sleep difficulties) could be responsible for the impaired temporal preparation ability. For the average individual, engagement in temporal preparation requires focused processing and a high level of cognitive effort (Correa et al., 2011). In comparison, for a particular fibromyalgia patient experiencing sleep deprivation, chronic pain, and fatigue
(among other symptoms), the majority of the patient’s cognitive effort as well as most of the patient’s information processing abilities may be focused on experiencing and/or managing these symptoms; consequently, the patient may be unable to allocate enough cognitive effort and/or redirect the necessary processing information abilities in order to engage in temporal preparation (Correa et al., 2011). For a patient experiencing impaired temporal preparation ability, the cognitive cost of engaging in temporal preparation may be too high given the cognitive demands of the symptoms that are constantly experienced; as such, temporal preparation may be partially engaged in, which may result in ineffective (or no) planning and organizing for future events (Correa et al., 2011). Being unable to effectively plan for future events such as upcoming work meetings or social activities could contribute to declines in functioning in various contexts, which could contribute to overall impaired daily functioning (Arnold et al., 2008).

Correa et al. (2011) suggest that there is a subset of the fibromyalgia syndrome population that experiences inhibition difficulty – these individuals are incapable of inhibiting their impulses. Furthermore, Correa et al. (2011) have proposed that for these particular fibromyalgia patients, the common symptoms of fibromyalgia (e.g., chronic pain and fatigue) could be responsible for the inhibition deficits. To elaborate, most of these patients’ cognitive effort and information processing ability tends to be directed towards experiencing and/or managing common symptoms such as pain and fatigue; consequently, only a minimal amount of their overall cognitive effort and/or information processing ability (if any) is likely available to them for enactment of inhibition (Correa et al., 2011). For this particular type of fibromyalgia patient, the cognitive cost of
inhibiting an impulse may be too high given the cognitive demands of the symptoms that are constantly experienced (Correa et al., 2011).

To illustrate, imagine several above-described FMS patients at a social function. Throughout the function, these patients would likely be preoccupied with their symptoms (i.e., the majority of their cognitive resources would likely be directed towards their inherently attention-demanding symptoms such as pain and fatigue) such that their access to cognitive resources for the enactment of executive functions such as inhibition would likely be limited. When other people engage these patients in conversation throughout the function, they (the patients), due to a lack of available cognitive resources, would likely not be able to enact inhibition to appropriately engage in conversation (i.e., they would likely not be able to resist the impulse to maintain focused attention towards their symptoms). Consequently, these patients would likely maintain focused attention towards their symptoms while simply responding shortly or abruptly to other people to end conversations as quickly as possible and resume their focus on their inherently cognitively-demanding symptoms. Subsequently perceiving the communication style of these fibromyalgia patients as cold or detached, other people might decide to distance themselves from these patients, not only in the context of the function but in the context of the broader social environment, which could result in reductions in the quality and/or quantity of these patients’ social relationships. Given that adaptive social functioning is a critical component of adaptive daily functioning, any reduction in the quality or quantity of a fibromyalgia patient’s social relationships could impair the patient’s daily functioning (Arnold et al., 2008).
**Paresthesias**

A review of the literature reveals that paresthesias are a common symptom of fibromyalgia syndrome (Sharpe & O’Malley, 2005; Wolfe et al., 1990). Paresthesias are sensations of tingling, prickling, or numbness that can be experienced in the upper and/or lower extremities (e.g., in the hands/fingers and/or feet/toes, respectively), as well as in other body parts such as the buttock and mouth (McDonough, 2011). In the context of fibromyalgia syndrome, paresthesias tend to be localized to the upper extremities (Simms & Goldenberg, 1988); they tend to occur sporadically (e.g., during sporadic exacerbations of primary symptoms such as pain and fatigue; Simms & Goldenberg, 1988); and they are often described as “numb” or “pins and needles” sensations (Leavitt, Katz, Golden, Glickman, & Layfer, 1986) as well as tend to be chronic (Simms & Goldenberg, 1988). Furthermore, while some fibromyalgia patients tend to experience paresthesias in both the upper and lower extremities, other patients tend to experience paresthesias only in the lower extremities, and a small minority of patients tend to experience diffuse or widespread paresthesias (Simms & Goldenberg, 1988). In addition, paresthesias have been observed among 84 to 26% of fibromyalgia syndrome study patients (Bengtsson et al., 1986a; Bennett, Jones, Turk, Russell, and Matallana, 2007; Simms & Goldenberg, 1988; Wolfe et al., 1990; Yunus et al., 1989; Yunus et al., 1981).

**Subjective Joint and Soft Tissue Swelling**

A moderate number of fibromyalgia patients report joint and soft tissue swelling (Bradley & Alarcón, 2005). The term joint refers to any body site (e.g., the knee or hip) wherein two or more bones connect (National Institute of Arthritis and Musculoskeletal
Ligaments, which are an example of a soft tissue, are responsible for connecting bones to bones in joints (Asher, 2006). The term soft tissue refers to any body structure that encloses, connects, supports, and/or moves the surrounding body structures; additional examples of soft tissue include muscles and tendons, which are responsible for supporting and moving bones and connecting muscles to bones, respectively (Asher, 2006). Soft tissue swelling refers to the accumulation of an abnormal amount of excess bodily or serous fluid in soft tissue (e.g., in muscles or tendons); when soft tissue swelling occurs in joints (i.e., when swelling occurs in the ligaments of joints, for ligaments are soft tissue), it is termed joint swelling (Asher, 2006; “Edema,” 2011).

In the context of fibromyalgia syndrome, joint and soft tissue swelling tend to be experienced in the upper and lower extremities (e.g., in the hands or fingers and feet or ankles, respectively; Yunus et al., 1981; Yunus et al., 1988). Furthermore, the swelling sensations are subjective; in other words, although the extremities that are endorsed by a patient as swollen may not appear swollen to an observer, they will feel swollen to the patient (Bradley & Alarcón, 2005; Yunus et al. 1981). To elaborate, in all studies (e.g., Jacobsen, Petersen, & Danskiold-Samsøe, 1993; Yunus et al., 1981; Yunus et al., 1988) wherein physical examinations were performed on fibromyalgia patients who reported joint and soft tissue swelling at the time of examination, no objective or noticeable swelling was observed in any of the patients. Moreover, some fibromyalgia patients report that they have had to stop wearing particular pieces of jewelry (e.g., rings) and/or purchase new and larger shoes on account of subjective joint and soft tissue swelling in their fingers/hands/wrists and/or feet/toes, respectively (Bradley & Alarcón,
2005). Jacobsen et al. (1993) observed subjective joint and soft tissue swelling among 100% of their FMS study sample; in contrast, Bengtsson et al. (1986a), Goldenberg (1987), Yunus et al. (1989), and Yunus et al. (1981) observed subjective joint and soft tissue swelling among 64, 52, 40, and 32%, respectively, of their respective FMS study samples.

**Impaired Balance**

Impaired balance is another common symptom of fibromyalgia syndrome (Bennett et al., 2007, K. D. Jones, Horak, Winters-Stone, Irvine, & Bennett, 2009; Katz, Ferbert, & Leavitt, 2007). Balance or postural stability refers to a complex cognitive system wherein the neuromuscular activity that is needed for the maintenance of a desired body position is determined and subsequently executed based on the integration and processing of various sensory inputs (“Balance,” 1999; Horak, 2006; K. D. Jones et al., 2009). There are many neural subsystems involved in balance including the sensory subsystem, wherein sensory information from the visual, vestibular, and somatosensory systems is constantly integrated so that the constantly changing sensory environment can be accurately interpreted, and the biomechanical subsystem, wherein equilibrium or the balanced alignment of the body’s center of mass and base of support (i.e., the feet) is dependent on the functional status of the feet (e.g., their strength, range, and/or pain level) as well as the limits of stability (i.e., how far individuals can lean, in all directions, without losing their balance; Horak, 2006).

Impaired balance refers to a loss of balance or no execution of the neuromuscular activity that is needed for the maintenance of a desired body position, on account of
deficits in one or more of the neural subsystems that comprise the balance system (“Balance,” 1999; Horak, 2006; K. D. Jones et al., 2009). In the context of FMS, it is possible that impaired balance is a consequence of the impact of other FMS symptoms on the functioning of particular neural subsystems of the balance system (K. D. Jones et al., 2009). Researchers (e.g., K. D. Jones et al., 2009) have observed deficits in the biomechanical, sensory, movement, and dynamics subsystems of the balance systems of FMS patients; consequently, these researchers have proposed the idea that the other symptoms of FMS (e.g., fatigue and dual-tasking difficulty) could be interfering with the functioning of particular neural subsystems of the balance system (e.g., the biomechanical and dynamics subsystems, respectively), resulting in impaired balance. Katz et al. (2007) conducted an Internet survey with FMS patients and found impaired balance to be prevalent among 68% of the patients. In contrast, Bennett et al. (2007) conducted an Internet survey with a larger sample of FMS patients and found impaired balance to be prevalent among 45% of the patients.

**Raynaud’s Phenomenon**

Raynaud’s phenomenon (RP) is a condition that is characterized by vasospastic attacks in the extremities (i.e., in the fingers and/or toes; NIAMS, 2009). Vasospastic attacks are episodes of blood vessel constriction or narrowing that are typically triggered by cold stimuli such as low weather or water temperature, as well as emotional stimuli such as daily life stress (NIAMS, 2009; Vaerøy, Helle, Førre, Kåss, & Terenius, 1988). Vasospastic attacks are often described as painful and/or numb sensations (Dinerman, Goldenberg, & Felson, 1986; Vaerøy et al., 1988), and are typically accompanied by
changes in the color of the extremities (NIAMS, 2009; Vaerøy et al., 1988). The color typically changes from baseline to white as the blood vessels constrict, to blue/purple during full blood vessel constriction, to red as the blood vessels dilate or relax and regular blood flow resumes, and then returns to baseline (“Cyanosis,” 2011; NIAMS, 2009; “Pallor,” 2011; “Rubor,” 2011; Vaerøy et al., 1988).

In the context of fibromyalgia syndrome, Raynaud’s phenomenon tends to be accompanied by heightened sensitivity to low temperature or cold weather, such that many fibromyalgia syndrome patients who endorse Raynaud’s phenomenon also tend to report feeling cold in environments where others do not feel cold (Arnold et al., 2008; Bennett, 2009). Furthermore, RP has been observed among 53 to 10% of FMS study samples (Bengtsson et al., 1986a; Bennett et al., 1991; Dinerman et al., 1986; Vaerøy et al., 1988). In addition, heightened sensitivity to temperature was indicated by 78% of the FMS patients in Bengtsson et al.’s (1986a) study; these patients endorsed that they “often [feel] cold” (p. 343).

**Oral and Ocular Symptoms**

A review of the literature reveals that a fair number of FMS patients tend to experience the following oral and ocular symptoms: Sjögren’s syndrome (Bonafede, Downey, & Bennett, 1995), xerostomia (Dinerman et al., 1986; Katz et al., 2007; Rhodus, Fricton, Carlson, & Messner, 2003; Yunus, Hussey, & Aldag, 1993), keratoconjunctivitis sicca (Günaydın et al., 1999), temporomandibular joint (TMJ) dysfunction (Bennett et al., 2007; Hedenberg-Magnusson, Ernberg, & Kopp, 1997; Plesh, Wolfe, & Lane, 1996; Rhodus et al., 2003; Wolfe, Katz, & Michaud, 2005), glossodynia
(Rhodus et al., 2003), dysphagia (Rhodus et al., 2003), and dysgeusia (Rhodus et al., 2003). Sjögren’s syndrome is a condition in which the body’s immune system perceives the glands that produce moisture as threats and consequently attacks those glands (NIAMS, 2010). Examples of such glands include the saliva-producing glands in the mouth, the tear-producing glands in the eyes, and the moisture-producing glands in the throat, nose, and skin (NIAMS, 2010). Moreover, the immune system attacks on the glands impair the functioning of the glands (i.e., the glands are not able to produce moisture on account of the physical damage caused by the immune system), leading to the development of dry mouth (xerostomia), dry eyes (keratoconjunctivitis sicca), chronic dry cough, dry skin, and skin rashes (among other symptoms; Günaydin et al., 1999; NIAMS, 2010; Rhodus 1989). Furthermore, dry mouth (xerostomia) and dry eyes (keratoconjunctivitis sicca) can be experienced independent of Sjögren’s syndrome and of each other (Günaydin et al., 1999; Rhodus, 1989).

Regarding the prevalence of Sjögren’s syndrome, xerostomia, and keratoconjunctivitis sicca among fibromyalgia syndrome patients, Bonafede et al. (1995) observed Sjögren’s syndrome among 7% of the fibromyalgia syndrome patients in their study whereas Rhodus et al. (2003) and Katz et al. (2007) observed xerostomia among 71 and 63%, respectively, of their respective fibromyalgia syndrome study samples. Furthermore, Dinerman et al. (1986) and Yunus et al. (1993) observed xerostomia among 18 and 12%, respectively, of their respective fibromyalgia syndrome study samples. In addition, Günaydin et al. (1999) observed keratoconjunctivitis sicca among 38% of their fibromyalgia syndrome study sample.
Temporomandibular joint dysfunction refers to an irregularity in the functioning of one or more of the temporomandibular joints (American Dental Association [ADA], n.d.; National Institute of Dental and Craniofacial Research [NIDCR], 2011). The temporomandibular joints (of which there are two in the human body, one located on the left side of the skull and the other located on the right side of the skull) are complex systems of muscles, ligaments, discs, and bones that connect the mandible or lower jaw to the skull and facilitate the following behaviors: speaking, chewing, and yawning (ADA, n.d.; NIDCR, 2011). According to the American Dental Association (n.d.), any irregularity in the functioning of any component (e.g., the ligaments or discs) of either of the two temporomandibular joints can lead to gross TMJ dysfunction. If the functioning of any component of either temporomandibular joint becomes impaired, then the overall functioning of the temporomandibular joints becomes impaired, resulting in the following symptoms: jaw pain (i.e., sensations of pain that originate in the jaw and spread throughout the face and neck), stiff jaw muscles, impaired speaking/chewing/yawning, crepitation (i.e., a crackling sensation or sound that is experienced in the mandible upon movement, such as in the act of speaking or chewing), and restricted jaw movement (i.e., the extent to which the mandible can be lowered is reduced; ADA, n.d.; “Crepitation,” 2011; Hedenberg-Magnusson et al., 1997; NIDCR, 2011). In addition, TMJ dysfunction has been observed among 75 to 29% of FMS study patients (Bennett et al., 2007; Plesh et al., 1996; Rhodus et al., 2003; Wolfe et al., 2005).

Glossodynia or burning mouth syndrome is a condition in which burning or pain sensations are experienced in the tongue (“Glossodynia,” 2009). Dysphagia or swallow disorder is a condition in which the act of swallowing is difficult to perform (National
Institute on Deafness and Other Communication Disorders [NIDCD], 2010). According to the NIDCD (2010), some dysphasic individuals lose the ability to swallow altogether whereas other dysphasic individuals lose particular swallowing abilities (e.g., an individual may lose the ability to swallow food, but retain the ability to swallow liquids). Dysgeusia or taste disorder is a condition that is marked by the development of a taste sensation that is both long-term and qualitatively abnormal (i.e., the newly-developed taste sensation is a taste sensation that is not typically experienced by the individual, such as a metallic, foul, rancid, and/or salty taste sensation; “Dysgeusia – Taste Disorder,” 2010).

A review of the fibromyalgia syndrome literature reveals a lack of research regarding the prevalence of glossodynia, dysphagia, and dysgeusia in the fibromyalgia syndrome population. Nonetheless, Rhodus et al. (2003) observed glossodynia and dysgeusia among 33 and 34%, respectively, of the fibromyalgia syndrome patients in their study. Regarding dysphagia, Katz et al. (2007) and Rhodus et al. (2003) observed dysphagia among 42 and 37%, respectively, of their respective fibromyalgia syndrome study samples.

Subjective Dizziness and Vertigo

A moderate number of fibromyalgia patients tend to experience dizziness and vertigo (Arnold et al., 2008; Bayazit, Gürsoy, Özer, Karakurum, & Madenci, 2002; Waylonis & Heck, 1992). Dizziness refers to the condition wherein an individual experiences both a generally unsteady sensation in the body and a marked sensation of movement in the head (“Dizziness,” 2011); it can be described as a sensation of
lightheadedness and/or lead to a loss of balance (NIDCD, 2011). Vertigo is the condition wherein an individual experiences a motion sensation that is marked by the perception of a whirling self or environment (“Vertigo,” 2011). In the context of fibromyalgia syndrome, the dizziness and vertigo are subjective; although most fibromyalgia syndrome patients reporting dizziness and/or vertigo do not suffer from an audiollogically or clinically detectable ear disease that is characterized by dizziness or vertigo, they do feel dizzy and/or experience vertigo (Bayazit et al., 2002). Bayazit et al. (2002) conducted clinical and laboratory assessments on fibromyalgia patients who reported dizziness and vertigo (among other conditions) as symptoms and failed to discover any objective evidence for the reported symptoms. Nonetheless, for the fibromyalgia patients experiencing dizziness and/or vertigo, activities of daily living (e.g., driving) as well as basic human movements (e.g., walking) can be difficult to perform (Arnold et al., 2008). In addition, subjective dizziness has been observed among 61 to 4% of fibromyalgia syndrome study patients (Bayazit et al., 2002; Katz et al., 2007; Rosenhall, Johansson, & Örndahl, 1996; Tamber & Bruusgaard, 2009) whereas subjective vertigo has been observed among 47 to 8% of fibromyalgia syndrome study patients (Bayazit et al., 2002; Bengtsson et al., 1986a; Rosenhall et al., 1996).

**Headaches**

Headaches are another moderately common symptom of fibromyalgia (Arnold et al., 2008; Bengtsson et al., 1986a; Bennett et al., 2007; Goldenberg, 1987; Jacobsen et. al., 1993; Marcus, Bernstein, & Rudy, 2005; Wolfe et al., 1990; Yunus et al., 1981). In general, a headache is a pain that is experienced within the head. Headaches have been
observed among 76 to 44% of fibromyalgia study patients (Bengtsson et al., 1986a; Bennett et al., 2007; Goldenberg, 1987; Jacobsen et al., 1993; Marcus et al., 2005; Wolfe et al., 1990; Yunus et al., 1981). According to the National Institute of Neurological Disorders and Stroke (NINDS, 2011), the tension headache is the most common type of headache. A tension headache is a headache that occurs on both sides of the head for a variable amount of time; it is characterized by pain of mild-to-moderate intensity, and it typically involves the contraction of the scalp and neck muscles. Another common type of headache is the migraine headache, which is a recurrent and severe unilateral (i.e., occurring on one side of the head) headache. Nausea and vomiting typically occur during a migraine headache; sleep commonly occurs following a migraine headache. In the context of fibromyalgia, the migraine headache seems to be the type of headache that is experienced the most by fibromyalgia patients (Marcus et al., 2005). Among the 76% of fibromyalgia patients experiencing headaches in Marcus et al.’s (2005) study, 84% of the patients indicated that their general functioning (i.e., their physical, social, emotional, and mental functioning) had been severely or substantially impacted by their headaches, and 48% of the patients endorsed the migraine headache as the type of headache that they typically experience (the migraine headache was the most endorsed type of headache in the study). In addition, Hudson, Goldenberg, Pope, Keck, and Schlesinger (1992) interviewed a group of FMS patients and observed current migraine headaches among 45% of the patients; 55% of the patients indicated a lifetime history of migraine headaches. Moreover, the tension headache seems to be the second most commonly experienced type of headache among fibromyalgia patients (Marcus et al., 2005).
Increased Sensory Sensitivity

The FMS literature (e.g., Arnold et al., 2008; Katz et al., 2007) suggests that a moderate number of FMS patients tend to experience increased sensitivity in one or more sensory domains. That is, a moderate number of fibromyalgia patients tend to demonstrate increased skin, light, sound, temperature, and/or odor sensitivity (Arnold et al., 2008; Katz et al., 2007). It is important to note that the fibromyalgia literature is currently void of any explicit research regarding the manifestation of increased light, sound, temperature, or odor sensitivity among FMS patients; increased skin sensitivity (in particular, its manifestation in an FMS patient) seems to be the only type of increased sensory sensitivity that has received attention (albeit minimal attention) in the fibromyalgia literature. Some FMS patients report that when contact occurs between their skin and common household chemicals (e.g., the chemicals in cosmetic products, perfumes, laundry detergents, etc.), their skin tends to react in an “unusual” (p. 119) way (Arnold et al., 2008). Among the “unusual” (Arnold et al., 2008, p. 119) skin reactions that have been reported by FMS patients are a “burning” (Arnold et al., 2008, p. 119) sensation on the skin, a change in skin texture, a change in skin pigmentation, and hives on the skin (Arnold et al., 2008; Katz et al., 2007). Regarding the prevalence of increased skin sensitivity among fibromyalgia syndrome patients, Katz et al. (2007) observed “hives and skin sensitivity” (p. 605) among 43% of the fibromyalgia syndrome patients in their study. In addition, Katz et al. (2007) observed “sensitivity to light” (p. 605) and “sun sensitivity” (p. 605) among 69 and 45%, respectively, of the fibromyalgia syndrome patients in their study.
Sexual Dysfunction

Sexual dysfunction refers to any biological, psychological, or social problem that interferes with an individual’s sexual response to a stimulus of an erotic nature (Da Costa, Kneubil, Leão, and Thé, 2004). A review of the fibromyalgia literature reveals that sexual dysfunction is a common symptom of fibromyalgia syndrome (Aydin et al., 2006; Ryan, Hill, Thwaites, & Dawes, 2008; Shaver et al., 2006; Tikiz et al., 2005). For example, Ryan et al. (2008) observed sexual dysfunction among 63% of the fibromyalgia patients in their study. Nonetheless, only a few studies (e.g., Aydin et al., 2006; Da Costa et al., 2004; Ryan et al., 2008; Shaver et al., 2006; Yunus, Trotter, & Inanici, 1999) have investigated the specific types of sexual dysfunction that are typically experienced by fibromyalgia patients.

One type of sexual dysfunction that is commonly experienced by fibromyalgia patients is decreased sexual desire (Aydin et al., 2006). Decreased sexual desire can include absent or significantly reduced sensations of sexual desire or interest, nonexistent sexual fantasies or thoughts, significantly reduced responsive desire, and/or nonexistent or very few motivations/incentives/reasons for making the effort to become sexually aroused (Basson et al., as cited in Basson, 2007). In their study of fibromyalgia patients, Aydin et al. (2006) observed decreased sexual desire among 63% of the patients. Another type of sexual dysfunction that is commonly experienced by fibromyalgia patients is decreased sexual arousal (Aydin et al., 2006; Shaver et al., 2006). A female experiencing decreased sexual arousal may have difficulty generating sufficient vaginal lubrication for an upcoming sexual activity; a male experiencing decreased sexual arousal may have difficulty achieving or maintaining an erection for an upcoming sexual activity ("Sexual
Arousal Disorder,” 2006). Aydin et al. (2006) and Shaver et al. (2006) each observed decreased sexual arousal among 54% of the fibromyalgia syndrome patients in their respective studies.

Difficulty achieving sexual climax or orgasm is another sexual dysfunction that is common among FMS patients (Aydin et al., 2006, Da Costa et al., 2004; Shaver et al., 2006). In Aydin et al.’s (2006) study, 56% of the FMS participants indicated that they experience difficulty achieving orgasm. In Shaver et al.’s (2006) study, 33% of the FMS participants indicated that they experience difficulty achieving orgasm. A fourth type of sexual dysfunction that is commonly experienced by fibromyalgia patients is decreased sexual satisfaction (Aydin et al., 2006; Ryan et al., 2008; Shaver et al., 2006; Yunus, Trotter, et al., 1999). Researchers (e.g., Ryan et al., 2008) have identified two factors that seem to significantly contribute to decreased sexual satisfaction among FMS patients: exacerbated pain sensations that are experienced during sexual intercourse (Shaver et al., 2006) and general fatigue (which can diminish a patient’s physiological energy level for sexual activity; Ryan et al., 2008). Aydin et al. (2006) observed decreased sexual satisfaction among 48% of the FMS patients in their study. Moreover, Shaver et al. (2006) observed a reduction in the frequency of sexual intercourse and a reduction in the frequency of self-pleasuring or masturbation among 66 and 30%, respectively, of the fibromyalgia syndrome patients in their study.

**Psychological Distress**

A moderate number of fibromyalgia patients tend to experience psychological distress (Bennett, 2009). In particular, anxiety and depression tend to be the forms of
psychological distress that are the most frequently experienced by FMS patients (Arnold et al., 2008). Researchers (e.g., Sayar, Gulec, Topbas, & Kalyoncu, 2004) have proposed the idea that for some fibromyalgia patients, anxiety can exacerbate other symptoms such as pain and muscle tension by altering perceptions of symptom severity. For example, if an FMS patient experiences a flare during an episode of anxiety, then the patient’s abnormal and exaggerated sense of fear will likely influence the patient’s perception of the flare in an adverse manner, such that the patient will likely perceive the flare as significantly more intense and unsettling than usual (Sayar et al., 2004). Moreover, anxiety has been observed among 76 to 38% of fibromyalgia syndrome study patients (Bengtsson et al., 1986a; Bennett et al., 2007; Katz et al., 2007; Martinez, Ferraz, Fontana, & Atra, 1995; Wolfe et al., 1990; Yunus et al., 1989; Yunus et al., 1981). Furthermore, anxiety disorders such as simple phobia, social phobia, generalized anxiety disorder (GAD), panic disorder with and without agoraphobia, post-traumatic stress disorder (PTSD), and obsessive-compulsive disorder (OCD) have been observed among 60 to 27% of FMS study patients (Aaron et al., 1996; Arnold et al., 2006; Epstein et al., 1999; Thieme, Turk, & Flor, 2004).

Regarding depression, in the context of fibromyalgia syndrome it tends to be marked by feelings of shame or guilt as well as isolation (Arnold et al., 2008). Some FMS patients report that they tend to feel guilty when they have to rearrange their plans such that their health care takes precedence over social or familial commitments (Arnold et al., 2008). FMS patients also tend to report that they feel physically lonely on account of a decline in social invitations from both friends and family members (‘. . . you’re unreliable . . . that’s how I feel, and that is actually how you present yourself, and you
lose your friends” [p. 118]) as well as emotionally lonely on account of non-empathic / non-supportive family members and friends (“I’ve had . . . a family member say to me, ‘I think you like to be sick’ ” [p. 118]; Arnold et al., 2008). In addition, researchers (e.g., Epstein, Williams, Osbeck, & Clauw, 1995; Hawley, Wolfe, & Cathey, 1988) have indicated that depression has the potential to exacerbate the severity of fibromyalgia syndrome; however, the dynamics of the relationship between depression and FMS severity are currently not fully understood, although alterations in pain tolerance and threshold have been identified as potential mediating factors.

Depression has been observed among 80 to 29% of FMS study patients (Ahles, Yunus, & Masi, 1987; Bennett et al., 2007; Ercolani et al., 1994; Martinez et al., 1995; Yunus et al., 1989). Furthermore, mood disorders such as major depression, major depression – recurrent episode, dysthymia, and bipolar disorder have been observed among 35 to 29% of FMS study patients (Aaron et al., 1996; Epstein et al., 1999; Thieme et al., 2004).
CHAPTER 3
THE DIAGNOSTIC CRITERIA FOR FIBROMYALGIA SYNDROME

In 2010, the American College of Rheumatology (ACR) published a new set of criteria for the diagnosis of fibromyalgia syndrome (Wolfe et al., 2010). The creation of the new set of diagnostic criteria was prompted in response to research wherein it was observed that the 1990 ACR diagnostic criteria for fibromyalgia were rarely being employed correctly (Buskila, Neumann, Sibirski, & Shvartzman, 1997; Fitzcharles & Boulos, 2003). Further support for the creation of new diagnostic criteria was derived from relatively recent research wherein the symptomatology of FMS was clarified (Choy et al., 2009; Mease et al., 2007). The 2010 ACR diagnostic criteria is expected to become the premier criteria for an FMS diagnosis in the near future; however, at present researchers (e.g., Wolfe et al., 2011) recommend that physicians employ either the 2010 ACR criteria or the 1990 ACR criteria when diagnosing fibromyalgia syndrome. What follows is a review of both sets of criteria.

According to the 2010 ACR diagnostic criteria for fibromyalgia, a diagnosis of fibromyalgia is warranted if the following three conditions are satisfied: 1. the patient reports pain in 7 or more of 19 particular body areas (termed the widespread pain index or WPI) for over the last week as well as a symptom severity (SS) scale score of 5 or more OR the patient reports pain in 3-to-6 of 19 particular body areas as well as an SS scale score of 9 or more, 2. the patient has been experiencing symptoms for at least 3 months and at a generally consistent level, and 3. the patient’s symptomatology cannot be explained by the presence of another disorder (Wolfe et al., 2010).
The WPI includes the following body areas: the left and right shoulder girdle, the left and right upper arm, the left and right lower arm, the left and right hip/buttock, the left and right upper leg, the left and right lower leg, the left and right jaw, the upper and lower back, the neck, the chest, and the abdomen (Wolfe et al., 2010). The SS scale score is calculated by summing the level of severity of 3 common FMS symptoms (fatigue, cognitive symptoms, and nonrestorative sleep) and the general level of severity of the patient’s somatic symptoms; the overall score ranges from 0 to 12 (Wolfe et al., 2010). To elaborate, for each of the 3 common symptoms listed above, the patient is asked to describe the level of severity of the symptom for the past week using the following scale: 0 = no problem or difficulty; 1 = slight/mild problem or generally mild/intermittent difficulty; 2 = moderate/considerable problem or difficulty that is often present and/or at a moderate level; and 3 = severe problem or pervasive, continuous, and life-disturbing difficulty (Wolfe et al., 2010). Regarding the patient’s somatic symptoms, they can include muscle pain, headache, dizziness, Raynaud’s phenomenon, and paresthesias (among many others, most of which are listed earlier in this manual), and the patient is asked to indicate the general (i.e., past and current) presence of somatic symptoms using the following scale: 0 = no symptoms; 1 = a few symptoms; 2 = a moderate number of symptoms; and 3 = many or a significant number of symptoms (Wolfe et al., 2010).

According to the 1990 ACR diagnostic criteria for fibromyalgia, a diagnosis of fibromyalgia is warranted if two criterions are satisfied (Wolfe, et al., 1990). The first criterion is a history of widespread pain (Wolfe et al., 1990). Widespread pain refers to pain in all four quadrants of the body as well as in at least one structure of the axial skeletal system (Wolfe et al., 1990). According to Wolfe et al. (1990), the four quadrants
of the body are the right and left side of the body as well as above and below the waist. The axial skeletal system refers to the cervical spine, anterior chest, thoracic spine, and lower back (Wolfe et al., 1990). Moreover, the onset of the widespread pain must be at least three months prior to the date of diagnosis (Wolfe et al., 1990).

The second criterion is that digital palpation results in the experience of pain at a minimum of eleven tender point sites (“Fibromyalgia Tender Points,” 2008; Wolfe et al., 1990). A tender point site refers to a location on the body that is sensitive to pressure, such that pain is experienced by a patient when the sensitive location is pressed (“Fibromyalgia Tender Points,” 2008). Digital palpation, which refers to a physician’s application of pressure to a tender point site with his or her thumb pad, represents the type of pressure to which a tender point site is highly sensitive (“Fibromyalgia Tender Points,” 2008; Sharpe & O’Malley, 2005; Wolfe et al., 1990). Furthermore, the application of pressure to a tender point site can result in the experience of pain in the area surrounding the tender point site (“Fibromyalgia Tender Points,” 2008). For example, the application of pressure to a tender point site on a patient’s shoulder blade can result in widespread back pain (“Fibromyalgia Tender Points,” 2008).

There are nine pairs of tender point sites (eighteen tender point sites total) located on the surface of the human body (Wolfe et al., 1990). Specifically, five pairs of tender point sites are located on the back of the human body, whereas the other four pairs of tender point sites are located on the front of the human body (“Fibromyalgia Tender Points,” 2008). Each pair of tender point sites is bilateral (“Fibromyalgia Tender Points,” 2008). In other words, for any given pair of tender point sites, one site will be at a
particular location on the left side of the body and the other site will be at the same location, but on the right side of the body (“Fibromyalgia Tender Points,” 2008).

The “back” pairs of tender point sites are located on the back of the neck (there is a site behind each ear, at the location where the muscles of the neck attach to the base of the skull); midway between the base of the neck and the tip of each shoulder; on the back (there is a site on each shoulder blade, where the back muscles connect to the shoulder blades); on the buttocks (there is a site on the upper region of each buttock); and on the upper legs (there is a site on the back of each thigh, close to the bottom of the hip; “Fibromyalgia Tender Points,” 2008). Regarding the “front” pairs of tender point sites, they are located on the front of the neck (there is a site on the left side as well as on the right side of the front of the neck, just above the collarbone); to the left and right of the sternum or breastbone, and approximately two inches below the collarbone; on each forearm (specifically, each site is immediately below and towards the outside of the crease of each elbow), and on each knee (specifically, each site is above the kneecap and towards the inside of each knee; “Fibromyalgia Tender Points,” 2008).

Digital palpation refers to a physician’s application of pressure with his or her thumb pad to each of a patient’s eighteen tender point sites; pressure is applied to each site at an increasing rate of 1 kg/second, until 4 kg of pressure is applied (which is indicated by the whitening of the physician’s thumbnail bed; Sharpe & O’Malley, 2005; Wolfe et al., 1990). As such, a tender point site is classified as “positive” for fibromyalgia when palpation at the site is experienced and described as “painful” by the patient (Wolfe et al., 1990). Furthermore, if palpation at a tender point site is experienced and described as “tender” by the patient, then the site is not classified as “positive” for
fibromyalgia (Wolfe et al., 1990). In accordance with the ACR criteria, “tender” is not synonymous with “painful” and therefore, does not warrant the same classification as “painful” (Wolfe et al., 1990).

There is a third set of diagnostic criteria for FMS that was developed in 1981 by Yunus et al., in which the criteria are grouped into the following three categories: Obligatory, Major, and Minor Criteria. The Obligatory Criteria include widespread aches and pain or pronounced stiffness at three or more anatomic sites, in which the duration of pain or stiffness is at least three months and neither pain nor stiffness is attributable to secondary sources such as trauma or infection (Yunus et al., 1981). The Major Criteria include experiences of pain at a minimum of five tender point sites (Yunus et al., 1981). It is important to note that in this diagnostic system, a tender point site refers to any area on the surface of the body where the application of pressure to that area results in the experience of pain at that area (Yunus et al., 1981). Moreover, the Minor Criteria include exacerbation of symptoms due to anxiety or stress; sleep difficulties; general tiredness or fatigue; anxiety; chronic headaches; irritable bowel syndrome (IBS); swelling; numbness; and modulation (i.e., moderate exacerbation and/or reduction) of symptoms due to physical activity as well as weather conditions (Yunus et al., 1981). As such, an individual is diagnosed with FMS when the individual satisfies the Obligatory Criteria, the Major Criteria, and a minimum of three Minor Criteria (Yunus et al., 1981).
CHAPTER 4

THE EPIDEMIOLOGY OF FIBROMYALGIA SYNDROME
IN THE UNITED STATES

According to Wolfe et al. (1995), approximately 2% of the general U.S. adult population meets the criteria for a diagnosis of fibromyalgia syndrome. As such, approximately 5,000,000 adults in the United States experience FMS (Lawrence et al., 2008). Fibromyalgia is prevalent among 3.4% of U.S. adult females and 0.5% of U.S. adult males (Wolfe et al., 1995). According to Sharpe and O’Malley (2005), the onset of FMS typically occurs between the ages of 30 and 50 years. Childhood and geriatric onset are also possible and have been documented in the literature (e.g., Romano, 1991; Wolfe et al., 1995). Furthermore, the literature (e.g., Wolfe et al., 1995) indicates that the prevalence of FMS increases as the age of the adult population increases. Moreover, a review of the literature reveals that there is a lack of research regarding racial/ethnic differences in the prevalence of FMS in the United States.

Fibromyalgia syndrome patients in the United States tend to be of low socioeconomic status or SES (in which the average household income for an FMS patient tends to be $20,900) as well as less educated (i.e., they tend to have less than a twelfth grade education; Wolfe et al., 1995). The unemployment rate for FMS patients ranges from approximately 28% (Bombardier & Buchwald, 1996) to approximately 64% (Assefi, Coy, Uslan, Smith, and Buchwald, 2003). Furthermore, the literature (e.g., Assefi et al., 2003) indicates that approximately 45% of FMS patients receive a social security disability income.
A seven-year study on medical service utilization and medical costs among fibromyalgia patients revealed that they tend to visit medical professionals about ten times a year, undergo a radiographic examination about once a year, and undergo laboratory examinations about three times a year (Wolfe et al., 1997). Furthermore, researchers (e.g., Wolfe et al., 1997) have observed that FMS patients tend to be hospitalized at a rate of one hospitalization every three years for reasons that include neurological, cardiovascular, gastrointestinal, depressive, and genitourinary problems. In addition, fibromyalgia syndrome tends to cost a patient approximately $2,274 annually in terms of medical expenses (Wolfe et al., 1997). Medical expenses include hospitalization, medications, outpatient services, and other medical services such as laboratory and radiologic testing (Wolfe et al., 1997).
CHAPTER 5
THE MOST COMMON COMORBID DISORDERS
OF FIBROMYALGIA SYNDROME

A review of the fibromyalgia syndrome literature reveals that chronic fatigue syndrome (CFS), irritable bowel syndrome (IBS), and multiple chemical sensitivity syndrome (MCSS) are three of the most common comorbid disorders of fibromyalgia syndrome (Wallace, 1997; see Table 2).

Table 2

Lowest and highest prevalence rates of associated disorders among FMS study populations

<table>
<thead>
<tr>
<th>Associated Disorder</th>
<th>Lowest Prevalence Rate</th>
<th>Highest Prevalence Rate</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chronic fatigue syndrome</td>
<td>18% (Aaron et al., 2000)</td>
<td>70% (Buchwald &amp; Garrity, 1994)</td>
</tr>
<tr>
<td>Irritable bowel syndrome</td>
<td>32% (Sperber et al., 1999)</td>
<td>70% (Veale et al., 1991)</td>
</tr>
<tr>
<td>Multiple chemical sensitivity syndrome</td>
<td>N/A</td>
<td>55% (Buchwald &amp; Garrity, 1997)</td>
</tr>
</tbody>
</table>

Chronic fatigue syndrome is a disorder of unknown etiology that is marked by constant and debilitating fatigue (Holmes et al., 1988). The duration of each occurrence of fatigue tends to be at least six months (Holmes et al., 1988). Unresolved by any form of sleep or rest, the fatigue tends to severely impair an individual’s ability to engage in activities of daily living, such that the individual’s daily activity level tends to be reduced by 50% (from premorbid baseline; Holmes et al., 1988). Furthermore, in order to receive a CFS diagnosis an individual’s history must be void of any similar type of chronic and/or debilitating fatigue (Holmes et al., 1988).
The typical age of onset for chronic fatigue syndrome is approximately 30 years of age (Chronic Fatigue Syndrome, 2011). Additional symptoms of CFS include frequent sore throat; painful or tender lymph nodes; muscle pain; multi-joint pain without redness or swelling; post-exertional malaise (which refers to extreme levels of exhaustion and sickness that occur in response to physical or mental activity and typically last for more than 24 hours); new headaches (in terms of pattern, severity, or type); nonrestorative sleep; and self-reported impaired short-term memory ability and/or concentration ability (which causes marked reductions in occupational, social, educational, and/or personal functioning; Centers for Disease Control and Prevention [CDC], 2010; Fukuda et al., 1994; Holmes et al., 1988; Komaroff et al., 1996). CFS has been observed among 70 to 18% of FMS study patients (Aaron, Burke, & Buchwald, 2000; Buchwald & Garrity, 1994; Hudson et al., 1992).

Irritable bowel syndrome is a functional disorder of the colon that is chronic and of unknown etiology (“Irritable Bowel Syndrome,” 2011; National Institute of Diabetes and Digestive and Kidney Diseases [NIDDK], 2011). The symptoms of IBS include abdominal pain or discomfort during defecation or in response to a change in bowel habit (“Irritable Bowel Syndrome,” 2011; Manning, Thompson, Heaton, & Morris, 1978; Thompson et al., 1999); irregular stool frequency (e.g., more than three bowel movements a day or less than three bowel movements a week; Thompson et al., 1999); atypical stool form (e.g., hard/lumpy stool or watery/loose stool; “Irritable Bowel Syndrome,” 2011; Thompson et al., 1999); irregular stool passage (i.e., the passage of stool is marked by a sensation of strain, a feeling of urgency, or a sensation of incomplete or unfinished evacuation; “Irritable Bowel Syndrome,” 2011; Manning et al., 1978;
Thompson et al., 1999); the passage of mucus in stool (“Irritable Bowel Syndrome,” 2011; Manning et al., 1978; Thompson et al., 1999); and abdominal distension or the sensation of abdominal swelling or bloating (“Irritable Bowel Syndrome,” 2011; Thompson et al., 1999). It is important to note that although the functioning of the colon is altered by IBS, the colon itself is not damaged by the disorder (NIDDK, 2011).

Irritable bowel syndrome has been observed among 70 to 32% of fibromyalgia syndrome study patients (Aaron et al., 2000; Bengtsson et al., 1986a; Bennett et al., 2007; Campbell et al., 1983; Goldberg, 1987; Hudson et al., 1992; Kurland, Coyle, Winkler, & Zable, 2006; Nishikai, 1992; Romano, 1988; Sperber et al., 1999; Veale, Kavanagh, Fielding, & Fitzgerald, 1991; Wolfe et al., 1995; Wolfe et al., 1990; Yunus et al., 1989; Yunus et al., 1981).

Multiple chemical sensitivity syndrome is a chronic disorder of unknown etiology wherein exposure to multiple chemical compounds at low levels leads to the development of multiple symptoms in multiple organ systems (Buchwald & Garrity, 1994; Consensus on Multiple Chemical Sensitivity, 1999; Council on Scientific Affairs – American Medical Association [AMA], 1992; Nethercott, Davidoff, & Curbow, 1993; Slotkoff, Radulovic, & Clauw, 1997; Saito et al., 2005). Examples of chemical compounds to which many MCSS patients tend to be susceptible include the following common and chemically unrelated compounds: solvents such as paint thinner and kerosene; pesticides; environmental compounds such as smoke, new carpet, perfume, food, office machines, and ammonium compounds; metals such as nickel and lead; and dusts such as wood dust and beet sugar dust (Nethercott et al., 1993; Terr, 1989). Furthermore, the low level of chemical exposure that tends to be toxic for the MCSS patient is a level that is well
tolerated by members of the general population as well as well below the established level of toxicity (Buchwald & Garrity, 1994). Regarding the symptoms of multiple chemical sensitivity syndrome, they tend to occur in the respiratory, gastrointestinal, musculoskeletal, dermatologic, and central nervous systems (Slotkoff et al., 1997). Examples of common MCSS symptoms include hoarseness / trouble speaking, burning sensations in the nose and/or mouth, sensations of shortness of breath, itchy and/or irritated skin, fatigue, fever, headache, insomnia, depression, and irritability (Buchwald & Garrity, 1994; Council on Scientific Affairs- AMA, 1992). In addition, although a recent review of the literature revealed a paucity of research regarding the prevalence of MCSS among FMS patients, one study (Buchwald & Garrity, 1997) was found wherein MCSS was observed among 55% of the FMS study sample.
CHAPTER 6
THE ETIOLOGY OF FIBROMYALGIA SYNDROME

The exact etiology of fibromyalgia syndrome is currently unknown (Chakrabarty & Zoorob, 2007; Sharpe & O’Malley, 2005). Among the etiological models that have been proposed in the fibromyalgia literature, the biopsychosocial etiological model has been garnering the most support over the last decade (Bradley & Alarcón, 2005; Dadabhoy & Clauw, 2008; Sharpe & O’Malley, 2005). The biopsychosocial etiological model of fibromyalgia suggests that three sets of factors are responsible for the development and maintenance of fibromyalgia (Dadabhoy & Clauw, 2008; Sharpe & O’Malley, 2005). Sharpe and O’Malley (2005) have labeled them predisposing factors, precipitating factors, and perpetuating factors. Predisposing factors influence an individual into developing fibromyalgia syndrome, whereas precipitating factors trigger the onset of fibromyalgia syndrome (Sharpe & O’Malley, 2005). Perpetuating factors sustain fibromyalgia syndrome (Sharpe & O’Malley, 2005). Furthermore, each set of etiological factors is comprised of a unique combination of biological, psychological, and/or social factors (Sharpe & O’Malley, 2005).

Predisposing Biological Factors

Researchers (e.g., Arnold et al., 2004; Buskila & Neumann, 1997; Buskila, Neumann, Hazanov, & Carmi, 1996; Yunus, Khan, et al., 1999) have identified genetics as the predominant predisposing biological factor of fibromyalgia syndrome. Buskila et al. (1996) and Buskila and Neumann (1997) observed fibromyalgia among 28 and 26%, respectively, of the blood relatives of the fibromyalgia patients in their respective studies.
Given that the prevalence of fibromyalgia in the general population is approximately 2% (Wolfe et al., 1995), the increased prevalence of fibromyalgia among blood relatives of fibromyalgia patients seems to indicate that there is a genetic factor involved in the development of fibromyalgia (Buskila & Neumann, 2005; Buskila et al., 1996). Moreover, fibromyalgia researchers are currently investigating the possible role of chromosome 6 (Yunus et al., 1999), as well as the possible role of genetic polymorphisms (which are benign and normal genetic changes that can impact the functioning of neurotransmitters that are involved in the stress response system, such as serotonin and dopamine; Buskila & Neumann, 2005, Dadabhoy & Clauw, 2008; A. T. Williams & Northrup, 2005), in predisposing an individual to developing fibromyalgia.

**Predisposing Psychological Factors**

Personality (Van Houdenhove, Neerinckx, Ongena, et al., 2001) and victimization (Van Houdenhove, Neerinckx, Lysens, et al., 2001; Walker et al., 1997) have been identified in the fibromyalgia literature as the predominant psychological factors that predispose an individual to developing fibromyalgia. Regarding the personality factor, research has suggested that the personality trait of high “action-proneness” predisposes an individual to developing fibromyalgia syndrome (Van Houdenhove, Neerinckx, Ongena, et al., 2001). An individual who is highly “action-prone” will likely be strong-willed, passionate, driven, and energetic; consequently, the individual will likely live an “overactive” lifestyle, which is a lifestyle that is marked by perfectionism, “workaholism” (pp. 574), high achievement motivation, and self-sacrificing tendencies (Van Houdenhove, Neerinckx, Lysens, et al., 2001). It has been
suggested that living an “overactive” lifestyle for an extended period of time will likely lead to musculoskeletal overuse, sleep deprivation, and/or the development of a negligent attitude towards physical and psychological self-care, which in turn, will likely contribute to the onset of fibromyalgia syndrome (Van Houdenhove, Neerinckx, Lysens, et al., 2001).

Victimization refers to the experience of feeling powerless and/or helpless in response to a long-term situation of violence, neglect, unpredictability, chaos, and/or insufficient family limits (Van Houdenhove, Neerinckx, Lysens, et al., 2001). In the context of FMS, victimization on account of childhood and/or adult physical abuse (Boisset-Pioro, Esdaile, & Fitzcharles, 1995; Haviland, Morton, Oda, & Fraser, 2010; Van Houdenhove, Neerinckx, Lysens, et al., 2001; Walker et al., 1997) and victimization on account of childhood and/or adult emotional abuse and neglect (Van Houdenhove, Neerinckx, Lysens, et al., 2001) have been identified as predisposing psychological factors. Regarding victimization on account of childhood and/or adult sexual abuse, research findings are mixed with some researchers (e.g., Haviland et al., 2010) identifying it as a predisposing psychological factor for fibromyalgia and other researchers (e.g., Boisset-Pioro et al., 1995; Taylor, Trotter, & Csuka, 1995) determining that it is not such a factor.

Researchers (e.g., Haviland et al., 2010; Van Houdenhove, Neerinckx, Lysens, et al., 2001) have posited that for some victims of physical abuse, emotional abuse, sexual abuse, and/or emotional neglect, the impact of the abuse and/or neglect can be long-term and involve the victims’ physiological and psychosocial functioning, eventually leading to the development of fibromyalgia. A victim of physical abuse, emotional abuse, sexual
abuse, and/or emotional neglect may develop physiological symptoms such as chronic sleep difficulties and chronic muscle hypertension (Van Houdenhove, Neerinckx, Lysens, et al., 2001) as well as psychosocial symptoms such as poor self-esteem (Van Houdenhove, Neerinckx, Lysens, et al., 2001), negative affectivity or a predisposition to typically experience a mood that is marked by anger, anxiety, and/or depression (Van Houdenhove, Neerinckx, Lysens, et al., 2001; Watson & Pennebaker, 1989), and a maladaptive attachment style, which is a pattern of inflexible behavior that is marked by hypersensitivity to real or perceived threats as well as persistent attempts at eliciting care from other people (Schofferman, Anderson, Hines, Smith, & Keane, 1993; Stuart & Noyes, 1999). Over time, these chronic symptoms may weaken the individual’s stress response system, such that the individual’s ability to physiologically, emotionally, and cognitively tolerate stress becomes very limited, which in turn, may contribute to the individual developing fibromyalgia syndrome (Demitrack & Crofford, 1998; Heim, Ehlert, & Hellhammer, 2000; Teicher, Glod, Surrey, & Swett, 1993; Van Houdenhove, Neerinckx, Lysens, et al., 2001; Watson & Pennebaker, 1989).

**Predisposing Social Factors**

Researchers (e.g., Sharpe & O’Malley, 2005) have identified low SES and a low level of education as the predominant social factors that predispose an individual to developing FMS. In several studies (e.g., White, Speechley, Harth, & Østbye, 1999; Wolfe et al., 1995), FMS has been observed to be associated with decreased household income, which is indicative of lower SES, and failure to graduate from high school / less than a high school education. It has been posited in the fibromyalgia literature that for
certain individuals, chronic exposure to the type of stress that is associated with a lifestyle marked by low SES and/or a low level of education (which can include particular types of financial, familial, physical, cognitive, and emotional stress) may reduce the overall strength of their stress response systems, which in turn, may contribute to the individuals developing fibromyalgia (Dadabhoy & Clauw, 2008). It is important to note that the above-stated position is a recent position in the fibromyalgia literature and as such, awaits proper elucidation.

Precipitating Biological Factors

Infection (Dadabhoy & Clauw, 2008; Sharpe & O’Malley, 2005) and physical trauma (Bradley & Alarcón, 2005; Dadabhoy & Clauw, 2008; Sharpe & O’Malley, 2005) have been identified in the fibromyalgia literature as the predominant biological factors that precipitate or trigger the onset of fibromyalgia. Regarding infection, many FMS patients (e.g., approximately 10-55% of FMS study patients) tend to report that prior to the onset of the syndrome they had a viral infection (Buchwald, Goldenberg, Sullivan, & Komaroff, 1987; Goldenberg, 1988; Goldenberg, Simms, Geiger, & Komaroff, 1990; Hsu, Patella, & Sigal, 1993). The following viruses have been identified in the fibromyalgia literature as possible triggers for the onset of fibromyalgia: Borrelia burgdorferi (which causes Lyme disease; Dinerman & Steere, 1992; Sigal, 1990), human immunodeficiency virus type I (HIV; Buskila, Gladman, Langevitz, Urowitz, & Smythe, 1990; Simms et al., 1992), hepatitis C virus (HCV; Buskila et al., 1997; Rivera, Diego, Trinchet, & Monforte, 1997), human parvovirus B19 (Leventhal, Naides, & Freundlich, 1991), Coxsackie B2 virus (Nash, Chard, & Hazleman, 1989), and human herpesvirus-6
Several researchers (e.g., Duna & Wilke, 1993; Goldenberg, 1993; Moldofsky, 1995) have postulated that infections trigger fibromyalgia through biopsychosocial mechanisms. To elaborate, it has been postulated that infections alter cytokine (i.e., immunoregulatory chemical) levels, neurohormonal (e.g., serotonin) levels, and growth hormone levels, as well as prompt an individual to start engaging in maladaptive health behaviors such as decreased exercise and avoidance of activities of daily living (which in turn, alter neurohormonal levels; American Cancer Society [ACS], 2011; American Geriatric Society [AGS] Foundation for Health in Aging, 2005; Duna & Wilke, 1993; Goldenberg, 1993; Moldofsky, 1995; Russell, 1989). Consequently, neuroendocrine and immune system functioning become impaired, which in turn, triggers the onset of various FMS symptoms such as musculoskeletal pain, impaired sleep, fatigue, and cognitive dysfunction (ACS, 2011; AGS Foundation for Health in Aging, 2005; Duna & Wilke, 1993; Goldenberg, 1993; Moldofsky, 1995; Russell, 1989). Although the above-stated biopsychosocial postulation warrants further research, it is commensurate with the leading etiological model of fibromyalgia syndrome.

Regarding physical trauma, many FMS patients (e.g., approximately 7-100% of FMS study patients) tend to report that prior to the onset of FMS they experienced a physically traumatic event (Aaron et al., 1997; Al-Allaf et al., 2002; Buskila, Neumann, Vaisberg, Alkalay, & Wolfe, 1997; Greenfield, Fitzcharles, & Esdaile, 1992; Magnússon, 1994; Moldofsky, Wong, & Lue, 1993; Romano, 1990; Saskin, Moldofsky, & Lue, 1986; Waylonis & Perkins, 1994). Commonly reported physically traumatic events include motor vehicle accidents and work-related accidents, wherein fractures, neck injuries,
back injuries, and/or shoulder injuries occur, as well as surgeries such as spinal surgery, cranial surgery, knee surgery, lung surgery, and childbirth (Aaron et al., 1997; Al-Allaf et al., 2002; Greenfield et al., 1992; Moldofsky et al., 1993; Romano, 1990; Saskin et al., 1986; Waylonis & Perkins, 1994). Several researchers (e.g., Turk, Okifuji, Starz, & Sinclair, 1996) have suggested that a physically traumatic event has the potential to trigger FMS through alterations of an individual’s pain processing system. Exposure to physical trauma may lead to subtle changes in an individual’s physiology (which in turn, can lead to significantly increased physiological sensitivity to pain; Lee, Giles, & Drummond, 1993) and/or marked changes in the individual’s psychological processing of pain (i.e., the individual may become preoccupied with sensations of pain and/or start experiencing increased anticipation of an experience of pain; Hodge, 1971; Turk et al., 1996). These alterations in the pain processing system may consequently lead to an increased focus on physical sensations as well as frequent incidents wherein relatively benign sensory stimuli are interpreted as pain sensations, all of which in turn, may increase the likelihood of FMS onset (Turk et al., 1996).

**Precipitating Psychological Factors**

Emotional trauma has been identified in the fibromyalgia syndrome literature as the predominant psychological factor that precipitates fibromyalgia syndrome (Bradley & Alarcón, 2005; Dadabhoy & Clauw, 2008; Sharpe & O’Malley, 2005). A moderate number of fibromyalgia patients (e.g., approximately 14-39% of fibromyalgia study patients) tend to report that prior to the onset of fibromyalgia they experienced an emotionally traumatic event (Aaron et al., 1997; Goldenberg, 1993). Commonly reported
emotionally traumatic events include financial difficulties, conflicts with spouses/partners, movement to a new place of residence, the death of a loved one (e.g., a close relative or a close friend), marital separation, and the occurrence of a disease or an accident in the life of a close relative (Anderberg, Marteinsdottir, Theorell, & Von Knorring, 2000). Researchers (e.g., Anderberg et al., 2000) have suggested that an emotional trauma has the potential to trigger fibromyalgia through personality mechanisms.

Regarding personality mechanisms, research has revealed that many FMS patients tend to demonstrate a personality type that is marked by a high level of harm avoidance (Anderberg, Forsgren, Ekselius, Marteinsdottir, & Hallman, 1999). According to Cloninger (1987), harm avoidance refers to “a heritable tendency to respond intensely to signals of aversive stimuli” (pp. 575), such that an individual tends to learn to inhibit his or her behavior so as to avoid experiences that are perceived as stressful or harmful. As such, it has been suggested that these individuals will likely respond to an emotionally traumatic event with pessimistic thoughts, intense anticipation of future harm, anxiety / a sense of unease, passivity, and withdrawal behavior, all of which will likely lead to the experience of marked fatigue as well as the onset of fibromyalgia (Anderberg et al., 1999; Anderberg et al., 2000; Cloninger, 1987; Davis, Zautra, & Reich, 2001).

**Perpetuating Biological Factors**

Physiological deconditioning, abnormal sleep, reduced level of cortisol, and dysfunctional central pain processing have been identified in the FMS literature (e.g., Sharpe & O’Malley, 2005) as the predominant biological factors that perpetuate FMS.
Physiological deconditioning is a progressive phenomenon that can occur in many patients, including fibromyalgia patients (Sharpe & O’Malley, 2005). In physiological deconditioning, a patient starts to avoid engagement in physical activity to prevent an increase in symptom frequency as well as intensity; the patient’s increased avoidance of physical activity is because of recent experiences wherein engagement in physical activity led to exacerbations in symptomatology (Bazelmans, Bleijenberg, Van Der Meer, & Folgering, 2001; Wessely, David, Butler, & Chalder, 1989). Over time, the patient’s decreased physical activity leads to the development of decreased physical fitness, such that engagement in lower levels of physical activity (e.g., those activities of daily living that the patient once performed without incident) tends to result in increased symptom frequency and intensity; consequently, the patient tends to stop engaging in many activities of daily living, for the activities have become significantly difficult or impossible to perform (Bazelmans et al., 2001; Wessely et al., 1989). In the context of FMS, decreased physical fitness on account of physiological deconditioning tends to be marked by maladaptive changes in muscle metabolism (Bengtsson, Henriksson, & Larsson, 1986c; Lindh, Johansson, Hedberg, Henning, & Grimby, 1995; Lund, Bengtsson, & Thorborg, 1986) as well as maladaptive changes in muscle morphology (Awad, 1973; Bengtsson et al., 1986b; Brendstrup, Jespersen, & Asboe-Hansen, 1957; Kalyan-Raman, Kalyan-Raman, Yunus, & Masi, 1984). Moreover, it has been suggested in the FMS literature (e.g., Bengtsson et al., 1986b; Bengtsson et al., 1986c) that the changes in muscle metabolism and morphology that occur in FMS patients on account of physiological deconditioning are responsible for the progressive FMS symptoms of fatigue and muscle pain.
Abnormal sleep, as previously indicated, is a common symptom of many fibromyalgia syndrome patients (Wolfe et al., 1990). Many fibromyalgia patients report the following sleep abnormalities: sleep initiation and maintenance difficulties (e.g., Bengtsson et al., 1986a); nonrestorative sleep and early awakening (e.g., Campbell et al., 1983); and sleep disorders such as sleep apnea, restless legs syndrome, and upper-airway resistance syndrome (e.g., Bennett, 2009). Furthermore, several researchers (e.g., Agnew, Webb, & Williams, 1967; Chase & Murphy, 1973; Moldofsky et al., 1975; Smythe & Moldofsky, 1977) have suggested that when stage four of NREM sleep is disturbed on account of a stressful life event (e.g., a minor automobile accident) or a sleep abnormality, then overall serotonin metabolism tends to be disrupted (for serotonin metabolism typically occurs during NREM sleep), which in turn, tends to exacerbate / perpetuate particular FMS symptoms such as pain and fatigue (for serotonin is involved in pain perception and fatigue). It is important to note that the role of abnormal sleep in perpetuating fibromyalgia syndrome is yet another topic that is currently not fully understood (Sharpe & O’Malley, 2005). For a more detailed discussion regarding the stages of sleep, see the symptomatology section above.

Cortisol is a hormone that is released into the bloodstream by the adrenal cortex in response to physical as well as psychological stress ("Cortisol," 2012). When an individual experiences physical or psychological stress, the individual’s stress response system is activated, which involves the interdependent functioning of the individual’s hippocampus, hypothalamus, pituitary gland, and adrenal cortex (Parker, Wessely, & Cleare, 2001). Given the particular neuroanatomical structures involved, the human stress response system is commonly referred to as the hypothalamic-pituitary-adrenal (HPA)
It is the explicit function of the HPA axis to regulate the release of cortisol (Parker et al., 2001). Among the many functions of cortisol is that of an anti-inflammatory agent, naturally reducing tissue swelling and pain (“Hydrocortisone Injection,” 2012; NIDDK, 2009). Detailed discussions regarding the mechanisms of the HPA axis and the functions of cortisol can be found in Parker et al. (2001) and NIDDK (2009), respectively.

In the context of fibromyalgia syndrome, several researchers (e.g., Crofford et al., 1994; McCain & Tilbe, 1989; Van Denderen, Boersma, Zeinstra, Hollander, & Van Neerbos, 1992) have investigated the cortisol levels of fibromyalgia patients and observed reduced levels of cortisol in many of the fibromyalgia patients in their respective studies. In addition, Griep, Boersma, and De Kloet (1993) observed relatively impaired functioning of cortisol in many fibromyalgia patients. Given that reduced levels of cortisol tend to be associated with increased pain perception, it has been suggested in the fibromyalgia literature that for fibromyalgia patients who experience reduced levels of cortisol, their sensitivity to pain is likely increased such that their perception of the frequency and severity of their pain is likely exacerbated (Crofford et al., 1994). The perpetuation of fibromyalgia pain may be due, in part, to a generally reduced level of cortisol. Moreover, given that the HPA axis is responsible for the regulation of cortisol, research is currently underway regarding the functioning of the HPA axes of FMS patients, as the HPA axis may have a significant etiological role in FMS (Crofford et al., 1994; Griep et al., 1993; Parker et al., 2001).

Central pain processing refers to the latter portion of the pain processing system wherein the interdependent functioning of particular central nervous system (CNS)
structures/regions is responsible for the perception of sensory stimuli as painful (Casey, 1996). The particular CNS structures/regions that are involved in central pain processing include the thalamus, cingulate cortex, somatosensory cortex, and prefrontal cortex (Casey et al., 1994; Coghill et al., 1994; Gracely, Petzke, Wolf, & Clauw, 2002; Hsieh, Belfrage, Stone-Elander, Hansson, & Ingvar, 1995; A. K. P. Jones, Brown, Friston, Qi, & Frackowiak, 1991). Researchers (e.g., A. K. P. Jones et al., 1991) have observed that when an individual experiences pain on account of exposure to subjectively intense mechanical, thermal, and/or electrical stimulation, there is a subsequent increase in blood flow to the above-listed structures/regions of the CNS that are involved in processing the pain. Several functional neuroimaging tools can be used to observe such increases in blood flow, including positron emission tomography (PET) and functional magnetic resonance imaging (fMRI; Gracely et al., 2002; A. K. P. Jones et al., 1991). A detailed discussion regarding the dynamics of central pain processing can be found in Casey (1996).

In the context of FMS, a number of researchers (e.g., Arroyo & Cohen, 1993; Gibson, Littlejohn, Gorman, Helme, & Granges, 1994; Gracely et al., 2002; Kosek, Ekholm, & Hansson, 1996; Lautenbacher, Rollman, & McCain, 1994) have observed dysfunctional central pain processing in many FMS study patients. Various observations using pain rating scales and neuroimaging tools have found lower pain thresholds for mechanical (Gibson et al., 1994; Gracely et al., 2002; Kosek et al., 1996), thermal (Gibson et al., 1994; Kosek et al., 1996), and electrical (Arroyo & Cohen 1993) stimuli, as well as more widespread activation of the CNS structures/regions involved in processing pain (Gracely et al., 2002), in many fibromyalgia study patients. For these
fibromyalgia patients, not only is pain experienced in response to a level of stimulus exposure that typically does not result in an experience of pain in healthy controls, but the number of CNS structures/regions that are activated in response to the stimulus exposure is significantly more than the number of CNS structures/regions that are activated in healthy controls in response to the same level of stimulus exposure (Gracely et al., 2002). It has been suggested in the FMS literature that the chronic pain that marks FMS is maintained/perpetuated in part by lower sensory (i.e., mechanical, thermal, and electrical) pain thresholds as well as an endogenous tendency towards more widespread CNS activation in response to pain (Gracely et al., 2002).

**Perpetuating Psychological Factors**

Illness beliefs have been identified in the fibromyalgia literature (e.g., Sharpe & O’Malley, 2005) as the predominant psychological factors that perpetuate fibromyalgia. In general, an illness belief is a strong belief or concern that a patient has regarding the cause(s) of her or his illness (Sharpe & O’Malley, 2005). In the context of fibromyalgia syndrome, common illness beliefs include the belief that symptoms are due to a physical disease (biological attribution; Neerinckx, Van Houdenhove, Lysens, Vertommen, & Onghena, 2000) and the belief that engagement in increased physical, mental, emotional, or social activity will lead to marked exacerbations of symptoms (catastrophizing; Hassett, Cone, Patella, & Sigal, 2000; Sharpe & O’Malley, 2005). Several researchers (e.g., Chapman, 1978; Heijmans, 1998; Sharpe & O’Malley, 2005) have suggested that in response to illness beliefs, many fibromyalgia patients tend to become hypervigilant (i.e., significantly increase the focus of attention) regarding symptoms, less likely to engage in
behavioral and psychological treatments that are potentially effective (e.g., physical therapy and psychotherapy, respectively), and more physically, socially, and mentally inactive, all of which in turn, contributes to the maintenance or perpetuation of fibromyalgia symptoms through mediating mechanisms such as consequent lower pain thresholds / increased perceived symptom intensity and deconditioning.
A recent review of the literature revealed that fibromyalgia syndrome is best managed through a multifaceted treatment program wherein psychoeducation regarding the disorder, certain pharmacological therapies, and particular nonpharmacological therapies are utilized (Bradley & Alarcón, 2005; Dadabhoy & Clauw, 2008; Sharpe & O’Malley, 2005). What follows is a review of the multiple facets of fibromyalgia management.

**Psychoeducation**

Fibromyalgia treatment starts at the time of diagnosis, wherein the physician educates the patient regarding the disorder (Dadabhoy & Clauw, 2008; Sharpe & O’Malley, 2005). When patients are diagnosed with fibromyalgia, they are often provided with an explanation of the symptomatology and etiology of fibromyalgia syndrome that is both scientifically accurate and comprehensible (Sharpe & O’Malley, 2005). Given that the etiology of FMS is currently unknown, it is common for many physicians to present said etiology in a concise manner, emphasizing the interplay between biological and environmental factors as the cause of the disorder (Sharpe & O’Malley, 2005). Referrals to reputable sources of information such as the Arthritis Foundation, as well as referrals to nationally-based support organizations such as the National Fibromyalgia Association, are often provided to patients for knowledge enhancement (Dadabhoy & Clauw, 2008). The posited therapeutic benefits of psychoeducation include an enhanced ability to
conceptualize physiological, mental, emotional, and social changes that have been occurring in daily functioning for extended periods of time, as well as an enhanced ability to discuss current health functioning with others (Sharpe & O’Malley, 2005; Woodward, Broom, & Legge, 1995). Moreover, during the psychoeducation intervention it is common for the physician and patient to collaboratively develop a management plan (Sharpe & O’Malley, 2005). Common foci of the plan include symptom relief using pharmacological therapies such as antidepressants, and general enhancement of coping skills using nonpharmacological therapies such as aerobic exercise and cognitive-behavioral therapy (CBT; Sharpe & O’Malley, 2005).

**Pharmacological Therapies**

In general, the effectiveness of pharmacological therapy or pharmacotherapy in managing fibromyalgia syndrome is variable (Bradley & Alarcón, 2005; Dadabhoy & Clauw, 2008; Sharpe & O’Malley, 2005). Nonetheless, several pharmacological therapies including antidepressant (Arnold et al., 2002; Arnold, Keck, & Welge, 2000; Dwight et al., 1998; Fishbain, 2003; Goldenberg, Burckhardt, & Crofford, 2004; O’Malley et al., 2000), muscle relaxant (Tofferi, Jackson, & O’Malley, 2004), and anticonvulsant (Crofford et al., 2002) therapies have been observed to be generally effective in reducing the symptoms of FMS. Regarding antidepressant therapies, several researchers (e.g., Arnold et al. 2000; Fishbain, 2003; Goldenberg et al., 2004; O’Malley et al., 2000) have observed tricyclic antidepressant (TCA) therapies to be the most effective antidepressant therapies. Among the observed benefits of TCA therapies are improved sleep, reduced fatigue, decreased pain, and a generally improved sense of well-being (Arnold et al.
2000; Fishbain, 2003; Goldenberg et al., 2004; O’Malley et al., 2000). Although research on the effectiveness of selective serotonin reuptake inhibitor (SSRI) therapies in managing FMS is limited and generally unfavorable, Arnold et al. (2002) observed fluoxetine (an SSRI) to be effective in decreasing pain, reducing fatigue, and alleviating depression in FMS patients. Similarly, Dwight et al. (2004) observed venlafaxine (a serotonin-norepinephrine reuptake inhibitor or SNRI) to be effective in improving the overall quality of sleep / increasing nonrestorative sleep, decreasing pain, reducing fatigue, decreasing morning stiffness, and generally improving the quality of life in fibromyalgia patients.

Tofferi et al. (2004) observed cyclobenzaprine, a tricyclic antidepressant that is commonly used as a muscle relaxant, to be effective in moderately improving sleep and modestly decreasing pain in fibromyalgia patients. Crofford et al. (2002) and Roth, Lankford, Bhadra, Whalen, and Resnick (2012) observed pregabalin, an anticonvulsant that was specifically developed for the treatment of chronic pain syndromes such as fibromyalgia, to be effective in decreasing pain, improving sleep, and improving overall quality of life in FMS patients. Moreover, there are several symptom-specific pharmacotherapies that are commonly prescribed to FMS patients, including tramadol therapy (Bradley & Alarcón, 2005; Dadabhoy & Clauw, 2008). Tramadol is an analgesic that targets the central pain processing system; it has been observed to significantly reduce pain in FMS patients (Bennett, Kamin, Karim, & Rosenthal, 2003; Russell et al., 2000). In addition, many other pharmacotherapies for FMS have been investigated, such as nonsteroidal anti-inflammatory drug (NSAID) therapies (Goldenberg, Felson, & Dinerman, 1986), opiate therapies (Sharpe & O’Malley, 2005), benzodiazepine therapies,
(Sharpe & O’Malley, 2005), and corticosteroid therapies (Clark, Tindall, & Bennett, 1985); overall, the effectiveness of these particular pharmacotherapies has been observed to be minimal, at best (Sharpe & O’Malley, 2005).

**Nonpharmacological Therapies**

In the context of fibromyalgia syndrome, there are several nonpharmacological therapies that are commonly utilized by patients (Bradley & Alarcón, 2005; Dadabhoy & Clauw, 2008; Sharpe & O’Malley, 2005). One of the most popular nonpharmacological therapies is an exercise program (Bradley & Alarcón, 2005; Dadabhoy & Clauw, 2008; Sharpe & O’Malley, 2005). A review of the fibromyalgia literature reveals that there are many different exercise programs that can be employed with fibromyalgia patients, most of which tend to result in generally improved functioning / general symptom reduction (most notably, reductions in pain and fatigue; Busch, Barber, Overend, Peloso, & Schachter, 2006). Examples of effective exercise programs include pool-based exercise programs (Jentoft, Kvalvik, & Mengshoel, 2001; Mannerkorpi, Nyberg, Ahlmén, & Ekdahl, 2000); combined exercise programs wherein physical training and psychoeducation regarding exercise are utilized (Burckhardt, Mannerkorpi, Hedenberg, & Bjelle, 1994; Gowans, DeHueck, Voss, & Richardson, 1999; King, Wessel, Bhambhani, Sholter, & Maksymowych, 2002); a progressive, home-based, low-impact aerobics program (Schachter, Busch, Peloso, & Sheppard, 2003); a combined exercise program wherein progressive strength training and cardiovascular exercise are utilized (Rooks, Silverman, Kantrowitz, 2002); a combined exercise program wherein aerobic, flexibility, and strengthening exercises are utilized (Martín et al., 1996); and a
cardiovascular fitness training program (McCain, Bell, Mai, & Halliday, 1988). Regardless of the exercise program that is utilized by the patient, it has been recommended in the fibromyalgia literature (e.g., Dadabhoy & Clauw, 2008) that program facilitators initially emphasize slower and less intense exercises; as the patient progresses in the program, facilitators are encouraged to emphasize more intense exercises (within appropriate limits for the patient) and in collaboration with the patient, develop an appropriate long-term exercise program.

One popular set of nonpharmacological therapies for FMS includes local rubs and creams (e.g., topical analgesics and counterirritants), vitamins, herbal products, special diets, spiritual practices such as prayers, meditation, heat therapy, aromatherapy, massage therapy, chiropractic services, acupuncture, and literature (e.g., books, newsletters, and videos regarding FMS; Barbour, 2000; Dimmock, Troughton, & Bird, 1996; Henriksson, 1994; Nicassio, Schuman, Kim, Cordova, & Weisman, 1997; Pioro-Boisset, Esdaile, & Fitzcharles, 1996). These nonpharmacological therapies are commonly referred to as complementary or alternative therapies, as many fibromyalgia patients tend to complement or replace traditional (i.e., pharmacological) therapies with one or more of the above-listed nonpharmacological therapies (Bradley & Alarcón, 2005). Among the above-listed nonpharmacological therapies, acupuncture is the only therapy that has garnered modest attention regarding its efficacy in alleviating the symptoms of fibromyalgia syndrome (Deluze, Bosia, Zirbs, Chantraine, & Vischer, 1992; Langhorst, Klose, Musial, Irnich, & Häuser, 2010; Singh et al., 2006). Among the observed benefits of acupuncture therapy are decreased acute pain (Deluze et al., 1992; Langhorst et al., 2010), decreased long-term / chronic pain (Singh et al., 2006), improved mood /
decreased depression (Singh et al., 2006), and improved quality of sleep (Deluze et al., 1992).

Cognitive-behavioral therapy (CBT) is another common nonpharmacological therapy that is utilized by fibromyalgia syndrome patients (Bradley & Alarcón, 2005; Dadabhoy & Clauw, 2008; Sharpe & O’Malley, 2005). The underlying premise of CBT is that an individual’s behavior (e.g., an FMS patient’s engagement in a physical exercise) is influenced by the individual’s internal cognitive factors (e.g., the patient’s self-efficacy regarding the physical exercise or belief regarding his or her ability to complete the exercise), the individual’s internal emotional factors (e.g., the patient’s current mood), and the type of reinforcement that is present in the environment (e.g., positive social reinforcement in the form of accolades from a loved one or exercise program facilitator; Bradley & Alarcón, 2005). As such, the interventions that tend to be utilized in CBT with FMS patients include psychoeducation regarding the psychological maintenance of FMS as well as the potential impact of the therapeutic process on alleviating FMS symptoms (educational interventions; Bradley & Alberts, 1999); relaxation as well as coping skills training, reinforcement of healthy behavior patterns, and cognitive restructuring / challenging maladaptive beliefs regarding symptom control (skills acquisition-oriented interventions; Bradley & Alarcón, 2005; Bradley & Alberts, 1999); cognitive and behavioral rehearsal, wherein the patient practices newly acquired cognitive and behavioral skills in both the treatment setting and the outside (home and/or work) environment, which often involves collaborations with the therapist in identifying and correcting problems that have the potential to interfere with the efficacy of the newly developed skills in the future (Bradley & Alarcón, 2005; Bradley & Alberts, 1999); and
relapse prevention, wherein the therapist assists the patient in learning how to anticipate environmental or internal events that may prevent the use of newly acquired coping skills, which often involves rehearsal of the newly acquired skills so that the patient can cope with the early signs of relapse in an adaptive manner (and consequently prevent a full relapse; Keefe & Van Horn, 1993).

According to Sharpe and O’Malley (2005), although CBT can be employed with fibromyalgia patients in either an individual or group format, there is limited research on the efficacy of individual-based CBT in relieving the symptoms of fibromyalgia syndrome. In contrast, researchers (e.g., Anderson & Winkler, 2007; Bennett et al., 1996; Turk, Okifuji, Sinclair, & Starz, 1998; Van Wilgen, Bloten, & Oeseburg, 2007; Vázquez-Rivera et al., 2009; D. A. Williams et al., 2002) have observed group-based CBT, which tends to be psychoeducational in nature, to be effective in providing symptom relief to FMS patients. Among the observed benefits of group-based CBT are improved physical functioning (D. A. Williams et al., 2002); improved quality of life (Bennett et al., 1996; Turk et al., 1998; Van Wilgen et al., 2007); decreased tenderness (Bennett et al., 1996); decreased feelings of distress (Vázquez-Rivera et al., 2009); improved mood (Anderson & Winkler, 2007; Turk et al., 1998; Van Wilgen et al., 2007); decreased muscle stiffness (Van Wilgen et al., 2007); and decreased pain, fatigue, and anxiety (Anderson & Winkler, 2007; Turk et al., 1998; Van Wilgen et al., 2007). Given that the research on CBT with FMS patients tends to favor/focus on the psychoeducational group therapy format over the individual therapy format, it seems appropriate to end the discussion concerning the management of fibromyalgia syndrome with a review of the prominent
nuances concerning the facilitation of psychoeducational group therapy with a medically ill population such as the fibromyalgia syndrome population.

Psychoeducational Group Therapy Approach

When conducting psychoeducational group therapy with a group of medically ill patients such as fibromyalgia patients, there are several facilitative approaches available (Spira, 1997). Two of the most common approaches are the deductive approach and the inductive approach (Spira, 1997). In the deductive approach, the therapist assumes the role of a health educator, selecting the topic for discussion for each session; each session is generally structured as a lecture (Spira, 1997). In addition, the therapist is primarily responsible for the dissemination of information to group members, wherein empirically-validated educational and public health principles are utilized in order to present information to patients in a patient-contextualized manner, which in turn, facilitates the patients’ integration of the information (Spira, 1997). In contrast, in the inductive approach each session is structured as a combination of lecture, discussion, and experiential exercise, and the therapist assumes the role of an emcee, wherein she or he provides the lecture / introduces the guest lecturer, facilitates the subsequent discussion regarding the information presented in the lecture, leads the experiential exercise, and consistently interacts or dialogues with the patients (Spira, 1997). The inherent structure of the treatment program will determine the most adaptive facilitative approach to employ (Spira, 1997).
**Psychoeducational Group Therapy Method**

Psychoeducational group therapy can range from employing a strict educational treatment method to a strict cognitive treatment method to a combined educational-cognitive treatment method (Spira, 1997). A strict educational treatment method tends to use a classroom-like setting/structure wherein information and experiential skills are taught to patients by the therapist; there tends to be limited dialogue between the therapist and the patients (Spira, 1997). In contrast, a strict cognitive treatment method tends to use a more informal meeting-like setting/structure wherein the therapist consistently interacts with the patients as information is provided and experiential skills are practiced (Spira, 1997). According to Spira (1997), it is common for a psychoeducational group therapist to integrate the educational and cognitive treatment methods in order to enhance the overall functioning of the group, as there are benefits to both treatment methods.

**The Problem of the Subgroup**

One of the prominent problems in psychoeducational group therapy is the development of a subgroup or clique (Spira, 1997). It is common for particular group members to bond / form a subgroup because of similar interests, worldviews, life experiences, etc. (Spira, 1997). The formation of a subgroup can be adaptive in that it can lead to the establishment of strong socially-emotionally supportive relationships (Spira, 1997). However, if a subgroup begins to exert dominance towards another group member or another subgroup (e.g., a subgroup of active problem solvers begins to suggest multiple and unsolicited solutions to another group member), then it is the responsibility of the therapist to diffuse the situation by validating the subgroup’s problem solving
efforts, reflecting to all of the group members the variable nature of problem solving, and reminding all of the group members about the value of respect for others’ decisions (Spira, 1997).

**Introverted and Extroverted Group Members**

Among the different types of group members that can interfere with the functioning of psychoeducational group therapy, two of the most common types are the introverted group member and the extroverted group member (Spira, 1997). The introverted group member is the remarkably frightened, shy, or quiet group member who does not contribute in any capacity to the group dialogue (Spira, 1997). In response to an introverted group member, therapists are encouraged to use the more expressive group members as models; in other words, after a more expressive group member states a point, then the therapist can direct attention to the introverted group member and ask the introverted group member to share a similar experience, comment on the expressed point, etc. (Spira, 1997). In addition, therapists are encouraged to find opportunities for introverted group members to assist (i.e., communicate with) other group members (Spira, 1997).

The extroverted group member is the remarkably verbose and externally focused group member who tends to interrupt other group members as well as the therapist (Spira, 1997). In response to an extroverted group member, therapists are advised to politely interrupt the group member after an appropriate amount of time has passed, typically by employing a reflective statement and subsequently seeking confirmation from the group member regarding the accuracy of the reflection, and then redirect the attention of the
group to another group member or to another topic (Spira, 1997). By employing these types of active listening techniques, not only is the extroverted group member’s experience validated, but the member’s verbosity is limited. In addition, by employing this approach with one extroverted group member, other extroverted group members receive vicarious feedback regarding their overly expressive behavior, which may motivate them to better monitor their expressiveness during future sessions (Spira, 1997).

Specific Considerations for the Facilitation of Psychoeducational Group Therapy with Fibromyalgia Patients

When facilitating psychoeducational group therapy with fibromyalgia patients, a common challenge is the management of fibromyalgia monopolists or fibromyalgia patients who frequently express their vast or detailed knowledge concerning fibromyalgia syndrome, such that they dominate the group (K. Boyd, personal communication, May 16, 2012; Yalom & Leszcz, 2005). According to Yalom and Leszcz (2005), the motivation for the monopolist’s domineering in-group behavior varies from one monopolist to another monopolist; however, some of the common motivations include a need to control (e.g., a need to control the ambiguity of the future as it relates to the progression of FMS), a fear of being influenced in a maladaptive manner by others (such that there is a drive to defend statements using personally obtained knowledge), and a self-perception that ideas and observations are so valuable that they warrant immediate expression (Yalom & Leszcz, 2005). The impact of a monopolist on the functioning of a psychoeducational group can be severe and include the silencing of other group members as well as the development of anger towards the monopolist (Yalom & Leszcz, 2005). To elaborate, other group members may hesitate to silence a monopolist on account of a fear
that should the monopolist be silenced, then they will have to consequently fill the silence (Yalom & Leszcz, 2005). In addition, for many of the silenced group members, they tend to “smolder quietly or make indirect hostile forays” (Yalom & Leszcz, 2005, p. 392) towards the monopolist, all of which in turn, tends to impair the therapeutic process / interfere with group members’ acquisition of information that is presented in session.

According to Yalom & Leszcz (2005), managing a monopolist requires a two-pronged approach that takes into consideration the dynamics of the group. The first prong of the approach involves asking the other group members about their motivations for allowing the monopolist “to carry the burden of the entire (session)” (Yalom & Leszcz, 2005, p. 393). In so asking the above question, the facilitator is assisting the other group members in gaining awareness as to their roles in the maladaptive dynamics of the group (Yalom & Leszcz, 2005). It is common for many of the other group members to endorse reduced responsibility as their primary motivation for allowing the monopolist to carry the burden of the session, which tends to stimulate discussion regarding the underlying factors for the reduced responsibility, which in turn, tends to result in therapeutic progress and stronger engagement in the group (Yalom & Leszcz, 2005). The second prong of the approach involves the facilitator and other group members providing feedback to the monopolist, wherein the underlying message is that “(we) want to hear more” (Yalom & Leszcz, 2005, p. 394). In other words, the monopolist will receive feedback from other group members as well as the facilitator that feelings of distance and detachment are commonly experienced in response to the monopolist’s constant sharing of fibromyalgia information (Yalom & Leszcz, 2005). The provision of feedback to the monopolist is intended to serve as an invitation to the monopolist to engage with the
group in an open and honest manner, which in turn, is expected to lead to the enhancement/improvement of the group therapy experience for all of the members (Yalom & Leszcz, 2005).

Another common challenge when facilitating psychoeducational group therapy with FMS patients is the management of FMS patients who strongly advocate for nonpharmacological treatments such as those presented earlier in this manual (e.g., acupuncture; K. Boyd, personal communication, May 16, 2012). These types of patients/group members tend to be highly motivated to learn about the newest nonpharmacological treatment options available and tend to advocate the use of these treatment options among the other members of the group (K. Boyd, personal communication, May 16, 2012). Although the use of nonpharmacological treatments in managing fibromyalgia has increased over the last two decades (Barbour, 2000; Dimmock et al., 1996; Visser, Peters, & Rasker, 1992), it is not a practice that is engaged in by all fibromyalgia patients, nor do all medical doctors/rheumatologists condone the use of such treatments (Visser et al, 1992). As such, these types of group members may be perceived as highly assertive by other group members and/or receive responses of rejection/detachment from other group members, in particular those who choose to engage in more conventional (and doctor-approved) treatment options, all of which in turn, may lead to maladaptive group dynamics and interfere with group members’ acquisition of information that is presented in session (K. Boyd, personal communication, May 16, 2012).

Managing these types of patients may require the facilitator to validate the patients’ efforts to manage fibromyalgia to the best of their abilities, normalize their
pursuit of nonpharmacological treatment for the management of their fibromyalgia, and invite the other members of the group to openly discuss the information that is presented by these particular patients. By employing these interventions, the facilitator is facilitating two of the beneficial processes of group therapy: modeling and the imparting of information (Yalom & Leszcz, 2005). Modeling refers to the phenomenon wherein group members tend to learn new coping skills in a more effective manner when they are modeled by other group members instead of experts (Fawzy, Fawzy, & Wheeler, 1996). The imparting of information phenomenon refers to the inherent psychoeducational nature of the psychoeducational group therapy format, wherein group members learn various types of fibromyalgia-related information not only from the facilitator and/or expert presenters, but from other group members (Yalom & Leszcz, 2005). Managing these types of patients in the above-described manner will likely lead to an enhancement of the group therapy experience for all of the members.

One additional challenge to facilitating psychoeducational group therapy with fibromyalgia syndrome patients is managing the common reaction of resistance to treatment topics such as processing the cognitive distortions that may exacerbate many of the symptoms of fibromyalgia syndrome including pain, difficulty falling asleep, depression, and anxiety (K. Boyd, personal communication, May 16, 2012). Many fibromyalgia patients have experienced disbelief from medical practitioners, family members, and friends in response to their fibromyalgia symptoms (Arnold et al., 2008), often being told that it is “in your head” (Barbour, 2000, p. 315); consequently, when topics arise in the group such as the employment of cognitive-behavioral therapy techniques in order to reduce the intensity of fibromyalgia symptoms, an underlying
message that may be conveyed to the patients is that their disorder is fictional and can be easily changed with alterations in thinking.

If a patient experiences resistance to the topic of psychological interventions for the effective management of FMS, and the facilitator does not appropriately address the patient’s reaction, then the patient may stop attending group altogether. As such, researchers (e.g., Anderson & Winkler, 2007; Yalom & Leszcz, 2005) suggest that the facilitator promotes open and honest communication among the group members, so that resistant reactions like that referenced above can be explored/processed among the group members in an adaptive manner. The author of this manual highly recommends that as part of this exploration / emotional processing, the facilitator not only validates the emotional reactions of these patients, but encourages other group members to share how they have coped with such responses from doctors and loved ones. In so doing, the facilitator will be facilitating another beneficial process of group therapy: cohesion or the formation of social support based on shared experiences (Yalom & Leszcz, 2005). For many of these types of patients, having the opportunity to openly discuss their typical reactions to disbelief from doctors and loved ones as well as to learn how others have coped with disbelief from others may be an extremely novel and insightful experience, which may in turn, increase their likelihood of remaining in treatment (Anderson & Winkler, 2007).

Overall, when facilitating psychoeducational group therapy with fibromyalgia syndrome patients it is important to remember that given the therapeutic format of the group, for it is psychoeducational and not process-oriented, the focus should consistently be on improving the general functioning of the patients (Spira, 1997). Should the focus of
any session progress from the featured skill set (e.g., strategies to improve sleep hygiene) to a comparison of symptoms and/or symptom-based deficits (e.g., “My pain is worse because I have fifteen active tender points whereas you just have seven active regions!” or “My doctor said that I am the worst case he has ever seen because I have memory problems on top of everything that you have!”), it is advised that the facilitator promptly re-direct the focus of the group to the skill set (K. Boyd, personal communication, May 24, 2012; Spira, 1997). Redirection may be facilitated by use of interventions discussed above, such as validation of patients’ symptoms and the impact of their symptoms on daily functioning, followed by group exploration of how said symptoms can be better managed by the featured skill set. By redirecting the focus of the group in this manner, not only is group cohesion maintained, but the therapeutic goal of improving FMS patient functioning remains intact.
Fibromyalgia syndrome is a chronic pain syndrome that is marked by a highly complex symptomatology, a relatively common prevalence, a recently revised set of diagnostic criteria, a currently unclear etiology, and a currently diverse/naive management plan (Sharpe & O’Malley, 2005). Nonetheless, one of the most consistent findings in the fibromyalgia literature is that the impact of fibromyalgia syndrome on daily functioning is severe (e.g., Arnold et al., 2008). For the average fibromyalgia syndrome patient, not only is everyday marked by pain, fatigue, tenderness, muscle stiffness, anxiety, depression, and impaired sleep, to name a few of the physiological and psychological symptoms, but daily social, familial, occupational, and academic (if appropriate) functioning tends to be impaired (Arnold et al., 2008). To illustrate, many fibromyalgia patients tend to report significant difficulties regarding the establishment as well as maintenance of friendships, often attributing the difficulties to the unpredictable nature of their fibromyalgia symptoms (Arnold et al., 2008). According to some FMS patients, committing to a social activity or event is incredibly challenging on account of symptom ambiguity and a fear of being judged as unreliable, as increased severity or intensity of symptoms on the day of the activity or event tends to be associated with an increased likelihood of not attending the activity or event (Arnold et al., 2008). Furthermore, many FMS patients report that they tend to lose friends on account of skepticism regarding their FMS symptoms as well as on account of a lack of sympathy/compassion on the part of their friends concerning the general disability that
they have experienced on account of fibromyalgia (Arnold et al., 2008; Bernard et al., 2000).

Regarding familial functioning, many fibromyalgia patients report that on account of fibromyalgia interfering with their ability to perform a number of household duties, there are increased household responsibilities for spouses or partners such as increased childcare and an increased number of household chores (Arnold et al., 2008). Furthermore, many FMS patients report that their marriages or significant intimate relationships are significantly impacted by the sexual symptoms of fibromyalgia syndrome, most notably the lack of sexual desire (Arnold et al., 2008; Bernard et al., 2000). In addition, many FMS patients report that on account of their FMS symptoms, they are unable to be as involved in child-rearing as they were prior to FMS onset (Arnold et al., 2008). As such, assisting children with homework is markedly reduced, as is being able to participate in school-related events as well as attend family trips (Arnold et al., 2000).

Regarding occupational functioning, many fibromyalgia syndrome patients report that on account of their FMS symptoms, most notably the pain and impaired attention abilities, they have had to quit their jobs or reduce the number of hours in which they work (Arnold et al., 2008; Bernard et al., 2000). Consequently, these people tend to experience increased financial hardships, which can exacerbate strained familial (i.e., spousal/partner and/or parent-child) relationships (Arnold et al., 2008). Regarding academic functioning, many fibromyalgia patients report that they have had to forgo higher education on account of the neuropsychological symptoms of fibromyalgia, most
notably impaired attention abilities and impaired memory abilities, as well as on account of the characteristic chronic and widespread pain (Arnold et al., 2008).

Lastly, many fibromyalgia syndrome patients report that on account of their fibromyalgia symptoms, most notably pain, fatigue, and depression, they experience significant difficulty regarding the completion of activities of daily living (Arnold et al., 2008). Examples of activities of daily living that many fibromyalgia patients tend to have difficulty completing include household chores such as house cleaning and grocery shopping, self-care activities such as bathing, cooking, and driving, and leisure activities such as camping, hiking, team sports, and traveling (Arnold et al., 2008). In sum, the experience of fibromyalgia is associated with significant lifestyle changes, not only for the patient, but for the patient’s entire family.
References


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