Remembering the pain: Psychosocial factors related to endometriotic pain and its recall

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REMEMBERING THE PAIN: PSYCHOSOCIAL FACTORS RELATED TO ENDOMETRIOTIC PAIN AND ITS RECALL

by

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A dissertation submitted in partial fulfillment of the requirements for the

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Doctor of Philosophy in Psychology

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ABSTRACT

Remembering the Pain: Psychosocial Factors Related to Endometriotic Pain and its Recall

by

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Endometriosis is a chronic and debilitating disease, affecting women of childbearing years in a variety of ways, including infertility, dyspareunia, dysmenorrhea, and chronic pelvic pain. Diagnosis and treatment planning are guided primarily by retrospective pain recall, regardless of the fact that 1) there is only a tenuous relationship between pain reports and physical pathology and 2) the accuracy of pain recall has never been assessed in this population. The current study investigated the accuracy of endometriotic pain recall over a 30-day period, as well as potential psychological mediators to pain recall accuracy, including general psychological wellbeing, distress specific to infertility, passive and active coping, and pain present at time of recall. Contrary to expectations, findings indicated that women were relatively accurate in their recall of pain. Only passive coping and pain present at recall were predictive of accuracy, with greater passive coping and lower pain at recall predictive of higher recalled than recorded pain. Study implications are discussed, including: 1) report of pain over a 30-day duration appears credible for the majority of endometriotic patients and 2) women exhibiting greater...
passive coping may benefit from psychological treatment in addition to medical intervention.
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Endometriosis, a disorder in which uterine tissues migrate and implant outside of the uterine cavity, is one of the most prevalent female reproductive diseases and pain syndromes, yet also one of the least understood. In fact, Wilson (1987) has even gone as far as to liken the disease to one of Winston Churchill’s famous quotations, “A riddle wrapped in a mystery inside an enigma.” A surge of research in the area has recently occurred, such that numerous funding proposals submitted to congress in the early 1990’s were turned down due to their sheer numbers, while less than a decade prior (i.e. 1983), research funds available through the Endometriosis Association had gone unclaimed (Ballweg, 1995). In spite of increased interest and investigation, the disease continues to puzzle the medical profession and researchers alike. Even diagnosis and classification continue to stir debate, and the variance in disease expression from one sufferer to the next is vexing to healthcare service providers.

Endometriotic pain, purportedly caused by lesions in the pelvic cavity, has been highlighted as the single most devastating symptom of the disease (Barlow & Glynn, 1993; Vercellini, 1997), yet remarkably little research has been conducted on psychological mediators and impact of such pain. Researchers have acknowledged both physical and emotional components to such pain (Barlow & Glynn, 1993), however the literature has focused almost exclusively on medical aspects such as physiological...
mechanisms and hormonal/surgical treatments, neglecting the emotional/psychological component of pain with few exceptions. This point is best illustrated by the scarcity of empirical studies on endometriosis within the health-psychology literature compared to the vast psychologically-oriented literature on multiple other pain syndromes such as back pain, arthritis, and migraine, just to name a few.

Perhaps the most vexing aspect of endometriosis for researchers and clinicians alike is the lack of a relationship between organic pathology (i.e. number, size and location of pelvic lesions) and report of pain. The paradoxical situation of a patient with limited pathology reporting extreme pain and the patient with extensive and severe pathology reporting little to none (Stout, Steege, Dodson & Hughes, 1991) is common. The lack of association between pain and organic pathology has only recently led a small number of researchers to consider psychosocial factors that may mediate the experience of endometrial pain (Peveler, Edwards, Daddow & Thomas, 1996; Stout, Steege, Dodson & Hughes, 1991). The paucity of investigations into these factors is surprising considering psychological factors have long played a prominent role in theories of pain (Jacob & Kerns, 2001; Melzack & Wall, 1965; Weisenburg, 1983) and elevated psychological distress levels have been previously implicated in endometriosis (Collins, 1979; Lewis, Comite, Mallouh, Zadunaisky, Huchingson-Williams, Cherksey & Yeager, 1987; Willis, 1996). However, an equally pressing, if not precursory, issue yet to be addressed within the exploration of endometriotic pain is the accuracy with which women report their pain, and the psychological factors mediating such accuracy. Considering diagnosis and decisions regarding the management of endometriosis are heavily reliant on pain reporting, the importance of accuracy becomes apparent. Given the belief
currently gaining support in the medical field to consider only symptomatic women (i.e. those reporting pain symptoms) as cases, the accuracy of pain report clearly has the potential for impact on diagnosis and treatment.

Pain recall, although investigated in other chronic pain populations such as arthritis and back pain patients (Bolton, 1999; Eich, Reeves, Jaeger & Radford, 1985; Erskine, Morley, & Pearce, 1990; Feine, Lavigne, Dao, Morin & Lund, 1998) and to a small extent within gynecological populations (Brodie & Niven, 2000; Niven & Murphy-Black, 2000) has been altogether ignored with regard to endometrial pain. Considering that pelvic pain in general is associated with more psychological distress than most other pain syndromes, it is likely that pain recall is strongly impacted by psychological factors in the case of endometriosis. The current study investigated the accuracy of such pain reports within the endometriosis population and more specifically, the psychosocial factors related to pain recall accuracy. The issue of recall accuracy in this population is particularly important given that pain report, which necessitates some aspect of pain recall, is the primary indication for pharmaceutical and surgical treatment (Bergqvist, 1999; Damario & Rock, 1995; Koninckx, Timmermans, Meuleman, & Penninckx, 1996; Thomas, 1995). Identifying psychological mediators to accurate pain reporting assists physicians and clinicians alike in assessing the pain of their patients more accurately, while identifying which patients might benefit from psychological in addition to medical intervention when treating endometriosis and generating pain management programs.
CHAPTER 2

LITERATURE REVIEW

Overview of Endometriosis

*Disease Profile*

Broadly defined, endometriosis is the migration, implantation and hormonal response of endometrial tissue in any location outside of the intrauterine cavity. The most common site for tissue implantation is the abdominal region, including the ovaries, uterosacral ligaments, broad ligaments, the outside surface of the uterus, lining of the pelvic cavity, and the peritoneum. Other sites include the ureter, bladder, bowel omentum, vagina and vulva. Infrequently, tissue may migrate to the lung, arm and thigh area. The endometrial tissue responds to the fluctuations in estrogen and progesterone that routinely take place throughout the menstrual cycle. As such, when conception fails to occur each month and hormone levels decline, the tissue responds as does that found in the uterine cavity, causing cell fragmentation, bleeding, inflammation and if left unchecked, scarring (Garner, 1997). Endometriosis has been likened to cancer in its clinical presentation, in that cancer-attributes such as speedy growth, spread, invasion and metastasis are mirrored in this condition. In severe cases, when growth occurs within the ovary, rapid enlargement and perforation occurs, such that cysts develop and can result in painful rupture, referred to as a “blowout.” Alternatively, cystic formations may result in seepage of menstrual fluid, plastering the ovary to any contiguous area and causing
painful adhesions (Fallon, Brosnan, & Moran, 1946). As is apparent by its clinical description, endometriosis is an extremely complicated, severe and potentially painful condition. It is therefore understandable that patients frequently criticize the medical profession for trivializing their symptoms and delaying accurate diagnosis (Ballweg, 1995).

Symptomology

Paradoxically, endometriosis may present with severe symptomology or alternatively, remain relatively asymptomatic (Stones, 2000). This has posed a particular challenge for researchers and physicians in the field, as symptoms often display an inverse relationship with disease pathology as determined by extent of lesions (Bernhard, 1982). The five most frequently encountered symptoms include dysmenorrhea, dyspareunia, chronic pelvic pain, infertility and menstrual abnormalities. Symptomatology can also include fatigue, painful urination, frequent bowel movements during menstruation and gastrointestinal complications such as diarrhea, constipation and nausea (Ballweg, 1995). Dysmenorrhea is thought to stem from cyclic bleeding or stretching of peritoneal bands (Bernhard, 1982). Pain generally starts a few days before menstruation, lasts throughout and continues for a few days following the menstrual period, is located in the lower abdominal area (sometimes lower back) and is usually experienced as dull (Bernhard, 1982). Dyspareunia frequently occurs with deep penile penetration (Bernhard, 1982) and is most common with disease involvement in the cul-de-sac or uterosacral ligaments (Williams, 1985). Pain may also radiate to the thighs when ovarian cysts are present and extreme pain may occur with cystic rupture (Bernhard, 1982). Chronic pelvic pain, often brought on by bleeding and scarring.
(Garner, 1997) is generally more complicated to diagnose, document, and treat, as complaints are commonly vague, intermittent, and often counterintuitive to known disease pathology (Yankauskas, 1990). Some women experience constant pain throughout the entire menstrual cycle, while others experience a more recurrent pattern (Dick, 2004). Ironically, the single consistent factor appears to be that severity of such pain remains variable. Infertility frequently causes women to seek medical care (Williams, 1985), and although endometriosis is implicated in a substantial proportion of cases, exact causal mechanisms remain unknown (Spangler, Jones, & Jones, 1971). It is speculated that infertility may be caused by endometrial implants, fibrosis, adhesions on/near fallopian tubes (i.e. tubal obstruction) or possible interference with the pickup and transfer of the ovum (Bernhard, 1982). However, endometriosis in and of itself does not preclude pregnancy, as many women with the disease have successfully carried out several pregnancies (Williams, 1985). Finally, with regard to menstrual abnormalities, bleeding is frequently excessive and prolonged.

Classification

Disease classification has been an area of controversy and thus, continual modification. Classification attempts originated in 1921 with the rudimentary identification of endometrial implantation (Sampson, 1921). It was not until 1973, however, that the first classification based on surgical staging of actual lesions was proposed. Classification consisted of a 3-stage model of mild, moderate, and severe degrees of pathology based on surgical findings, including site of lesions and presence of adhesions and scarring (Acosta, Buttram, Besch, Malinak, Franklin, & Vanderheyden, 1973; Hoeger & Guzick, 1999). In 1979, the American Fertility Society (AFS) produced
its first official classification, assigning a severity score based on location and extent of lesions. Patients were classified into mild, moderate, severe or extensive disease, and assessed on presence of adhesions in ovaries, fallopian tubes and peritoneum (American Fertility Society, 1979; Schenken, 1998). Additionally, size of endometriomas, as well as presence of dense versus filmy adhesions was recorded (Roberts & Rock, 2003). Less than a decade later, the AFS revised its original scoring system (R-AFS) in an attempt to rectify limitations of the original scheme (American Fertility Society, 1985). A separate category was created for minimal disease and the category of extensive disease was removed. Superficial disease was distinguished from invasive endometriosis. Deep endometriosis and dense adhesions were differentiated from superficial disease and filmy adhesions (Schenken, 1998; Hoeger & Guzick, 1999), and quantification of number of adhesions on tubes and ovaries was recorded (Roberts & Rock, 2003). Finally, cul-de-sac involvement was heavily weighted, placing such patients automatically into a diagnosis of severe disease. None of the schemes prior to this R-AFS classification had received widespread acceptance and utilization (Hoeger & Guzick, 1999; Roberts & Rock, 2003).

Several major weaknesses in the current system have, however, been noted, including the potential for observational error, incomplete knowledge of disease pathophysiology, failure to consider morphological characteristics of lesion-types, limited reproducibility, and arbitrariness of scoring system (see Roberts & Rock, 2003 for complete historical review of endometriosis classification). Several researchers have called for new directions in disease classification. Rock & Moutos (1992) suggested future classification models should include the functional status of implants, given that
newer implants may be more active whereas older implants may have "burnt out."

Schenken (1998) has suggested that site and invasiveness of disease as related to pelvic pain may be important in future refinements. In fact, research into degree of endometrial infiltration has provided evidence suggesting endometriosis is a progressive disease (i.e. incidence of endometriomas and depth of infiltration increase with age) and thus, measurement of infiltration may provide insight into the issue of fertility (Koninckx, Meuleman, Demeyere, Lesaffre, & Cornillie, 1991). Brosens, Donnez and Benagiano (1993) suggested that the search must press forward in order to meet theoretical standards for an ideal model. Such a model would include laparotomy findings, a high level of description and practicality, serve as a treatment guide, allow prognosis for likelihood of conception, indicate probability of disease recurrence, and allow for comparison of results.

Because of these weaknesses, in 1993, the American Fertility Society convened a panel of endometriosis experts to devise a form that organized and standardized the most important features of the disease as related exclusively to endometriotic pain. The AFS pain form was intended to document extent of endometriosis and pelvic pain, and included the following: detailed description of patient's pain including anatomical drawings of pain localization, a value of intensity and quality of pain (i.e. mild, discomforting, distressing, horrible or excruciating), documentation of physical findings and tenderness at examination, record of the presence and localization of adhesive disease, and finally, the form included a table to record the diameter, depth, appearance, histology and location of each implant. The AFS pain form was not intended for diagnostic or prognostic use (Whelan, 2003), but was instead designed for utilization in
the evaluation, management and progress of each patient with pain and endometriosis, the eventual goal being accumulation of enough data to construct a classification specific to endometriosis and pain (AFS, 1993). Unfortunately, since its publication, little attempt to utilize the form to create such a system has occurred.

Thus, despite attempts at refinement, the current classification system for endometriosis (R-AFS) remains sub-optimal. Researchers in this field have recognized these limitations and are attempting to rectify the situation. However, until a more useful system is developed, the current scheme, which relies heavily on physiological characteristics of pathology (i.e. number, appearance and location of endometriotic lesions) while ignoring more subtle characteristics (i.e. lesion histology) continues to be utilized by some and completely ignored by others.

Epidemiology

It is widely understood that accurate epidemiological data for endometriosis are difficult to assess given that major diagnostic obstacles exist. In fact, a definitive diagnosis requires pelvic surgery (generally laparoscopy or laparotomy), thus all prevalence estimates have the potential for bias because selective factors may influence which patients actually undergo surgery (Balasch, Creus, Fabregues, Carmora, Ordi et al., 1996; Eskenazi & Warner, 1997). Two issues pose particular difficulty in accurate estimation, especially for case-controlled epidemiological studies (Cramer & Missmer, 2002). First, the broadly defined clinical definition of the disease allows asymptomatic women to be considered cases, a concern given that researchers have begun to question whether mild forms of endometriosis might more accurately be considered a natural and nonpathological condition (Balasch, Creus, Fabregues, Carmona, Ordi, Martinez-Roman...
& Vanrell, 1996; Koninckx, Oosterlynck, D’Hooghe & Meuleman, 1994; Thornton, Lilleyman, Onwude & Crompton, 1997). This suggestion is derived from a variety of findings implicating early and even advanced stages of the disease in women undergoing laparoscopy for other indications who deny all pain symptoms typical of endometriosis (Rawson, 1991). The situation has spurred a recent movement to limit diagnosis of the disease only to symptomatic women (Hurd, 1998). Second, because endometriosis has traditionally been defined as the presence of functional endometrial tissue outside of the uterus and within the pelvic cavity, methods for control selection require surgical procedures to rule out asymptomatic disease, a major problem from a research perspective. It has been suggested that a non-invasive test reliably correlated with disease pathology would be helpful in conducting such research, however no such test presently exists (Rock & Moutos, 1992). This dilemma has also prompted suggestions to revise current disease definition to include only symptomatic women as cases, thus obviating the need to surgically inspect controls (Cramer & Missmer, 2002). However, even this suggestion poses its own unique problems, in that certain populations may be more likely than others to report pain due to cultural and social norms (Eskenazi & Warner, 1997).

Caveats aside, it is estimated that over 5 ½ million American and Canadian, or 10% of women of childbearing age are afflicted with endometriosis (Eskenazi & Warner, 1997). Current estimates suggest 4% of those undergoing tubal ligation are found to have asymptomatic endometriosis, while 17% of women with primary infertility and 5-21% with pelvic pain receive the diagnosis (Cramer & Missmer, 2002). Since initial clinical description in the late 19th century, recorded cases of endometriosis have increased in
frequency (Williams, 1985). What remains unclear is whether this represents a true increase in disease prevalence or simply an offshoot of advanced diagnostic procedures and changing diagnostic criteria (Houston, 1984; Thomas, 1995; Williams, 1985).

Long-standing debate over the characteristics of women with the disease continues unabated. Some empirical support has been found for the following correlates: professional occupation (Richardson, 1987), high socio-economic status (Houston, 1984; Obermeyer, Armenian & Azoury, 1986; Scott & TeLinde, 1950), Caucasian race (Houston, 1984), weight status (Parazzini, La Vecchia, Franceschi et al., 1989) and family history (Cramer & Missmer, 2002; Frey & Bluefield, 1957; Henricksen, 1953; Lamb, Hoffmann, & Nichols, 1986).

Thomas (1995) briefly attempted to link a subset of these findings to possible theories and offered the following: 1) social class and personality may demonstrate an association only in that women from higher social classes may delay childbearing longer, increasing their exposure to estrogen and thus resulting in higher disease risk; 2) susceptibility based on racial background suffers from innumerable confounding variables, including ease of access to laparoscopy; 3) studies suggesting a familial association fail to include the possibility that endometriosis in one family member raises awareness of the disease within the family and may encourage early initiation of medical assistance and; 4) low body mass index (a protective factor) is associated with lower levels of estrogen circulation, and increased estrogen levels are a known etiological factor.

Interestingly, out of almost 100 empirical studies, only 6 met sound epidemiological methodology standards (Eskenazi & Warner, 1997). The only three
factors that appear to demonstrate a stable association with endometriosis include menstruation, estrogen, and age, such that increased exposure to menstruation and estrogen increases disease risk and a positive relationship between disease and age in reproductive years has been demonstrated (Thomas, 1995; Eskenazi & Warner, 1997). As a result of improved diagnostic ability, endometriosis is currently conceptualized as the “equal opportunity” disease, cutting across race, SES, and educational level (Weinstein, 1987).

**Etiology**

Numerous etiological theories have been proposed within the medical literature, yet a definitive causal mechanism remains elusive. Perhaps the most well-known and long-accepted is the retrograde or transtubal migration theory proposed by Sampson (1927) in the early 20th century, speculating that a reverse or retrograde mechanism occurs within the normal menstrual process, such that endometrial debris move counteractively through the fallopian tubes and into the peritoneum. More recently, the role of immunological factors in disease development has also been explored (Evers, 1996; Oosterlynck, Meuleman, Waer, Vandeputte, Koninckx, 1992). Current theoretical speculation contends that such retrograde menstruation in women of reproductive age is the rule rather than the exception, in that the female pelvic cavity is prepared to dispose of such regurgitated debris via its natural immune defense, macrophages and natural cell killers. In women with endometriosis, the immune defense may not be intact, therefore rendering such women susceptible to disease development.

A possible relationship between stress and development of endometriosis has also been postulated (Koninckx, 1987). Although not directly addressed within the literature,
the immunological and stress theories dovetail each other quite nicely. Given that stress and the immune system are intimately related, it may be that increased levels of stress render the immune system weak, leaving women defenseless against invading endometrial debris from the retrograde mechanism. Increased exposure to menstruation, in the form of early menarche, shortened cycles, and long, heavy menstrual periods, also appears to be related to disease development (Richardson, 1987). Others have speculated transportation of endometrial tissue via the lymphatic or blood systems, which would explain rare cases of endometrial implantation in the lungs, arms and thighs (Weinstein, 1987). Iatrogenic causes, suggesting accidental endometrial transplantation during pelvic surgery or other medical procedures have also been supported (Weinstein, 1987). The Endometriosis Association’s extensive research in exposure to environmental toxins and disease susceptibility found a link between dioxin exposure (a chemical byproduct of pesticide manufacturing, waste incineration, and other products) and endometriosis (Endometriosis Association, 2003). However, none of the current theories can explain the rare cases in which endometriosis has been discovered in women who have never menstruated and males undergoing long-term estrogen therapy due to prostate removal. Only four cases of endometriosis in males have been documented. In such instances, metaplasia has been hypothesized to occur, in that the cell retains its embryotic capability to differentiate into another type of cell (in this case, an endometrial cell) when stimulated by estrogen (Endometriosis Association, 2003; Honore, 1999; Weinstein, 1987). In light of the vast array of theoretical paradigms regarding disease etiology, some have speculated that more than one causal factor is likely in operation (Lamb, Hoffmann & Nichols, 1986).
Treatment

Unfortunately, endometriosis is without cure, and as was recently discussed at the World Congress on Endometriosis (1999), the disease is chronic and recurrent, no ideal medical treatment exists and treatment effectiveness is generally short-lived. In considering pain control treatment options, all have limitations and side effects, which truncate effectiveness and use. Treatment selection is dependent upon several factors, including age, extent of symptomology, parity, reproductive desire, and threat to vital organs (Bernhard, 1982; Weinstein, 1987). Initial treatments are generally hormonal and often result in temporary suppression or regression of symptoms (Weinstein, 1987). Such treatment is based on the assumption that the disorder is due to endometrial stimulation, thus, with initiation of a hormonal environment analogous to pregnancy or menopause, ovulation is inhibited and such stimulation is decreased (Weinstein, 1987; Yankauskas, 1990). Hormonal therapies include 1) androgen administration, 2) estrogen therapy, 3) hormonal therapy to induce pseudopregnancy (i.e. progesterone/estrogen birth control pills), 4) Danazol, which increases testosterone concentrations, and 5) gonadotrophin-releasing hormone agonists (GnRH) which induce a pseudomenopausal state.

Conservative surgical treatment also can be conducted with the goal of removal of all visible endometriotic implants while retaining reproductive ability. Laser excision is the primary method of such surgery, and has recently gained widespread popularity. Electrocautery, endocoagulation and sharp excision are additional, albeit less popular, options. Radical surgery is generally only conducted when vital organs are in jeopardy and is seldom necessary. This definitive measure generally includes complete hysterectomy and bilateral salpingo-oopherectomy as the treatment of choice for those...
unresponsive to other less extreme measures (Hurst & Rock, 1989; Rock & Moutos, 1992). Biofeedback for endometriotic pain has recently been explored and initial findings look promising (Hawkins & Hart, 2003), although a greater number of studies with increasingly rigorous methodological standards are required. Understandably, many of the available treatment options are viewed by sufferers as less desirable than disease symptomology, a common sentiment echoed throughout the literature.

The Problem of Pain in Endometriosis

Pain has taken a back seat to fertility in the endometriosis literature. A strong focus on fertility exists within the healthcare system and among the media, and it appears that both socially and within the medical field, the disease is constructed primarily as a fertility problem. Women with the disease, however, experience endometriosis primarily as a chronic illness and report pain as the most troubling symptom (Carpan, 1996). In a recent article, Campbell (2003) suggested that, although infertility is still touted as the main symptom of endometriosis, in reality patients with the disease present to their general gynecologists three times more often with pain than infertility. Thus, there appears to be clear disjuncture in what women with endometriosis experience and what popular literature and the medical profession emphasize.

There is a growing body of research literature to support pain as the most prominent disease symptom (Barlow & Glynn, 1993; Campbell, 2003; Canavan & Radosh, 2000; Damario & Rock, 1995; Momoeda, Taketani, Terakawa, Hoshiai, Tanaka, Tsutsumi et al. 2002; Vercellini, 1997). The high frequency of pelvic pain due to endometriosis has been acknowledged (Damario & Rock, 1995), and both endometriosis-
linked dysmenorrhea and chronic pelvic pain have been recognized as principle reasons for lost employment days. Damario & Rock (1995) remarked upon the frequent physical and mental disability that pain imposes upon endometriosis patients, such as a diminished ability to pursue physical activity and sexual relations, the potential for increased mood disturbances such as depression and anxiety, and further complications with health and functioning. Barlow and Glynn (1993) noted that pain is a particularly distressing symptom given that most treatment options are associated with its eventual recurrence, contrary to patient expectations of such procedures as curative. In fact, Weinstein has likened the process to a “treatment carousel,” with women going round and round in search of the ever-elusive pain relief or cure. Following numerous surgeries with little pain relief, patients often feel a sense of hopelessness, powerlessness, frustration, and experience emotional complications (Garner, 1997; Weinstein, 1988). Campbell (2003) suggested that our failures to solve the endometriotic pain problem are possibly rooted in 1) the stigma attached to pain and societal mandate to “tough it out,” 2) gender differences in pain perception and gender discrimination regarding pain in the medical system, and 3) the focus on infertility rather than pain when treating the endometriosis population.

*Relationship Between Organic Pathology and Report of Pain*

To compound the issue of endometriosis-related pain further, the current classification scheme for the disease (R-AFS) is a poor predictor of pain. Even this system, which purportedly evaluates extent of disease, was designed to predict fertility rather than pain (Roberts & Rock, 2003). The R-AFS staging of endometriosis was initially generated for use with infertility patients to predict reproductive outcome,
however it is now arbitrarily used in the non-infertility population (i.e. pain population) to stage disease (Muzzi, Marana, Pedulla, Catalano, & Mancuso, 1997; Roberts & Rock, 2003). Unfortunately, the R-AFS does not meet even the basic criteria for most malignant diseases on which the system was modeled (Roberts & Rock, 2003). Specifically, the current system is neither predictive of infertility (American Society for Reproductive Medicine, 1997; Roberts & Rock, 2003) nor chronic pelvic pain (Roberts & Rock, 2003).

This lack of relationship between organic pathology (as staged in the current classification) and pain reporting has presented the greatest challenge to the medical profession. Although studies evaluating pathological organic characteristics of endometriosis (i.e. lesion-activity, prostaglandin production, altered peritoneal environment) and their role in pain production have been conducted for quite some time (Hurst and Rock, 1991; Sturgis & Call, 1954; Willman, Collins & Clayton, 1976), it was only recently that exploration into the association between disease stage based on current classification scheme and pelvic pain commenced. Investigations into this line of inquiry emerged forcefully in the early 1990s, and this issue continues to be hotly debated today.

Factors hypothesized to be related to endometriotic pain in past studies have included those currently subsumed under the R-AFS classification and factors altogether ignored or underrepresented in this system. The R-AFS classification includes visual characteristics such as number and location of endometriotic adhesions, yet fails to consider issues such as histological characteristics, activity, depth, type, and morphological features of endometriotic lesions, all of which have demonstrated promising associations to endometrial pain (Cornillie, Oosterlynck, Lauweryns, &
Koninckx, 1990; Koninckx, Meuleman, Demeyere, Lesaffre, & Cornille, 1991; Ripps & Martin, 1991; Vercellini, Bocciolone, Vendola, Colombo, Rognoni & Fedele, 1991). The following will review the research in pathology and pain, with increased attention to studies that have utilized the current classification and a brief overview of investigations that have implicated factors outside of the current classification schema.

Several researchers have examined the relationship between extent of disease, defined as stage and localization of pelvic endometriosis, and pain (Fedele, Parazzini, Bianchi, Arcaini & Candiani, 1990; Fukaya, Hoshiai, & Yajima, 1993; Marana, Muzii, Caruana, Dell’Acqua & Mancuso 1991; Porpora, Koninckx, Piazze, Natili, Colagrande & Cosmi, 1999; Vercellini, Trespidi, De Giorgi, Cortesi, Parazzini, & Crosignani, 1996). In such studies, subjects are categorized according to the R-AFS classification, and R-AFS scores are subsequently related to self-report of pain. All researchers have concluded that stage and localization of the disease are not associated with frequency and/or severity of symptoms. This is the case even when global disease and singular/unique aspects of symptomology are evaluated independently. Marana et al. (1991) correlated the total score, active score and adhesion score separately when determining the relationship between extent of disease and symptomology (in an effort to increase specificity and clarity) yet even then, a correlation failed to emerge.

In that same year, Stout, Steege, Dodson, and Hughes (1991) explored the relationship between chronic pain self-report and organic pathology. A total of 102 women with scheduled laparoscopies were categorized by the AFS classification and administered a battery of pain assessment measures, including the following: Pain Questionnaire for Women, McGill Pain Questionnaire, and the West Haven-Yale
Multidimensional Pain Inventory. Participants were further categorized into either a pain (of any variety including dysmenorrhea, dyspareunia, pelvic pain) (N=90) or no-pain group (N=12). Results indicated that the total score on the AFS classification was significantly related to patient’s self-assignment into pain or no-pain groups, however the extent of organic disease as classified by the AFS was not linearly correlated with duration or frequency of pain symptom, rated levels of dysmenorrhea, menstrual phase pain, dyspareunia, days in bed or medication taken. Stout et al. (1991) suggested that the extent of disease as currently evaluated by laparoscopy is at best only minimally related to a patient’s rated levels of pain and functional impairment.

Fedele, Bianchi, Bocciolone, Di Nola and Parazzini (1992) questioned whether endometriosis was exclusively the cause of associated pelvic pain, given Fedele et al.’s (1991) previous finding regarding the lack of correlation between extent of disease and pain symptomology. Thus, to determine if the probability of pain symptomology was greater in endometriosis versus other patients, the prevalence and severity of dysmenorrhea, pelvic pain and deep dyspareunia was compared in infertile women with endometriosis and infertile women with normal pelvises. In infertile women with endometriosis, pain was related to disease stage and site according to R-AFS classification. No differences were found between patients and controls in frequency of dysmenorrhea, however, severity of the symptom increased with greater extent of disease, as women with stages III and IV endometriosis reported a greater severity of dysmenorrhea than controls and those in stages I or II. Pelvic pain was more severe in stages III-IV in comparison with stages I and II. Finally, dyspareunia was significantly more frequent in endometriosis patients than controls, regardless of disease stage. Fedele
et al. (1992) concluded that endometriosis in infertile women causes pelvic pain, the severity of which is associated with extent of disease. A major limitation acknowledged by the authors was that only infertile women were considered, thus the findings are not representative of the endometriosis population in general, most of whom are not infertile.

As part of a larger study exploring psychological aspects of endometriosis, Peveler, Edwards, Daddow and Thomas (1996) sought to correlate degree of pathology as classified by the AFS score and pain, assessed via an interviewer-rated pain index and a visual pain analog. No correlation emerged upon initial analysis, however, in further analyses, after dividing the endometriosis group at the AFS score median into two groups (i.e. high and low AFS scores), a significant relationship emerged. It was found that more severe endometriosis (i.e. higher AFS score) was associated with greater pain severity on the visual analog scale, however, again, no linear relationship emerged between AFS score and the pain index. Peveler et al. (1996) suggested differences in pain measurement methods might explain these and other contradictory findings.

More recently, Muzii, Marana, Pedulla, Catalano and Mancuso (1997) have found an association between current R-AFS score and one type of pain, dysmenorrhea. Preoperative questionnaires assessing dysmenorrheic levels along a 10-point analog scale were administered to the 65 participants with endometriosis and a control group of 15 infertility patients. In the endometriosis sample, surgeons recorded both total R-AFS scores, and partial revised AFS scores for superficial implants, deep endometriosis and adhesions. Superficial lesions were additionally classified as typical or atypical and were recorded in number. They failed to find a difference in pain scores for endometriosis patients and controls. Interestingly, however, in the endometriotic population, the R-AFS
classification score did significantly correlate with dysmenorrhea. It is important to note that authors failed to examine other types of endometriosis-related pain, such as dyspareunia and chronic pelvic pain, thus the usefulness of the current scoring system with regard to these other symptoms was not addressed and remains inconclusive.

Stovall, Bowser, Archer and Guzick (1997) took a unique approach, in that the relationship between history of chronic pelvic pain and stage of endometriosis was explored. Archival data was reviewed and 48 subjects meeting inclusion criteria were admitted into the study. All historical data, which was initially staged with original AFS classification, was meticulously restaged according to the current R-AFS system, including objective pathological findings and subjective pain intensity ratings. Subjects were then administered a questionnaire assessing current intensity and severity of pelvic pain and current localization of most severe pain. Analyses were conducted to determine the persistence of pelvic pain throughout the reproductive years, the association between disease stage, pain intensity, and location of pain at initial and follow-up evaluations, and finally, if location of pain varies across time. Interestingly, at initial evaluation, severity of pain did not correlate with disease stage. However, in those with pain at initial and follow-up evaluations (i.e. persistent pain), an association emerged between stage of disease and persistence of pelvic pain, such that advanced-stage endometriosis at initial evaluation was related to a higher degree of pain at follow-up. Stovall et al. (1997) concluded that chronic pain related to endometriosis frequently persists throughout the reproductive years.

In a recent and rather extensive multi-center study, Gruppo Italiano per lo Studio dell'Endometriosi (2001) examined the relationship between site, stage and
morphological characteristics of lesions and frequency and severity of pain symptoms. Participants consisted of 469 women undergoing initial diagnostic laparoscopy of endometriosis for the primary complaint of pain. Each subject was assessed for presence and severity of dysmenorrhea, deep dyspareunia and non-menstrual pelvic pain, and all were identified under R-AFS classification for endometriosis. The main finding suggested stage of endometriosis was not associated with presence and severity of pain. In terms of localization of lesions, no significant difference emerged between site of endometriosis and either severity of dysmenorrhea or frequency and severity of non-menstrual pain and dyspareunia. It is important to note the robustness and generalizability of these findings considering the rigorous methodology employed, including: both researcher and participant unaware of cause of pain (i.e. blind to laparoscopy) at time of assessment, results stemming from 3 major and independent centers in Italy, minimal refusal to participate (i.e. <5% of eligible patients), standard methods of data collection and disease staging and finally, senior researchers supervising staging of lesions as well as a system of checks and balances to ensure accuracy. Consistent with the majority of studies, Gruppo Italiano per lo Studio dell’Endometriosi (2001) did not find a significant association between stage, site and morphological characteristics of endometriosis and the experience of pain.

Recently, Momoeda, Taketani, Terakawa, Hoshiai, Tanaka, Tsutsumi et al. (2002) questioned whether pain is truly related to the pathophysiology of endometriosis, and sought to determine if the link between pain and pathology differs in endometriotic women with a primary complaint of pain versus fertility. Participants were staged according to the R-AFS classification and categorized by chief complain, either infertility
or pain. Severity of pain symptomology was assessed via self-report in graded increments (i.e. no pain, mild, pain tolerable without analgesics, etc.). Findings indicated severity of dysmenorrhea increased with disease stage only in the infertility, but not pain group. In contrast to much of the previously existing research, Momoeda et al. (2002) determined that an increasing incidence of chronic pelvic pain and dyspareunia occurred with increasing staging of the disease when comparing the infertility group and pain group individually, but the relationship did not emerge when the two groups were analyzed concomitantly. Authors speculated that the large number of contradictory studies in the past on pain and extent of disease might have suffered confounds due to inclusion-biases allowing all patients with endometriosis-diagnosis, unrestricted by symptomology, into the study. Momoeda et al. (2002) concluded that chronic pelvic pain, dyspareunia, and dysmenorrhea are indeed related to disease extent.

It is clear that many of studies attempting to relate the current classification system (which dictates extent of disease) and report of pain have found little to no association. In fact, of the 14 studies directly examining pain in relation to AFS classification, 8 have determined no linear relationship at all (Fedele et al., 1990; Fukaya et al., 1993; Gruppo Italiano per lo Studio dell’Endometriosi, 2001; Marana et al., 1991; Porpora et al., 1999; Ripps and Martin, 1991; Vercellini et al., 1991; Vercellini et al., 1996), 5 have found a weak association with some current classification variables (Fedele et al., 1992; Muzii et al., 1997; Peveler et al., 1996; Stout et al., 1991; Stovall et al., 1997), and only 1 has conclusively determined a linear relationship exists (Momoeda et al., 2002). Due to the contradictory findings regarding AFS classification and organic pathology, many researchers have explored other characteristics of endometriosis outside...
of this scheme that may more accurately predict pelvic pain. This is the current trend in literature, in which an association between endometriosis lesion-characteristics (i.e. morphological appearance, type of lesion, lesion histology/biochemical viability, lesion-activity, depth of infiltration) and pain has emerged. Note that the current classification system does not incorporate such factors, instead focusing on physical appearance of lesions, which may help explain the previous lack of association. The following will consist of a brief overview of such factors and their role in endometriotic pain prediction.

Depth of lesion infiltration (DIE) has demonstrated some success in its association with pelvic pain. In fact, deeply infiltrating lesions (defined as ≥ 5 mm) are so strongly and consistently related to pain that recently a surgical classification based on location of DIE has been proposed to treat pain symptomology (Chapron, Fauconnier, Vieira, Barakat, Doussset, Pansini, Vacher-Lavenu & Dubuisson, 2003). In one of the earliest studies investigating DIE, Comillie, Oosterlynck, Lauweryns and Koninckx (1990), measured histological characteristics and activity of lesions. Results indicated that cyclicity and activity of lesions was significantly different at varying depths of infiltration, with deep implants demonstrating high levels of cellular activity and the presence of very deep endometriosis (defined as greater than 10mm) strongly related to severe pelvic pain. Conversely, superficial lesions were most frequent in patients with primary complaint of infertility. Two separate studies correlating depth of infiltration with pelvic tenderness upon examination found increased depth of infiltration to be significantly associated with increased tenderness upon examination, thus implant volume is thought to be directly related to pain symptomology (Ripps & Martin, 1991 & 1992).
As further support, Koninckx, Meuleman, Demeyere, Lesaffre, and Cornille (1991) found that women with pelvic pain have larger lesions that infiltrate deeper and have an increased number as well as larger-sized endometriomas. Interestingly, in regression analyses, after depth of infiltration was accounted for, neither pelvic area affected nor presence or volume of endometriomas contributed additional significance toward the prediction of pelvic pain, thus depth of infiltration appears to be the prominent predictor of pelvic pain. More recently, investigators have determined that localization of DIE is associated with specific pain symptomology, suggesting lesion involvement in the pouch of Douglas in dysmenorrhea, uterosacral ligament involvement in dyspareunia (Fauconnier, Chapron, Dubuisson, Vieira, Dousset and Breart, 2002; Porpora et al., 1999), bowel lesions in chronic pelvic pain (Fauconnier et al., 2002) and rectal or vaginal involvement in dysmenorrhea (Chapron, Fauconnier, Dubisson, Barakat, Vieira and Breart, 2003). Importantly, deep endometriosis on the uterosacral ligaments has been found to be the strongest predictor of total pain, chronic pelvic pain and deep dyspareunia, and notably, it has been cited that DIE does not always correlated with a high R-AFS score (Davis & McMillan, 2003).

Although depth of lesion infiltration has received the majority of research attention with regard to pain symptomology and endometriotic characteristics outside any formal classification scheme, other variables have been investigated with some success, including lesion type, histology and morphological appearance. Vercellini, Bocciolone, Vendola, Colombo, Rognoni and Fedele (1991) conducted an investigation on the frequency of pain symptomology as it relates to morphologic features of peritoneal lesions. When the three lesion types (typical, atypical and mixed) were considered, a
relationship with deep dyspareunia emerged, such that pain was associated more frequently with typical and mixed rather than atypical lesions. No linear relationship between morphological lesion characteristics and either dysmenorrhea or intermenstrual pain emerged.

The number of endometrial lesions and adhesions in relation to pain has also been studied (Duffy & DiZerega, 1996; Perper, Nezhat, Goldstein, Nezhat, Nezhat, 1995). Perper et al. (1995) found that extent of visible disease is related to intensity of pain symptomology. With subcategorization of patients into high and low pain groups, those with low reported pain had significantly fewer implants than those with high pain, yet no relationship emerged between adhesion-site and any specific symptom. They concluded that intensity of menstrual pain is strongly related to number of implants. As further support for the role of adhesions in pelvic pain, Duffy and DiZerega (1996) conducted a review of clinical data addressing the topic, and concluded not only that adhesions frequently cause pelvic pain, but additionally, that removal of such adhesions decreases pain in 60-90% of cases.

Prostaglandin production has also been linked to pelvic pain. Vernon, Beard, Graves and Wilson (1986) suggested that biochemical viability and morphologic appearance of implants may be more important mechanisms in pain production than the purely physical attributes that characterize the current classification scheme. They found that prostaglandin synthesis in endometriotic implants was inversely proportional to disease stage, as categorized by AFS classification. The low correlation between current classification and pain symptomology was hypothesized to be a direct result of the omission of morphological status; thus a patient may exhibit a large extent of physical
disease, yet report no pain if her lesions are less biochemically active and vice versa. Findings by Willman, Collins and Clayton (1976) supported these views, as they determined that the mean concentration of prostaglandin was significantly elevated in women with dysmenorrhea and specifically in cases of endometriosis.

Clearly, the current classification system is flawed when pain evaluation and prediction is the sole intent, as many studies evaluating the association between the current R-AFS classification and pain symptomology found no relationship whatsoever. A more recent trend in the literature appears to be moving toward identifying associations of organic pathology and pain not currently accounted for in the R-AFS system, such as morphological appearance of endometriotic lesions, type and histology or biochemical activity of lesion, and depth of infiltration. In contrast to studies strictly employing AFS classification, the current movement appears to have demonstrated consistent success. In light of this, many researchers are pushing for modification of the current classification to incorporate such factors. Ripps and Martin (1992) suggested that a system of predicting pain in endometriosis should more heavily weight deep/expansive lesions and those in the cul de sac and uterosacral ligaments. Suggested revisions of the R-AFS (which currently differentiates superficial from DIE lesions without quantifying deep lesions) include proper quantification and weighting of depth of lesion (Chapron et al., 2003) and inclusion of biochemical activity of lesions (Vernon et al., 1986).

It appears that our understanding of painful symptomology in endometriosis, especially with regard to the relationship between pain and organic pathology is burgeoning. Nonetheless, the field is still nascent regarding mechanisms of pain action, and practitioners continue to feel frustration when endometriotic patients experience non-
remittent pelvic pain. In fact the pain-pathology relationship remains so ambiguous that recently criteria have been established to help guide diagnosis and the determination of whether endometriosis is the actual cause of pain (Hurd, 1998). The need for such criteria stems from the fact that as many as 15-43% of asymptomatic women have known pathology at surgery, and in some women with pathology and pain, endometriosis does not seem to be the causal mechanism of their pain. Further, even basic pathophysiology regarding endometriotic lesions and pain is absent (Bergqvist, 1995). Given the limited relationship between pain and pathology in endometriosis as compared to other painful conditions (i.e. cancer), there is a demand to incorporate factors outside of pathology into the paradigm of endometriotic pain.

Importantly, some of the leading researchers in the field of endometrial pain have acknowledged psychosocial variables as inextricably tied to the pain experience (Barlow & Glynn, 1993; Momoeda et al., 2002; Ripps & Martin, 1991 & 1992; Stout, Steege, Dodson, & Hughes, 1991; Vercellini, et al., 1996). Barlow and Glynn (1993) commented on the potential need for psychological, in addition to medical, treatment in endometriosis. Momoeda et al. (2002) suggested examination of psychological factors might be warranted, considering their potentially influential role in report of pain, especially in those reporting their primary complaint as pain. Additionally, the inherent difficulty in pursuing unbiased pain reports in patients due to the influence of psychosocial issues has been noted (Vercellini et al., 1996).
Psychological Factors Linked to Endometriosis and Chronic Pelvic Pain

Our current framework for understanding pain in general extends from a biopsychosocial model (Melzack & Wall, 1965). Currently, it is believed that quantity and quality of pain are multi-determined, and acknowledgement of psychological variables in addition to sensory input has been recognized. Merskey and Spear (1967) have speculated that pain threshold appears to be dependent on physiological factors whereas pain tolerance is more highly influenced by psychosocial factors. Weisenberg (1983) commented that although pain is an initially sensory experience, its perception is influenced by a range of psychological factors, including motivational, emotional and cognitive variables, thus psychological interventions have demonstrated relative success with regard to pain control. It is now widely recognized that psychosocial factors play a role in both pain perception and pain experience, and additionally that pain has an impact on psychological functioning (Jacob & Kerns, 2001). In fact, Jacob and Kerns (2001) criticized the current dualistic perspective of pain and psychological functioning, which generally contends that psychological functioning is either implicated as a cause or effect of pain, preferring a reciprocal and dynamic relationship between these two factors, rather than a linear, unidimensional one.

Given our current multi-factor and multi-disciplinary framework for pain perception, a logical area in research expansion is toward the exploration of psychological mediators in endometriotic pain. The push for research into this area is even more pressing in light of the fact that psychological distress has been previously associated with endometriosis specifically, as well as other painful gynecologic
conditions such as premenstrual syndrome (Morse & Dennerstein, 1992) and dyspareunia (Meana, Binik, Khaife & Cohen, 1998), and has been extensively linked with chronic pelvic pain (Fry, Beard, Crisp and McGuigan, 1997; Hodgkiss and Watson, 1993; Low, Edelmann and Sutton, 1993; Pearce, 1987; Peveler, Edwards, Daddow and Thomas, 1995; Waller and Shaw, 1995; Savidge and Slade, 1997; Shatford, Hearn, Yuzpe Brown, & Casper, 1988). Further bolstering the need for psychological assessment in endometriotic pain is the finding that psychological management (i.e. CBT) of chronic pelvic pain has already successfully occurred in an adolescent population with endometriosis (Greco, 2003).

In order to fully understand the endometriotic pain experience, we must evaluate the issue within a more holistic context. The first step toward this goal necessitates a thorough understanding of psychological variables that have been linked to pelvic pain in the past. Due to the limited psychological literature specific to endometriosis, consideration of research from related areas such as chronic pelvic pain is required. Thus, we will now draw upon existing literature, presenting a full review of all investigations involving psychopathology, mood, and personality in endometriosis, followed by an abbreviated review of these factors as they relate more generally to chronic pelvic pain (CPP).

**Psychiatric Morbidity**

One highly controversial area has been that of psychopathology associated with gynecological conditions. Lewis, Comite, Mallough, Zadunaisky, Hutchinson-Williams, Cherksey et al. (1987) examined psychopathology specific to the endometriotic population, finding that 12 of 16 consecutive women being treated for laparoscopy-
diagnosed endometriosis met DSM-III criteria for mood disorder (including bipolar, manic and major depressive disorder) as assessed by the Hamilton Rating Scale for Depression, the Schedule for Affective Disorders and Schizophrenia, the Beck Depression Inventory and DSM-III criteria. However, as Walker, Katon, Jones, and Russo (1989) pointed out, the potential for bias was great given administrators were not blind to the diagnosis of endometriosis, and Simon (1988) highlighted that because the design failed to include a control group of women with confirmed asymptomatic endometriosis, Lewis et al.’s (1987) conclusion that psychiatric disorders are etiologically linked to endometriosis was premature. Two years later, Walker, Katon, Jones and Russo (1989) compared 14 women with and 55 women without endometriosis utilizing the NIMH Diagnostic Interview Schedule (DIS) with interviewers blind to laparoscopy results and found no higher prevalence of affective disorders in the women with endometriosis.

Most of the research in psychopathology and gynecological conditions has centered on the broad area of chronic pelvic pain (CPP). In an initial study, Castelnuovo-Tedesco and Krout (1970) examined psychosomatic aspects of chronic pelvic pain, comparing women with and without chronic pain. Authors reported a striking degree of psychopathology in the pelvic pain group, both with and without organic pathology. Pelvic pain patients were found to display a mixture of schizoid, hysterical, depressive, and hypochondriacal features. However, a major limitation to the research includes the almost exclusive reliance on subjective measures (i.e. Rorschach, TAT and psychiatric interview) by clinicians who were not noted to be blind to subjects’ organic pathology.
Walker, Katon, Harrop-Griffiths, Holm, Russo and Hickok (1988) compared psychiatric profiles of 25 women with chronic pelvic pain and a comparison group of 30 women with infertility or tubal ligation concerns (pain-free comparison). Findings indicated a greater prevalence of lifetime major depressive and current major depressive illness in the pain group when compared to the no-pain group. Chronic pain patients also displayed a significantly greater mean number of positive somatic symptoms on thesomatization disorder scale of the Diagnostic Interview Schedule (DIS), and a significantly higher mean score than the comparison group on all SCL-90 scales except interpersonal sensitivity.

More recently, Fry, Crisp and Beard (1997) reviewed the literature on psychopathology and chronic pelvic pain, and found that several studies have demonstrated greater degrees of psychopathology or scale deviations from the norm in CPP populations, and such patients have been identified primarily as hostile. Considering that CPP patients are suffering from a disabling and distressing disorder that is often not taken seriously by the medical profession and rarely successfully treated, it is entirely possible that this hostility is a function of their frustration with their condition and its treatment (or lack thereof). There is no empirical research available, however, that teases cause and effect apart in the relationship between psychopathology and CPP.

*Psychological Distress and Personality*

Both personality and mood have been theorized to contribute to pain in general (Merskey, 1978) and more specifically, to pain in the pelvic region. Several researchers have evaluated personality and mood in women with known pathology (including endometriosis), those with pelvic pain of unknown origin, and pain-free controls (Beard,
Belsey, Lieberman and Wilkinson, 1977; Hodgkiss & Watson, 1994; Low, Edelmann & Sutton, 1993; Renaer, Vertommen, Nijs, Wagemans, and Van Hemelrijck, 1979). Renaer et al. (1979) found that both women with pain of known and unknown origins demonstrated significantly higher neuroticism scores than controls, whereas Beard et al. (1977) found neuroticism scores to be significantly higher in the no-pathology pain group than the control, with scores for the pathology group falling between those of the no-pathology pain and control groups. Beard et al. (1977) suggested that women with pelvic pain of no identifiable origin appear to be psychologically different from women without pelvic pain, however, it is important to point out that no significant differences were found between no-pathology and pathology pain groups. Low et al. (1993) found endometriosis patients to have greater introversion, higher psychoticism and increased state and trait anxiety than patients with pain of other origins, however, only anxiety scores were significantly elevated, reaching clinical levels. Hodgkiss and Watson (1993) found that their CPP sample demonstrated significantly greater depression and illness behavior than that found in the no pain control.

To evaluate whether women with identified pathology are in fact different from those with pain of unknown origin, both Pearce (1987) and Peveler, Edwards, Daddow, and Thomas (1996) compared the two groups; both studies found no significant differences in mood or personality. Interestingly, unlike in the Beard et al. (1977) study, Pearce (1987) found that women with both types of CPP did not differ psychologically to normative data. Pearce (1987) concluded that her findings failed to bolster previous speculations that pain is a result of personality or mood disturbance, and Renaer et al.
(1979) suggested that the most reasonable explanation for the findings was that ongoing chronic pain resulted in psychological distress.

Collins (1979) questioned whether certain personality variables might differentiate women with endometriosis from women with other types of organic infertility and normal fertility. The author compared 20 women with endometriosis to 20 women with infertility due to other organic causes and 20 fertile women. All subjects were administered the MMPI, Rorschach, and the Ford Personality Survey. This study found that endometriosis patients deviated from the other two populations exhibiting problems with: hostility and resentment, utilization of ineffective coping strategies (i.e. tended to use denial and repression), emotional and interpersonal difficulties (i.e. self-centeredness, emotional immaturity, emotional-oversensitivity, decreased awareness of own emotional needs, need to conform to social regulations and a tendency toward passiveness) and conflict of a sexual nature (i.e. sexual adjustment problems, gender conflict, and rejection of the feminine role). Several major limitations to Collins’s (1979) research may limit the validity of her findings. Much of her research methodology included subjective measures (i.e. structured interview and Rorschach), thus experimenter biased may have intervened. Additionally, she accepted an error rate (p value) of .15 to determine significance between groups. Given this is three times the acceptable standard, capitalization on spurious findings may have occurred due to inflation of type I error.

Rosenthal, Ling, Rosenthal and McNeely (1984) explored personality in 103 women complaining of CPP (17% with endometriosis) in a pain clinic setting. Unlike previous research, a large proportion of the sample (76%) was African-American, whereas only 24% were of Caucasian background. Findings indicated elevations on
several MMPI scales in 18.4% of the sample. Only 19.4% of the patients could be classified as having completely normal profiles. The most frequent finding was somatization, however non-specific psychological distress and a depression-like symptom picture emerged as well. Highly elevated F scores with inconsistent and varied symptoms were interpreted as one or all of the following: a method of eliciting physician-compassion (i.e. “cry for help”), attempt to simulate psychiatric illness, or inattention due to illiteracy. A limitation to the study includes the lack of a control group to assess whether low SES and education could be the main contributing factor to elevated MMPI profiles.

Shatford, Hearn, Yuzpe, Brown, and Casper (1988), explored personality and mood among patients with differential diagnoses for organic infertility (including endometriosis). Psychological functioning was compared in 348 candidates for in vitro fertilization falling into 5 diagnostic categories of infertility, including: tubal problems, endometriosis, idiopathic, partner factors such as low sperm count, and multiple factors. Results suggested that in terms of personality scores, women with endometriosis reported more extroversion than did patients with multiple factors or idiopathic diagnosis. Additionally, a trend emerged but did not reach statistical significance, suggesting that women with endometriosis respond to stress with more aggression and anger, as well as behave more impulsively than women in the other diagnostic categories. No differences emerged among the diagnostic groups on any other measures. Caution must be utilized when generalizing to the endometriosis population at large, considering infertility (not pain) was the primary complaint and only a small portion of the total sample had endometriosis diagnoses.
Personality characteristics have been hypothesized to influence pain perception in the endometriotic population (Gomibuchi, Taketani, Doi, Yoshica, Mizukawa, Kaneko, et al. 1993). Authors measured personality, defined as subject’s character and disposition, via the Rosenzweig picture frustration study (a questionnaire assessing the subject’s type of response to frustrating situations) and personality findings were subsequently correlated with pain manifestation (here defined as expression of dysmenorrhea). This study found that the profiles of women complaining of dysmenorrhea suggested exaggerated pain with little attempt at pain resolution. Alternatively, the profiles of subjects without dysmenorrhea suggested an unconscious refrain from the expression, or indifference to the pain, even in situations that generally evoke pain. Findings were used to support the notion that women without dysmenorrheic pain simply do not perceive or attend to pain, despite its presence.

Waller and Shaw (1995) explored whether differences exist between symptomatic versus asymptomatic endometriosis patients with regard to psychological symptomology. The Beck Depression Inventory (BDI), Speilberger State-Trait Anxiety Inventory, Golombok Rust Inventory of Sexual Satisfaction, and Endometriosis Symptom Questionnaire were administered to women undergoing laparoscopy for pelvic pain symptoms, infertility or sterilization. Both patients and doctors were blind to the laparoscopy results. Anxiety scores did not differ among the groups and were all within normal ranges, however women in both pain groups displayed higher BDI scores than both control groups. Women with symptomatic endometriosis did manifest mild signs of sexual dysfunction, as well as depressive symptomology, whereas women with asymptomatic disease did not display abnormal psychological functioning. Authors
concluded that the supposition that chronic pain may lead to abnormalities in psychological functioning is plausible.

Recently, reviews of the literature have begun challenging earlier findings of increased psychologic symptomology in the CPP population (McGowan, Clark-Carter and Pitts, 1998; Savidge & Slade, 1997). Methodological flaws such as small and biased samples, lack of laparoscopy, lack or inappropriate selection of controls, and comparison of groups unmatched for pain duration have been highlighted. In a robust meta-analytic review of psychological variables in the chronic pelvic pain literature, a total of only 22 studies met the rigorous inclusion criteria, and results indicated that in terms of psychological morbidity (defined here as depression, anxiety, neuroticism and psychopathology), no differences emerged between CPP patients with and without organic pathology (McGowan et al., 1998). However, when CPP patients were compared with pain-free subjects, depression was highly associated with CPP, and anxiety and neuroticism were also significantly higher in CPP than pain free groups. Interestingly, when compared to patients with other types of chronic pain (non-pelvic), levels of depression were not significantly different, suggesting that differences in levels of psychological morbidity may be due to presence of pain in general, rather than specifically to pain of pelvic origin.

Psychological factors clearly play an integral role in chronic pelvic pain, yet controversy abounds as to the exact nature of that role. Researchers continue to debate whether such psychologic symptomology is a contributory to or resultant factor of pain. As pointed out in a review of chronic pelvic pain, research in general pain syndromes has implicated certain personality traits, coping strategies and health beliefs in the
predisposition of an individual toward the development of chronic pain (Moore & Kennedy, 2000). On the other side of the fence, researchers have suggested psychologic symptomology is more accurately viewed as a consequence rather than cause of the disease. Proponents of this view have elucidated the pitfalls to investigating psychopathology as a contributing factor in disease development, suggesting that separating cause from consequence is especially problematic, an issue that prevails even in such rigorous methodologies as longitudinal studies (Low & Edelmann 1991).

Regardless of the nature of the association, psychological and mood variables have been definitively linked to chronic pain populations, including endometriosis.

Not only do women with endometriosis experience a chronically painful condition, which naturally predisposes itself to increased psychological distress, but many will face the additional fear and increased distress of potential or actual infertility problems. In fact, it has been reported that at least 33% of women with a diagnosis of endometriosis will encounter fertility difficulties at some point in their history of the disease (Ballweg, 1995). Thus, it is important to explore the role that infertility plays in its relation to psychological distress.

**Infertility Concerns**

Pain and infertility have been acknowledged as the two most devastating aspects of endometriosis. Interestingly, much of the psychological distress and infertility literature, like the literature regarding distress and endometriosis, questions whether infertility is a result of or a contributory factor in psychological distress (Greil, 1997).

In one review of the literature, it was suggested that most researchers now presume infertility is a source rather than the cause of psychological distress (Greil,
Several themes in the qualitative research were identified in the review, including 1) infertility as a strong focus for identity, 2) feelings of loss of control and defectiveness, 3) stress in marital relationship, 4) feeling stigmatized or isolated from the “fertile world,” and 5) stress from treatment procedures as well as strained relationships with healthcare providers. Quantitative studies suggest that: 1) infertile couples differ in general psychological distress from norms either moderately or not at all, 2) the literature on depression and self-esteem is divided between studies that have found moderately (non-clinical) elevated levels of depression and lower self-esteem among the infertile as compared to non-infertile and those who have not, and 3) marital satisfaction of infertile couples is equal to or higher than that of fertile couples. In their recent review of the psychological and psychosomatic aspects of infertility, Henning and Strauss (2002), also confirmed these findings, citing neither significant personality differences nor increased prevalence of psychological disorders in infertile as compared to fertile women; some studies even indicated more psychological stability in infertile women. Clearly, the quantitative studies paint a different picture than qualitative research, however, methodological limitations such as inadequate sampling procedures, questionable controls and primitive statistical procedures have been highlighted, potentially accounting for part of the discrepancy.

It has been acknowledged that most of our current information regarding the psychosocial effects of infertility is based on clinical impressions by physicians and therapists (Andrews, Abbey, & Halman, 1991). The fact that qualitative research, as well as clinical reports and anecdotal comments regarding the distress level of infertile couples, does not correspond to quantitative research has been a continued area of
frustration for researchers (Edelmann & Connolly, 1998). One potential answer to this conundrum is that qualitative measures might be measuring psychiatric morbidity, whereas couples experiencing fertility concerns/treatment may instead experience a more mild "infertility strain reaction." Edelmann and Connolly (1998) sought to explore this possibility by administering both standardized measures to assess psychopathology (administered at initial assessment and 7-month follow-up) and weekly diaries to assess psychological strain in infertile couples (subjects recorded their "psychological state" once a week for a period of 22 weeks). In line with previous research, psychopathology was not evidenced in the sample either at initial or follow-up session. In contrast with expectations, results from the weekly report also indicated a general lack of psychological strain, suggesting minimal distress. The authors attempted to explain their confusing results by highlighting the wide variability in psychological strain reported in the diaries. The top 10% of the sample reported significant distress and also appeared to have higher psychopathology scores on the standardized measures, thus it is possible that these couples are more likely to openly discuss their distress, which may subsequently be reported in clinical/anecdotal reports.

In a study that same year, Markestad, Montgomery, and Bartsch (1998) evaluated the psychological, marital and sexual adjustment of 20 couples undergoing medical treatment for infertility. Not unlike previous research, no significant findings emerged on SCL-90-R scores for wives as related to their age, length of marriage and length of medical attention. In terms of number of medical treatments and psychological distress, there was very little reported distress in couples undergoing extensive treatment (i.e. those in treatment over 2 years). In fact, SCL-90-R symptomology scores on 7 subscale
scores, including depression, anxiety and hostility all decreased as the number of treatments the wife received increased. Similarly, Anderson, Sharpe, Rattray and Irvine (2003) found that couples newly referred to a fertility specialist clinic reported low levels of emotional distress, even when specifically asked about concerns related to the fertility problem. These findings are not consistent with Henning and Strauss's (2002) review, citing several studies indicating that medical treatment for fertility, especially with regard to repeated treatment failure, has been strongly associated with psychological distress. Nasseri (2000) also found increased distress to be a typical response to fertility treatment cross-culturally. Markestad et al., (1998) suggested their findings of low psychological distress at later points of treatment may reflect a high level of couple cohesion and an emotional adjustment in recognizing, dealing with, and eventually overcoming strong emotional reactions associated with the crisis. Levin and Sher (2000) have also recently addressed some of the potential reasons that infertile couples (especially those presenting for treatment) may be reporting such low levels of psychological and marital distress on standardized tests. They suggest infertility may create a strong bond between the couple, serving to combat marital distress, couples may report healthy levels of marital satisfaction and psychological functioning to avoid denial for medical treatment, and/or those who are presenting and remaining in infertility treatment (i.e. those who are most often studied), might be couples who are psychological healthy in general.

Although distress in infertile populations has provided a divided literature between quantitative and qualitative methodologies, one research group has devised a questionnaire that is both controlled and targeted specifically at distress related to fertility (Abbey, Andrews & Halman, 1991), in direct contrast to standard attempts to link

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infertility to standardized measures of global distress (e.g. SCL-90). Several investigations utilizing the Fertility Problem Stress measure suggest that women with fertility problems do in fact experience distress as a direct result of their fertility problems and such stress is: 1) strongly negatively related to marital life quality for both infertile men and women (Abbey, Andrews, & Halman, 1994), 2) negatively correlated with perceived internal control and self-esteem in infertile women (Abbey, Andrews, & Halman, 1992) 3) is higher in infertile women than their husbands (Abbey, Andrews, & Halman, 1991; 1995; Andrews, Abbey, & Halman, 1991) 4) is related to greater marital conflict, sexual dissatisfaction, and lower sexual self-esteem, as well as quality of life in infertile couples (Andrews, Abbey, & Halman, 1991), and 5) is positively related to number and cost of fertility tests and treatments in infertile couples (Abbey, Halman, & Andrews, 1992). It appears that with measures that specifically target fertility-related rather than more general global distress, we can see a narrowing of the gap between quantitative and qualitative results.

It seems likely that women with endometriosis are experiencing psychological distress both as a consequence of a chronically painful disease and with its association with known or potential fertility problems. How do such women deal with elevated levels of both pain and distress? Research suggests their choice of coping style greatly impacts their adjustment to chronic illness, as coping is believed to play an extremely important role in how patients adapt (Grant, Long, & Willms, 2002).

Coping and Chronic Pain

Currently, many researchers have turned to the investigation of two specific coping strategies, active and passive coping. An active coping strategy is defined as that
of a patient who attempts to control his or her pain or to function in spite of it.

Conversely, passive coping is defined as that of a patient who relinquishes control of pain to others or allows many areas of life to become adversely affected by pain (Snow-Turek, Norris, & Tan, 1996). It is thought that active coping is associated with more positive outcomes, such as good psychological adjustment and affect, whereas passive coping has been linked to high pain levels and psychological distress. We will now briefly evaluate the passive and active coping literature with specific emphasis on chronic pain populations.

In an extensive review of coping and chronic pain, Jensen, Turner, Romano and Karoly (1991) reported that passive coping has been linked to increased levels of pain severity, depression and disability, in contrast to active coping. In fact, active coping has been found to predict decreased levels of depression and functional disability up to 6-months after initial assessment whereas passive coping has been found to predict the inverse relationship. Passive coping has been positively related to depression, but only in patients reporting high levels of pain, a finding that has been replicated in longitudinal studies across a 6-month duration as well as cross-sectional designs. Jensen et al. (1991) additionally reported that pain patients’ cognitive beliefs directly influence their pain coping-style. Perceived helplessness has been associated with a passive coping style, whereas a strong internal rather than external locus of control was associated with utilization of active coping strategies.

Since the Jensen et al. (1991) review, active and passive coping styles have continued to be consistently associated with psychological and physical functioning. Arraras, Wright, Jusue, Tejedor and Calvo (2002) found passive coping to be related to
increased anxiety and depression in their sample of heterogeneous chronic pain patients, suggesting that a passive coping style is associated with poor pain adaptation. Both Nicassio, Radojevic, Schoenfeld-Smith, and Dwyer (1995) and Weickgenant, Slater, Patterson and Atkinson et al. (1993) found that a passive coping style was associated with increased levels of depression in a sample of chronic low back pain (Weickgenant et al., 1993) and fibromyalgia patients (Nicassio et al., 1995). Also examining a chronic low back pain population, Klapow, Slater, Patterson et al., (1995) found that subjects with good pain-control relied less on passive coping strategies, while those with chronic pain syndrome reported increased reliance on such strategies. Similarly, Novy, Nelson, Hetzel, Squitieri and Kennington (1998) found that higher levels of pain control in a chronic pain population were associated with greater use of active coping strategies.

In one study evaluating the effect of active and passive coping on functioning in a pain management clinic-population, Snow-Turek, Norris and Tan (1996) determined that passive coping demonstrated a strong positive association to psychological distress and depression, whereas active coping was positively associated to activity level and negatively related to psychological distress. Similarly, in the first study to evaluate the relative contribution of coping style to adaptation within a breast cancer population, an active coping style significantly predicted decreased disability and depression, whereas passive coping significantly predicted the inverse. Interestingly, the study found no association between anxiety and coping style (Bishop & Warr, 2003). As further support for the influence of coping style on (dis)ability, Evers, Kraaimaat, Geenen, and Bijlsma (1998) found that passive coping strategy as identified immediately following diagnosis of rheumatoid arthritis was associated with a decrease in functional status (e.g., mobility,
self-care and grip strength) one year later. Active coping strategy, however, was not predictive of functional status.

More recently, coping style has been linked to level of pain intensity. Hellstroem and Anderberg (2003) found passive coping to be linked with increased frequency of high levels of pain severity in daily pain diaries of women with chronic low-level pain, while Watkins, Shifren, Park and Morrell (1999) found that subjects with rheumatoid arthritis reported increased use of active coping strategies when faced with mild pain and more maladaptive strategies in the context of severe pain.

Participation in psychological and medical treatment has also been shown to impact coping style. In two separate studies, Nicholas, Wilson and Goyen (1991, 1992) found that the use of active coping strategies was improved after subjects with chronic low back pain underwent either cognitive or behavioral treatment (Nicholas Wilson & Goyen, 1991) or cognitive behavioral treatment plus physiotherapy (Nicholas, Wilson & Goyen, 1992), and that improvement in coping strategy was maintained up to 1 year later. Flor and Birbaumer (1993) found patients with musculoskeletal pain who underwent biofeedback treatment demonstrated an increase in active coping self-statements at posttreatment, 6-month and 24-month follow-ups. Congruent with these findings, both Evers, Kraaimaat, Floris, van Riel et al., (2002) and Newton-John, Spence and Schotte (1995) have found that after cognitive-behavioral treatment, participants with rheumatoid arthritis demonstrate improvement in active coping post-treatment (Evers et al., 1995; Newton-John et al., 1995) and at 6-month follow up (Newton-John et al., 1995).

So, it is clear that coping strategies are strongly related to psychological and physical functioning, and that passive coping appears to be negatively related while
active coping is positively related to optimistic adjustment to illness. However, psychological factors can influence not only how we perceive and respond to/cope with our pain, but additionally, how we remember it (Jamison, Sbrocco, & Parris, 1989; Lefebvre & Keefe, 2002; Linton, 1991). An equally pressing if not precursory issue in the exploration of endometriotic pain that has not yet been explored is the accuracy with which women report their pain. This issue, although investigated quite extensively in other chronic pain populations such as arthritis and back pain patients (Bolton, 1999; Eich, Reeves, Jaeger & Radford, 1985; Erskine, Morley, & Pearce, 1990; Feine, Lavigne, Dao, Morin & Lund, 1998) and to a small extent within the gynecological population (Brodie & Niven, 2000; Niven & Murphy-Black, 2000) has been altogether ignored with regard to endometrial pain.

Pain Recall and its Correlates

The importance of assessing the accuracy of pain recall is particularly important in the case of endometriosis, as these reports are the primary guides to the treatment and management of the condition. It is standard practice for physicians to rely on patients’ retrospective recall of pain to determine treatment. A growing body of literature currently attests to the importance of patient pain report as the primary indication for surgery and prescription dosage in endometriosis. Pain has been highlighted as the primary indication to initiate pharmaceutical treatment in women with endometriosis (Bergqvist, 1999; Canavan & Radosh, 2000; Damario & Rock, 1995; Thomas, 1995), although selection of specific medication is dependent primarily on side effects and cost (Mahutte & Arici, 2003). Both nonsteroidal anti-inflammatory drugs (NSAIDs) and oral
contraceptives are utilized with the initial report of endometriosis-associated pain
(Mahutte & Arici, 2003). If women don’t respond to either medication, more aggressive
hormonal therapy or surgery is considered, as alternation from one oral contraceptive or
NSAID to another is not beneficial. Progestins or GnRh agonists are considered second-
line treatment, indicated if patient report of pain continues after a period (generally 3-
month trial) of NSAID or oral contraception. As further support for the considerable role
patient self-report of pain plays in treatment, pain relief is widely accepted as the primary
measure of medical treatment success in clinical settings (Damario & Rock, 1995;
Mahutte & Arici, 2003).

When first-line pharmaceutical treatments fail to relieve pain, surgical treatments
are considered. Fliegner and Umstad (1991) determined that the most common
indication of presacral neurectomy (surgical interruption of pain fibers from the uterus)
was secondary dysmenorrhea, generally in association with endometriosis or pelvic
adhesions. Severe pelvic pain has been found to be the only indication for endoscopic
excision of deep endometriosis in 67%, 78% and 76% of women with Type 1, Type 2 and
Type 3 lesions respectively (Koninckx, Timmermans, Meuleman, Penninckx, 1996). In a
study examining predictors of hysterectomy, the strongest indication for surgery was
consulting a physician about a menstrual problem that included chronic or persistent
pelvic or abdominal pain (Treloar, Do, O’Connor, O’Connor, Yeo, & Martin, 1999). The
authors concluded that their results support the general tenet that women have
hysterectomies predominately for bleeding, pain or both. Clearly, the need for pain report
to be established as a reliable measure is paramount given its central role in treatment
planning.
Not only is accuracy of pain recall important from the pain management/clinical standpoint, but researchers also rely on this factor as a dependent measure in most pain studies. Thus, if pain recall is grossly inaccurate and this inaccuracy goes undetected, it can lead to both unnecessarily invasive and ultimately unsuccessful treatment as well as to faulty research conclusions, especially regarding the relationship between pain and pathology. This is particularly important considering that our understanding and definition of endometriosis is moving closer toward an exclusive reliance on pain reporting. As mentioned previously, a movement has occurred in treating endometriosis in which it has been suggested that only symptomatic (i.e. pain reporters) women should be considered cases (Balasch, Creus, Fabregues, Carmona, Ordi, Martinez-Roman et al., 1996; Hurd, 1998). Thus, if women are not accurate reporters of such pain, this will justifiably spur inquiry into how we view and define, as well as diagnose, endometriosis.

In addition to accurately assessing experienced pain, it is also important to understand the psychological factors that impact pain recall. Psychological mediators of pain recall accuracy, if identified in the endometriotic population, may prove to be useful indicators for treatment planning. This section now turns its focus to the main area of the current study, the pain recall literature. Primary attention will be granted to investigations with other pain populations, as there have been no investigations of this issue with endometriosis specifically.

Accuracy of Pain Recall in the General Pain Literature

Erskine, Morley and Pearce (1990) conducted a thorough review of the memory for pain literature, spanning over 30 studies and addressing a number of inherent difficulties in pain recall. Pain populations included individuals with: chronic continuous
pain of unspecified cause, pain due to a range of pathology, headache, rheumatoid arthritis, acute coronary pain, labor pain, dental pain, and experimentally or clinically induced pain. One of the strongest trends in the literature identified subjects as frequently inaccurate in retrospective reports of pain, most often in the direction of overestimation with only rare underestimations of pain at recall. One study in the review found no tendency to underestimate or overestimate recalled pain when examining mean scores, however when data was examined at the individual level, considerable inaccuracy was present. Several factors appeared to influence accuracy of recall, including present pain state, mood, emotional distress, method utilized to assess recall, and duration of pain. Although a number of studies have demonstrated reasonable accuracy of recall, their reliance on correlational analyses between actual experienced pain and recalled pain is a flawed indication of accuracy, as this method dictates only relative order of the two correlated variables (Erskine et al., 1990) and not the absolute reproducibility of values obtained on two occasions (Morley, 1994). In other words, two sets of data may have identical slopes, yet differ in their intercept of the y-axis. Authors characterized the research on a whole as deficient with regard to methodological quality and theoretical underpinnings, and in need of reform.

Given that assessment of treatment outcome for painful conditions is highly dependent upon recalled pre-treatment pain levels, Peine, Lavigne, Dao, Morin and Lund (1998) evaluated the potential for treatment-efficacy overestimation as a function of exaggerations in recalled pre-treatment pain. A total of 61 individuals with chronic myalgia of the jaw were randomly assigned to 1 of 3 groups: 1) treatment group- wore oral appliances covering biting surface of teeth 24 hours per day for entire study period,
2) control group 1- wore oral devices covering only hard palate for same time period and
3) control group 2- wore oral appliance for only 30 minutes during weekly treatment
appointments. During the 10-week study, the present pain level, recall of initial pre-
treatment pain rating, and estimated pain relief were assessed periodically in each subject.
Results indicated that errors in initial pain recall occurred bi-directionally, such that
patients with initially low pain ratings overestimated their pre-treatment pain and those
with high pre-treatment ratings underestimated initial ratings at recall. As hypothesized,
present pain levels and recalled pre-treatment pain significantly predicted reported pain
relief. Interestingly, both perceived pain relief and true pain relief (defined as pre-
treatment minus present pain) increased with time, however, perceived relief was
significantly greater than true relief. It appears that errors in pain recall increased with
time and were dependent on levels of pretreatment pain and pain present at recall.
Authors concluded that memory in chronic pain populations is generally inaccurate,
frequently in the direction of overestimation. Inaccurate pretreatment pain estimation
also appears to contribute to the tendency to report pain relief, thus reports of relief do
not necessarily reflect treatment efficacy and are inadequate indicators of true changes in
pain.

McGorry, Webster, Snook, and Hsiang (1999) took a more long-term approach
when evaluating pain accuracy recall. Subjects with either chronic or episodic lower
back pain (LBP) completed a daily pain diary method for a period of 18 months, of which
only data from the final 6 months were evaluated. Daily diaries asked for the patient’s
assessment of a single daily pain score on a 0- (no pain) to 10- (extreme pain) point scale.
At the completion of the study period, subjects were asked to recall the number of pain
free days in the previous week, month and six-month periods, and completed the Modified Zung Depression Index and Modified Somatic Perception Questionnaire. Results indicated that 1-week and 1-month data for diary and recall did not significantly differ. However, at the 6th month period, a significant difference emerged, with 29 subjects underestimating days in pain, 11 subjects accurately recalling pain, and 10 demonstrating overestimation. Importantly, all 11 with exact estimation reported lower back pain for every day of the recall period, thus accuracy in this group was expected, as variability was nonexistent. Variables such as age, psychological distress level, pain location, level and fluctuation, as well as pain history and pattern demonstrated no significant effect on pain recall accuracy. One major limitation to the study was the significant amount of missing data on which to base analyses. Because exit interviews (which included the recall-data) were frequently scheduled after a large time-gap following last diary entry, the researchers included only 16 participants for the 1-week recall, 30 for the 1-month recall and 50 for the 6-month recall. Thus, although inaccuracy in recall was not found for the 1-week and 1-month periods, the analyses for this timeline were based on a small subset of the original sample.

Given the historical trend to utilize inappropriate statistical methods in the examination of pain recall accuracy (i.e. correlational analyses), Bolton (1999) set out to evaluate accuracy in back pain patients utilizing more appropriate methodology (i.e. agreement statistic). Participants completed pain diaries for a 7-day duration, in which they recorded pain levels the same 4 times each a day. On the 8th day, patients recorded their current pain level and were asked to estimate (recall) level of pain during the previous week at its least, worst, and usual/average. Findings indicated that current pain

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at recall was lower than both recalled and actual average pain levels. Although patients with higher levels of current pain tended to overestimate recalled pain and those with lower levels of current pain to underestimate recalled pain, the trends failed to reach statistical significance. In fact, no significant difference emerged between recalled and actual average pain levels, thus subjects appeared to be accurate in their memory for pain. One caveat to this study is the real-world applicability of the findings. Given that the recall period was limited to 1 week, it is difficult to say how accurately this reflects real-world demands on recall. For example, a physician requesting a recalled estimate of a patient's pain over a 3 or 6-month period may understandably feel less confident in the patient's ability to recall.

Also based on a one-week recall period, Stone, Broderick, Shiffman and Schwartz (2004) more recently assessed accuracy of pain recall in 68 chronic pain patients. Participants were asked to electronically record their pain several times per day based on a random beep prompt over a two-week timeframe. After a period of one week, participants were asked to recall their “usual” pain over the prior week, which was subsequently compared to their electronic recordings. Paralleling previous findings, Stone et al. (2004) determined that pain recall was significantly inaccurate in the direction of overestimation at recall. The authors posited that at recall, subjects might consider only the moments when in pain, negating non-pain occurrences when estimating their average. Thus, authors computed the average pain in the electronic recordings excluding moments when participants were not in pain and found that pain recall corresponded more closely to this computation. Stone et al. (2004) also asked participants to estimate the change in pain (if any) between the two weeks of recording.
participants were inaccurate in this estimation as well. Over 40% of the sample reported pain to be worse in the final week, yet the recorded change in pain between the two weeks was non-significant. Clearly the importance of such findings has carryover effects to the medical setting. Patients are often asked to estimate change in pain from one office visit to the next, the time-interval between can range dramatically. Given that individuals appear to be inaccurate in this task, medical procedures and treatment based on such measurement may include inherent error.

Factors Influencing Accuracy of Pain Recall

Considering the potential importance of pain recall accuracy in determining medical treatment in a variety of pain populations, attention has turned to the role of mediating factors. Some factors such as general psychological distress, anxiety, coping style and pain and mood at time of recall have demonstrated associations with pain recall (Jamison et al., 1989; Kent, 1985; Lefebvre & Keefe, 2002; Linton, 1991; Bryant, 1993; Holroyd, France, Nash, & Hursey, 1993). Other factors, such as personality style, have not demonstrated any association with pain recall. Rofe and Algom (1985) examined personality in connection with pain recall in an OB/GYN population. Subjects included 235 pregnant women, immediately assessed post-delivery on over-all pain intensity along a 5-point verbal rating scale (no pain, weak, moderate, strong and extremely strong). Subjects then recalled their previous pain rating 24 to 48 hours later, at which time personality was measured. The majority of the patients reported none or weak pain at both times, however, a non-significant tendency to overestimate the pain at recall did emerge. Authors cautioned that the analyses did not specify amount or direction of change that may have occurred in the ratings of individual subjects, and that in
examination of consistency of individual responses over time, only 45.4% showed no discrepancy in their ratings. In those who demonstrated a change in rating from time 1 to 2 (i.e. “shifters”), none of the measured psychosocial traits were characteristic of this group. It was concluded that memory for pain is fairly accurate, regardless of personality type. However, it is important to note that this study examined acute (i.e. labor pain) rather than chronic pain, and as several researchers have pointed out (e.g. Jamison, 1989), sufferers of these types of pain are two divergent populations. Additionally, this study examined accuracy of recall for a short time span, and it has been suggested that accuracy of recall may be less consistent across longer periods of time (Kent, 1985).

In general, an association between psychological distress, (including anxiety, depression and general emotional distress), and accuracy of pain recall has been found (Gedney & Logan, 2004; Jamison et al., 1989; Kent, 1985; Lander, Hodgins, & Fowler-Kerry, 1992). Tasmuth, Estlanderb and Kalso’s (1996) study is one exception to this rule, in that these authors found memory of acute postoperative pain intensity after surgery for breast cancer was not influenced by the development of mood disturbances (i.e. depression or anxiety). Kent (1985), however, did find an association between distress and pain recall accuracy in a dental population. Participants were assessed on expected level of pain during dental procedure via a Visual Analog Scale (VAS) and were administered the Dental Anxiety Scale (DAS), which identified those who were high in anxiety and those who were low in anxiety. Following the dental procedure, patients indicated the degree of pain actually experienced. After a period of 3 months, patients were mailed the DAS and VAS measures and asked to recall amount of pain actually experienced during the procedure. Results indicated that pain recall was more highly
correlated with the patient's level of expected pain as reported before the procedure than actual pain experienced, a finding consistent with that of Dannecker, Price, and Robinson (2003) who determined that recalled pain (muscle) was associated with participant pain-expectations. Although all patients in the Kent (1985) study tended to report more pain in recall than was actually experienced (i.e. overestimation), this was particularly true of those in the high anxiety group. It was speculated that memory of pain is reconstructed over time in order to concur with existing anxiety levels. Similarly, Lander, Hodgins, and Fowler-Kerry (1992) also found that overestimation of pain was related to anxiety in a sample of 138 children.

Jamison et al. (1989) also found an association between both physical and psychosocial factors and pain recall in 93 patients with chronic pain of various origins (i.e. back pain, abdominal pain, headache). All patients recorded pain intensity for one week utilizing hourly pain rating cards. At the completion of one week, patients reported their current pain intensity and recalled their average pain intensity rating four times (i.e. 8am, 12pm, 6pm and 10pm) during the day for the previous week. All subjects additionally completed a comprehensive pain evaluation questionnaire and the SCL-90 to assess general emotional distress. Most patients overestimated pain levels. Contrary to other studies, no relationship emerged between pain level at recall and accuracy of pain estimate. Several factors emerged as most predictive of inaccuracy in pain recall, including disharmony in the home, low activity levels, and higher levels of emotional distress. Gedney and Logan (2004) also found distress was related to pain recall in their study experimentally manipulating pain with a cold pressor task. Specifically, authors determined that emotional distress present at the time of pain experience (due to
experimental manipulation) mediated the ability of actual pain levels to predict pain recall at 6-month follow-up. Clearly emotional factors appear to influence memory for pain intensity, with distress predisposing patients towards inaccuracy in pain recall.

Two studies have found that coping styles (including helplessness and catastrophization) appear to play a mediating role in pain recall accuracy (Lefebvre & Keefe, 2002; Linton, 1991). Linton (1991) explored psychosocial factors influencing accuracy of pain recall in chronic pain patients (N=61). Patients with lower back pain rated intensity of pain three times per day on a 100 mm Visual Analog Scale (VAS) for a one-week duration. After a period of 18 months (i.e. follow-up), participants were asked to recall the intensity of their pain utilizing the same VAS scale. During this follow-up interview, subjects were also administered questionnaires assessing depression level, functional level, current pain and sleep quality, helplessness, and psychosocial work environment. An average was computed for actual pain recordings and was compared to the recalled pain VAS. Results indicated that 70% of subjects had overestimated recalled pain. Only 16% had slight discrepancies (e.g. 5mm or less), and could be considered “relatively accurate” in recall. Differences between actual and recalled pain intensity were then correlated with the psychosocial measures. Findings indicated that only helplessness and psychosocial work environment (specifically relations with workmates, social support at work, influence over work, and amount of work to be done) were significantly related to accuracy of recall, however the direction of the association remained undisclosed by the author. Linton (1991) suggested that internal factors like coping (i.e. helplessness) and environmental factors (i.e. work) influence accuracy of
pain recall and urged continued research into the role of psychosocial variables in memory of pain.

Lefebvre and Keefe (2002) explored the influence of pain catastrophizing, (defined as the tendency to ruminate upon, overestimate the threat of, and negatively evaluate one’s ability to deal with pain), on accuracy of pain recall in rheumatoid arthritis patients (N=45). It was hypothesized that high catastrophizers would recall pain more accurately than those with low catastrophization due to increased vigilance to pain. Participants completed daily pain diaries for a 30-day period, recording a single rating of average pain each day. Following the diary period, a single evaluation session assessed current pain level ratings and pain recall over the diary period. Recalled pain was conducted via use of the Original Pain Recall Assessment (OPRA), a visual measure which has a reproduced 100-mm visual analog pain scale on the y-axis and the day of the month along the x-axis. Participants were asked to reproduce the pattern of their average daily pain during the entire diary period via use of a single continuous line from left to right. Level of pain catastrophization was assessed via the Coping Strategies Questionnaire (CSQ). As predicted, individuals who engaged in pain catastrophization were more likely to recall general pain intensity and changes in intensity over the 30-day period accurately. Pain level at time of recall and variability in diary-reported (actual) pain also influenced accuracy, such that those with higher pain were less accurate in recall and those with higher variability were more accurate in recall of changes in pain intensity over time. The authors posited that high catastrophization, which has been associated with increased somatic awareness, may lead to greater accuracy simply because pain is more salient and might therefore result in better memory encoding.
The factor most consistently associated with accuracy in pain recall is pain at time of recall (Bryant, 1993; Holroyd, France, Nash, & Hursey, 1993). Holroyd, France, Nash and Hursey (1993) evaluated recalled pain of headache sufferers with specific interest in pain present during assessment. As part of a larger study, subjects participated in an initial evaluation and were assessed on current pain state and provided a global report of headache activity, which required subjects to retrospectively assess “typical” headache frequency and duration. Daily headache recordings were then obtained for a one-month period for 146 individuals who completed the initial evaluation, in which subjects documented actual pain intensity and duration, number of headaches, and peak headache intensity. Findings revealed that subjects who reported pain at initial evaluation retrospectively reported more frequent headaches during the previous month than patients who were pain free at assessment. Interestingly, daily recordings of actual headache activity indicated that both individuals with and without pain recorded similar levels of headache activity. Authors concluded that global retrospective reports of activity are influenced by pain state at time of recall. One limitation that must be noted, however, is that the authors did not actually determine retrospective report of pain for the period of time that daily recordings were taken. Retrospective reporting of “typical” headache activity occurred before the one-month recording period. Thus, although one can speculate that a retrospective report of “typical” headache features would be consistent with recall at the conclusion of daily recording period, it may have been this was an uncharacteristic month in terms of headache activity (i.e. unusually high or low in headache activity).
A second study that same year individuals with chronic pain (N=30) recorded their pain levels “any time they noticed a change in their pain levels” via portable electronic diaries for the duration of one week (Smith & Safer, 1993). Subjects were matched for type of pain (i.e. back pain, headache) and randomly assigned to either the immediate physical therapy group (PT) or control group. The PT group received therapy before they were tested for pain memory accuracy, while the control group was tested first and then received therapy. It was predicted that PT patients would report less present pain than the control group at time of recall (i.e. low present pain) because this group had been instructed in pain management strategies. It was speculated that the PT patients would therefore recall pain as less severe than would control patients who had not received therapy (i.e. higher present pain). As expected, the PT group rated significantly less present pain at time of recall than the control group. Congruent with speculation, the two groups differed when asked to recall usual pain level during previous week, with the control group reporting a significantly higher level of usual pain than did the PT group, an overestimation on the part of the control group and an underestimation of pain in the PT group. It was concluded that the results provide strong, experimentally-derived support for the hypothesis that recall of past pain intensity and duration is influenced by present pain level.

Although previous research had focused on current pain and affect ratings on memory for pain, Bryant (1993) investigated the influence of changes in patient pain and affect upon pain recall. Utilizing visual analog scales (VAS), he assessed current pain (sensory and affective) and affect (i.e. anxiety and depression) levels of patients enrolled in an outpatient pain management group in an initial consultation. Subjects then
participated in a 6-week cognitive behavioral group treatment program designed to manage pain and stress through relaxation, cognitive distraction, visualization and positive self-talk techniques. At the final treatment session, subjects were asked to complete VAS measures identical to those at initial contact and were subsequently asked to complete another VAS packet in which they recalled their pain and affect at initial contact. Data was analyzed in three parts, including: 1) changes in VAS at initial contact and final treatment session, 2) accuracy of recall, and 3) factors influencing memory bias. Although a decreasing trend emerged, there was no significant difference in affective ratings of pain and depression between initial contact and final treatment session. Recall of pain and mood ratings, although overestimated, also did not reach a significant difference between initial contact and final session (i.e. when recall was assessed). Patients who reported an increase in sensory or affective ratings of pain at final session overestimated and those reporting a decrease at final session underestimated the severity of their sensory or affective pain at recall. It was concluded that memory for pain is susceptible to distortion, and pain and mood at recall influence such distortion. Further, the authors speculated that chronic pain patients might be particularly susceptible to the influence of changes in pain rather than its absolute values with regard to memory distortion simply because of the chronicity of their condition. Without clearly defined episodes of pain (as in acute conditions), any remediation or worsening of their condition may generalize current to past states when recalled.

Bryant’s (1993) conclusion that both mood and pain at recall are influential in distortion of pain accuracy was only partially supported by Salovey, Smith, Turk, Jobe and Willis’s (1993) findings that same year. In a series of six experiments, authors found
that only pain present at recall affects accuracy of recall. Subjects reporting a high level of pain at recall consistently overestimated their recall of pain intensity. In a separate experiment, authors found that mood, even when experimentally manipulated (happy, sad, or neutral), did not affect subjects' memory of pain intensity for their most recent painful experience.

In a recent review of the literature for memory of labor pain, the factor consistently linked to accuracy of pain recall was pain present at time of recall. This review was conducted with several objectives, including examining whether recall is accurate and the factors that affect accuracy (Niven & Murphy-Black, 2000). Some studies reported accurate recall of pain during both short (i.e. 2-week) and long periods of recall. Other studies demonstrated a decrease in pain at recall, especially when only the first or second stage of pain is considered. The decline in pain estimation at recall was speculated to potentially reflect a halo effect, given that pain/negative aspects of childbirth contrast with the positive event of a baby's birth. Only one study offered evidence that pain recall is inaccurate in the direction of overestimation. However, considering this study was the strongest methodologically and the only to assess pain during labor, its conclusions should perhaps be given more weight. Factors that have been shown to influence memory of labor pain include pain present at recall, peak level of pain during labor, and biochemical status of mother (i.e. hormonal levels) at recall. It was concluded that accuracy of labor pain recall remains controversial, as studies have found accurate, underestimation, and overestimation of pain at recall. Several methodological problems were addressed in the review, including the fact that most studies failed to assess pain during childbirth, especially in the later stages, and relied on
short-term recall immediately following birth to compare with memory of pain. Additionally, patients are frequently asked to recall pain at several stages of labor, a daunting task considering each stage fluctuates in pain level. Until memory for labor pain is determined accurate, Niven and Murphy-Black (2000) advocate that researchers discontinue reliance on pain recall.

Development of a chronic pain condition has also been shown to influence accuracy of pain recall. Tasmuth, Estlanderb, and Kalso (1996) set out to determine if the memory of acute postoperative pain intensity after surgery for breast cancer was influenced by chronic pain following the postoperative period. Patients undergoing either modified mastectomy or breast conserving surgery were assessed 1 day prior to surgery as well as 1, 6 and 12 months following surgery. Subjects were asked at 1, 6 and 12-month follow-up interviews to rate severity of the acute postoperative pain as they remembered it via a five point verbal rating scale (i.e. no pain, slight, moderate, considerable or severe). Questionnaires assessing anxiety and depression levels were administered pre-treatment as well as at 1, 6 and 12-month follow-up. Patients were also assessed regarding presence or absence of preoperative or chronic post-treatment pain. Authors failed to actually measure postoperative pain levels (i.e. immediately following surgery), thus number of doses of postoperative analgesics was utilized as an estimate of postoperative pain. Results indicated that women with chronic pain following surgery recalled postoperative pain levels that were significantly greater than women without chronic pain. Memory of postoperative pain-intensity increased with time in those with chronic pain whereas it decreased in those without chronic pain. In all patients preoperative anxiety and depression levels were higher than those found in healthy
populations, however, a year after surgery those elevations had returned to normal levels in all but women with chronic pain, whose levels remained elevated even a year after surgery.

In conclusion, it appears that the memory for pain literature has provided contradictory results, with some studies reporting accuracy in recall (Rofe and Algom (1985; Salovey et al., 1993; Von Korff, 2001), and others reporting underestimation (Niven & Murphy-Black, 2000) or overestimation (Erskine et al., 1990; Feine et al., 1998; Jamison et al., 1989; Kent, 1985; Linton, 1991, Stone et al., 2004) of pain at retrospective report. The majority of studies have reported overestimation of pain, and of the few studies demonstrating pain underestimation, considerable positive outcome of pain (e.g. childbirth, athletic competition) is the most frequent explanation for this effect (Salovey, Sieber, Jobe, and Willis, 1994).

Some of the contradiction in the recall accuracy literature may be a direct result of the difficulty inherent in pain assessment and the differing methodology employed to assess recall (i.e. different pain populations, pain quality vs. pain intensity, 1 day vs. 1 month recall). Several researchers have highlighted these problematic issues, which warrant consideration when exploring pain recall accuracy. Erskine, et al. (1990) addressed just a sample of the difficulties currently plaguing this line of research, including: 1) the near impossibility of recalling the sensory quality of pain, a topic that continues to be explored (Guastadisegni, 1997), 2) problems with the validity of recall measures in assessing subjective pain states, and 3) study reliance on verbal descriptors of pain, perhaps evaluating subject's memory of the words rather than memory for the pain. Although historically patients have been requested to provide an average of their
pain for the recall period (i.e. a single pain rating), several researchers have expressed doubt that patients have the ability to compute such a mathematical expression accurately (Erskine et al., 1990; Lefebvre & Keefe, 2002). In contrast, others have empirically determined that a single rating representing patient’s “on average” pain estimate is an accurate measure of actual “average pain intensity” (Bolton, 1999). Some researchers have suggested that because recalled pain reports are highly influenced by what people theorize pain to be, less obvious measures of pain memory, such as willingness to accept a previously endured pain (WTAP) might be more desirable to measure pain memory (Read & Loewenstein, 1999).

Additionally, it also appears that several factors mediate accuracy of recall, including anxiety (Kent, 1985; Lander et al., 1992), disharmony in the home, low activity levels, and higher levels of emotional distress (Jamison et al., 1989) helplessness and psychosocial work environment (Linton, 1991), pain at recall (Bryant, 1993; Smith and Safer, 1993; Holroyd, France, Nash & Hursey, 1993) and mood at recall (Bryant, 1993), whereas other researchers have not found psychosocial factors, including personality (Rofe & Algom, 1985) and mood (Salovey, Smith, Turk, Jobe and Willis, 1993; Tasmuth, Estlanderb & Kalso, 1996), to be related to pain recall. The one factor that seems to be not only consistently tied to accuracy of pain recall, but has also been the focus of the majority of studies, is pain at the time of recall. Salovey, Sieber, Jobe, and Willis (1994) have suggested that present pain may affect recall in two ways, by anchoring pain (e.g. participants utilize current pain as springboard for evaluating past pain) and/or by cuing pain (e.g. participants’ present pain may cue memories for previous painful situations and make those memories more salient). With only one exception
(Jamison et al., 1989), pain present at recall has demonstrated a clear association with recall accuracy.

CURRENT STUDY AIMS

In the present study, accuracy of pain recall and its correlates was investigated in a sample of women reporting to have endometriosis. Given the reliance on retrospective pain reports in disease definition and treatment, assessing the accuracy of pain recall and its correlates is likely to be central to effective diagnosis and treatment planning. Much of the general pain literature suggests that pain recall is generally inaccurate, a surprising finding given that such studies frequently employ a diary method, which by virtue of the assessment process, naturally increases vigilance to the pain. If patients are inaccurate in recall even when highly pain-vigilant, the validity of real-world pain recall (i.e., physician asking patient to estimate average pain over one month with no recording period) may be questionable. If patients with endometriosis are indeed inaccurate in their pain recall to the extent that they overestimate, this may result in unnecessarily invasive treatments and their attendant physical and psychological complications. Thus, in the present study, our first aim was to investigate pain recall accuracy for a one-month period in women reporting to have endometriosis. Although it was expected that a significant proportion of women would be inaccurate in their pain recall, especially in the direction of overestimation, we did not posit a hypothesis as to the exact distribution of pain recall accuracy in this population.

Additionally we investigated potential psychological mediators of pain recall accuracy. Given the tenuous relationship between organic pathology and pain, it seems
relevant to explore other factors associated with the pain experience. If in fact the experience and recall of endometriotic pain is strongly mediated by psychological factors, these could be targeted therapeutically with the aim of avoiding more invasive treatments. More specifically, this study focused on four such potential mediators: psychological distress, concerns about fertility, coping style, and current pain level at the moment of recall.

Because psychosocial distress has been linked to overestimation of pain at recall in other pain populations (Jamison et al., 1989; Linton, 1991; Lander et al., 1992), our first hypothesis was that decreased psychological wellbeing would predict pain overestimation (i.e. high estimation) at recall. Our second hypothesis concerned pain and fertility distress. Given that the two are repeatedly emphasized as the primary concerns in women with endometriosis, we posited that memory for pain and fertility distress would be associated. Thus, our second hypothesis was that increased fertility distress would predict high estimation at recall. This likely association has never been tested in any pain population, including women with endometriosis. Because active coping has been associated with positive adjustment to pain, our third hypothesis was that active coping would predict low estimation of pain at recall. Our fourth hypothesis was that passive coping, a non-adaptive response to chronic pain, would predict high estimation at recall. Our fifth hypothesis was that pain present at time of recall would predict the accuracy of pain recall. It was expected that the higher the pain present at recall, the more likely there would be high estimation of past pain.

Outside of our formal hypotheses, two other areas were investigated. First, previous studies have shown a link between actual pain-level experienced (i.e. pain
intensity) and both psychological distress (Castro, 1997; Naidoo & Pillay, 1994; Zautra, Marbach, Raphael, & Dohrenwend, 1995) and coping style (Estlander, 1989), therein isolating actual pain as an important variable that must be assessed and accounted for. Thus, the current study examined whether a relationship existed between actual pain (average pain reported over the 30-day diary period) and any of our predictor variables, and we planned to include actual pain in a factorial discriminant function analysis, if warranted.

Second, because a paucity of information exists regarding what factors might influence the accuracy of pain recall, this study included an analysis to determine if pain recorded at any one particular time of the month (i.e. beginning, middle or end of month) is more salient and therefore more influential in predicting the average pain that patients recall experiencing.
CHAPTER 3

METHODOLOGY

Participants

Although 114 participants initially began the study, 108 followed through to completion. Of these 114 initial participants, four failed to respond to efforts to meet for the follow-up, one insisted that the VAS daily pain record was not an appropriate measure of her pain and she discontinued participation. Another participant withdrew from the study due to scheduling difficulties. Of the six women who failed to complete the study, demographic background information was available for five participants, and although the statistics are compromised by a small sample size, analyses indicated that they did not differ significantly on any of the demographic variables from participants who did complete the study protocol. Of the 108 women who completed the study, seven participants were excluded because they were over the age of 40 and unlikely to have fertility concerns despite being pre-menopausal. Additionally, one case proved to be a univariate outlier, with a recall accuracy ratio score of 21.10, well above 3 standard deviations from the mean (M = 1.68, SD = 1.20). Given the undue influence this single participant might have had over the statistical analyses, a decision was made to exclude her data. Consequently, analyses were limited to 100 women who self-reported as having been diagnosed with endometriosis and who also reported chronic pelvic pain associated with the endometriosis.

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Recruitment strategies were varied and included flyers on university and community college campuses and gathering places (i.e. shopping malls, community boards, church bulletins, fitness clubs) in Las Vegas and San Diego, postings in two Ob/Gyn clinics, and on a variety of internet sites (including online endometriosis support groups and OB/Gyn sites), newspaper ads, and a listing of the study in the Psychology 101 subject pool at the University of Nevada, Las Vegas. A cost-free pain management session targeting adaptive adjustment to chronic pain was offered as compensation to all participants. Participants from the Psychology 101 classes were additionally compensated with the fulfillment of the course research requirement. Inclusion criteria for the study included: 1) self-report of a prior diagnosis of endometriosis from a medical professional; 2) a symptom profile including chronic pelvic pain; and 3) pre-menopausal status. Exclusion criteria included: 1) hysterectomy (as defined by the removal of uterus); 2) bilateral oopherectomy; 3) other medical conditions that could reasonably account for chronic pelvic pain; and 4) self-report of a diagnosis of any other pain syndrome. Pre-menopausal status, hysterectomy and oopherectomy were exclusion criteria because fertility distress was one of the dependent variables.

Table 1 lists the socio-demographic characteristics of participants and other background variables related to endometriosis history and treatment, as well as recruitment sources. The average age was 29 years, with 83% of the sample between 19 and 35 years of age. The majority were also Caucasian (80%), college-educated (93%), and married (53%) with no children (71%). The average lifetime duration of pelvic pain was 11 years and time since diagnosis of endometriosis at the time of the study averaged close to 5 years. Of those participants who reported experiencing onset of pelvic pain

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before their endometriosis was diagnosed (N=94), the average delay until diagnosis was 6.4 years.

Procedure

Formal approval for this study was obtained from the Institution Review Board (IRB) Committee at the University of Nevada, Las Vegas (see Appendix A). Once women contacted the researcher and expressed interest in participation after a brief description of the study, they met with the primary researcher in person or were interviewed and instructed by phone. At this time participants signed the informed consent (see Appendix B) in person or signed, scanned, and emailed it back as an attachment (the original copy was obtained when the complete packet was mailed back). The background measure was completed and participants were instructed in the use of the pain diary, wherein they were asked to monitor their daily pain levels over a period of 30 days. All participant questions were addressed at this time. A follow-up appointment was then made for one month hence. Participants utilized the daily pain diary method to record a single “average” rating of pain intensity on a daily basis for a 1-month period. Women were instructed to place the pain diary record into a provided envelope immediately after each daily entry in an attempt to prevent diary-review. During the diary recording process, participants were called once a week as a reminder to complete the pain diary and to address any questions that may have arisen since the initial session. Until the exit interview, subjects remained unaware that they would be asked to recall their pain at a later time. After the completion of the diary phase, participants met with the primary researcher in person (or over the phone if participant was out of state) for the exit interview, and turned over their pain diaries. At this time, patients were assessed on
current pain level and were administered coping, psychological wellbeing and fertility distress measures. Finally, they were asked to attempt to recall their average pain over the diary period. For those women who resided out of state, their packets were mailed back to the primary investigator immediately following their exit interview over the phone.

Measures

Background Measure

The background measure consisted of a one-page questionnaire inquiring into the following areas: 1) age, 2) marital status, 3) number of children, 4) ethnicity, 5) educational background, 6) length of time since diagnosis, 7) history of treatment, 8) duration of pelvic pain, 9) average number of annual Ob/Gyn visits, and 10) referral source (see Appendix C)

Pain Assessment

Daily Pain Measurement

The daily diary of pain intensity consisted of 30 individual visual analog scales (VAS), one for each day of the diary period (see Appendix D). The VAS was also utilized in the exit interview to assess subject’s current pain level (see Appendix E) as well as their pain recall (see Appendix F). The VAS consists of a 100 mm-length line (vertical line in this study) representing a continuum of pain intensity. The top end of the scale is anchored at “worst pain imaginable” and the bottom end of the scale is anchored at “no pain.” Patients indicated their pain level each day by marking the line at that point which best represented their overall level of pain that day. The range of scores is thus 0
to 100. Use of the VAS in this diary format has been supported in a variety of studies evaluating pain recall (Bolton, 1999; Linton, 1991; Lefebvre & Keefe, 2002; McGorry; Webster Snook & Hsiang, 1999). Importantly, reactive effects to pain diary assessment have not been demonstrated (Bolger, Davis & Rafaeli, 2003; Cruise, Broderick, Porter, Kaell, & Stone, 1996; Salovey, Smith, Turk, Jobe, & Willis, 1993). The reliability of VAS scores has been cited between 0.95 and 0.99 for literate groups, however the reliability for horizontal scale scores was non-significantly lower than that of vertical scale scores. It has been suggested that a vertical scale is preferable to a horizontal scale, and that neither adjectives (i.e. severe, moderate, slight pain) nor numbers be placed along the length of the line, as a clustering of responses along these arbitrary points tends to occur (McDowell & Newell, 1996).

Validity of VAS scores is moderately strong. Correlations of the VAS and 4 to 5-point verbal descriptive scales of pain have been cited to be between 0.71 and 0.81 (McDowell & Newell, 1996). The VAS has been correlated with the McGill Pain Questionnaire (Melzack, 1975; a multi-dimensional pain measure) at 0.60 to 0.63. In general, the VAS has been identified as more sensitive to change in pain than verbal rating scales. The VAS has also been touted as preferable to descriptive pain assessment given that it yields a more precise and sensitive measurement (McDowell & Newell, 1996).

Accuracy of Pain Recall

Subjects’ accuracy in recalling intensity of their pain was calculated by determining the discrepancy between actual pain levels (from diary record) and recalled pain levels. The 30 daily diary pain recordings for each participant were arithmetically...
averaged to generate a single “average actual pain” rating for the month. Participants also provided us with their single-point “recalled average pain” for the one-month period. Research has supported utilizing a patient’s single-point “on average” pain as a measure of recall. Bolton (1999) suggested that this measure is more real-world valid and, in her sample of 200 back pain patients, she found that patients’ recalled average pain was an accurate measure of their actual average pain intensity over a one-week period.

Although most of the research into pain recall has utilized change/difference scores, it is widely understood that such measurement is unreliable, a sentiment recently reiterated within an article specific to pain recall accuracy (Stone, Broderick, Shiffman, & Schwartz, 2004). Thus, a ratio score was instead selected to assess accuracy of pain recall, utilizing the recalled average pain score as the numerator and the actual average pain score as the denominator for each participant. Subsequently, any deviation from a ratio score of 1.0 is considered inaccurate pain recall, with a score below 1 representing underestimation and a score above 1 representing overestimation of the pain experienced over the past 30 days. For the purposes of this study, accuracy of pain recall was treated as a dichotomous variable using the median-split to distinguish those with high estimation (i.e. greater ratio scores) from those with low estimation (lower ratio scores); both groups were identified as such throughout the study.

Psychological Distress Assessment

Coping Strategies Questionnaire (CSQ)

Coping style was assessed via the Coping Strategies Questionnaire (CSQ: Rosenstiel & Keefe, 1983) (see Appendix G). Each of the 42 items in this version of the measure are accompanied by a 6-point scale assessing how frequently subjects utilize
certain coping strategies, anchored at 0 (never do) to 6 (always do that) (Lorig, Stewart, Ritter, Gonzalez, Laurent, & Lynch, 1996). The CSQ is the most widely utilized measure in assessing pain coping strategies (Stewart, Harvey, & Evans, 2001; Swartzman, Gwadry, Shapiro, & Teasell, 1994). The CSQ was generated utilizing a sample of chronic low-back pain patients and is composed of 7 scales, including 6 cognitive strategies (coping self-statements, catastrophizing, diverting attention, ignoring pain sensations, praying or hoping, reinterpreting pain sensations) and one behavioral strategy (increasing activity level). The psychometric properties of the measure appear to be relatively strong (Main & Waddell, 1991; Rosenstiel & Keefe, 1983; Spinhoven, Ter Kuile, Linssen & Gazendam, 1989). However, the original 8-factor theory-driven structure proposed by Rosenstiel and Keefe (1983) has been challenged; the measure seems to be more accurately conceptualized as a 5-factor model (Swartzman, Gwadry, Shapiro & Teasell, 1994; Tuttle, Shutty, & Degood, 1991). Additionally, several researchers have suggested evaluating the CSQ in terms of individual scales rather than as composite scores, stating that composite scores do not allow for a clear understanding of the relationship between adjustment and specific coping strategies (Geisser, Robinson, Henson, 1994; Swartzman et al., 1994). Face validity has been supported, as Swartzman et al., (1994) determined that professionals in the area of chronic pain patient-care accurately classified CSQ items into their corresponding categories. When compared with three other cognitive measures of pain, the CSQ was highlighted as the most ideal coping strategies instrument (Main & Waddell, 1991), yielding an 86% (moderate to substantial) concordance of individual items in a 24-hour test-retest period. Main and Waddell (1991) cited test-retest correlations between 0.88 to 0.95 on 6 of the 7 scales.
Others have deemed all 7 subscales to have satisfactory reliability, reporting Cronbach alphas ranging from 0.67 to 0.78 (Spinhoven, Ter Kuile, Linssen & Gazendam, 1989). Concurrent validity of the CSQ has been demonstrated, with statistically significant correlations (r = 0.41 to r = 0.55) between the subscales of the CSQ and the Chronic Pain Coping Inventory (CPCI: Hadjistavropoulos, MacLeod, & Asmundson, 1999), a conceptually-similar measure.

With regard to coping style, the CSQ can be utilized to assess whether individuals exhibit an active or passive coping strategy in dealing with their chronic pain. As mentioned previously, an active coping strategy is characterized by the patient’s attempt to control his or her pain or to function in spite of it. Conversely, passive coping is characterized as the patient relinquishing pain control to others or allowing many areas of life to become adversely affected by pain (Snow-Turek, Norris, & Tan, 1996). The passive subscale scores ranged from 0 to 72, whereas active subscale scores ranged from 0 to 180; in both cases greater scores indicated greater utilization of either coping strategy. In terms of the measure’s average scores on the passive or active coping subscales, a study methodologically similar to our own reported a passive coping mean of 25.06 and active coping mean of 75.11 (Lefebvre and Keefe, 2002). The CSQ has been found to be an appropriate measure of active and passive coping (Nicholas, 1988; Nicholas, Wilson, Goyen, 1991; Nicholas, Wilson, Goyen, 1992; Snow-Turek, Norris, & Tan, 1996). A factor analysis conducted on CSQ subscale scores of 132 chronic back pain patients utilizing the active/passive division supported this scoring system (Nicholas, 1988). In our sample, Cronbach alphas for the active and passive coping scales were 0.90 and 0.84, respectively, indicating strong internal consistency.
Rand Mental Health Inventory

To assess psychological wellbeing, the Rand Mental Health Inventory, 18-Item (MHI-18) scale was administered (Stewart, Ware, Sherbourne et al., 1992) (see Appendix H). This scale is an alternative abbreviated form of the full 38-item MHI. Several abbreviated forms of the MHI exist, including 5-item, 15-item, 17-item and 18-item versions of the scale. The 17- and 18-item measures are identical, with exception of the omission of one item regarding ability to relax without difficulty. Given that the majority of psychometric data is available on the full MHI, a lengthy description of the properties of the full-item scale will be followed by a brief description of those specific to the 18-item abbreviated form.

The original MHI was derived to measure both psychological distress and wellbeing, and has been endorsed for use with medical populations (McDowell & Newell, 1996). Two separate scores and/or one overall Mental Health Index can be generated; this study will utilize the overall score, with higher scores indicating greater psychological wellbeing. The MHI is not intended to measure severe psychiatric symptomology, but rather to assess the more prevalent symptoms of depression and anxiety (Ware, Manning, Duan, Wells, & Newhouse, 1984). The measure has been extensively evaluated psychometrically. Test-retest reliability for a one-year time period was originally established on a sample of 3,525 participants, and ranged from 0.56 to 0.64, depending on the scale. Internal consistency has been cited between 0.83 and 0.92 for the 5 individual scales, and 0.96 for the overall scale. Although the divergence between internal consistency and test-retest reliability is salient, it appears sound to posit that such disparity may reflect the nature of the population under consideration. Given
that health status varies in those afflicted with chronic medical conditions and that
distress can fluctuate concomitantly (Frazier, 2002), it is not surprising that test-retest
reliability proved rather low, especially over a one-year time frame. Perhaps such
findings speak more to the instability of distress in this population rather than the
psychometric properties of the measure.

Validity of MHI scores has also been established. Factor analysis has identified 2
higher-order factors, termed psychological distress and psychological wellbeing. The
factors correlate at -0.75, and are viewed as two ends on a continuum of distress versus
wellbeing (Veit & Ware, 1983). Additionally, the MHI also correlates well with other
measures and criteria indicating psychological wellbeing, such as mental health service
usage and life satisfaction measures (McDowell & Newell, 1996), as well as stressful life
events, social support, a history of care for emotional problems, physical illness, and
general health perceptions (Ware, Manning, Duan, Wells, & Newhouse, 1984). The MHI
is promulgated as incorporating the most appropriate questions from some of the leading
mental health questionnaires currently available, and as a promising measure for medical
non-psychiatric populations (McDowell & Newell, 1996).

The MHI-18 abbreviated form is comprised of 18 items and assesses frequency of
emotion (i.e. anxiety and depression) over the previous month. This version contains at
least four items from each of the anxiety, depression, behavioral control, and positive
affect subscales of the original MHI (McDowell & Newell, 1996). Responses to the
items range from 1 (all of the time) to 6 (none of the time) and eight items are reverse-
scored. A composite score ranging from 18 to 108 is computed, with higher scores
indicative of greater psychological wellbeing. When comparing the MHI 15-item and
MHI 18-item forms, as well as the 30-item General Health Questionnaire (GHQ: Farmer & Harvey, 1975) and 28-item Somatic Symptom Inventory (SSI: Barsky, Wyshak, & Klerman, 1986) in detecting psychological distress against criterion diagnosis using the Diagnostic Interview Schedule (DIS: Robins, Helzer, Croughan, & Ratcliff, 1981), the MHI-18 outperformed all other scales in detecting any DIS disorder. Additionally, both forms of the MHI performed as well as the GHQ and outperformed the SSI in detecting depressive symptomatology (Berwick, Murphy, Goldman et al., 1991; Weinstein, Berwick, Goldman, 1989). Although internal consistency and normative sample means for the 18-item scale are not available, consistency for the 17-item scale has been reported between 0.94 - 0.96, depending on the sample, and the MHI-17 had a total mean score of 72.8 in Stewart et al.’s (1992) sample of 3,053 individuals. As a general note, it has been suggested that overall scores be utilized, as the stability of subscale scores has been questioned with abbreviated forms of the MHI (McDowell & Newell, 1996). In our sample, the internal consistency of the MHI-18 was high, with a Cronbach’s alpha of 0.93.

Fertility Problem Stress

The Fertility Problem Stress scale (FPS: Andrews, Abbey, & Halman, 1991) was utilized to assess the amount of stress and life-disruption related to fertility problems in the past year (see Appendix I). The FPS scale has a total of 9 questions, three of which assess the general or overall impact of fertility stress (e.g., “How much has your life been disrupted because of this fertility problem?”) and 6 of which assess how stress impacts specific aspects of one’s life (e.g., “How much stress has your fertility problem placed on your sex life?”). Questions are answered via a 5-point Likert-type scale, ranging from
none at all (1) to a great deal (5). Scores range from 9 to 45, with greater scores indicative of increased fertility distress. Support for the scale structure (i.e. domains assessed) were selected and based on pilot interviews with infertile couples. Cronbach’s coefficient alpha was cited at 0.88 (Abby, Halman, & Andrews, 1992). In terms of normative mean scores for the measure, Abby, Andrews and Halman (1995) reported that the average stress level for each item was 2.5 for infertile women, and 2.1 for both infertile men and fertile women/men. The FPS scale is one of the few measures to assess stress in a variety of areas as related specifically to fertility, yet it has not been extensively utilized. In our sample the internal consistency of the FPS was high, with a Cronbach’s alpha of 0.91.

Missing Data

Missing data will be addressed under one of two categories: 1) in the VAS daily pain record and 2) in the questionnaires. As a general note on the entire data set, no single subject evidenced greater than 3 missing data points, and outside of the VAS for day 30 (12 cases having no values for that day), all other variables had missing values on less than 5% of cases. Thus no single variable was deleted.

With regard to the VAS data, a number of women were unable to meet exactly on day 30 due to scheduling difficulties. These subjects elected to complete the exit interview either before or after their target date, resulting in VAS values for either slightly less or more than 30 days. When scheduling difficulties did arise, every effort was made to meet with women after the target date rather than before. A total of 8 subjects met on day 29 instead of day 30 (i.e., 1 missing data point), two concluded the
study on day 28 (i.e., 2 missing data points), and 2 concluded the study on day 27 (i.e., 3 missing data points). Four subjects inadvertently neglected to complete one of the VAS daily records (days 14, 22, 28, 29). For all missing VAS data, the arithmetic mean specific to each individual woman’s pain record was employed. With regard to excess data, 56 subjects completed daily pain records for more than 30 days. Specifically, 26 subjects completed 1 additional daily record, 12 subjects completed 2 additional daily records, 16 subjects recorded an extra 3 days to 1 week, and 2 subjects recorded over one extra week (i.e., one recorded 8 and one recorded 10 additional days). Given that analyses were limited to a 30-day period, in cases where women had completed greater than 30 daily records the decision was made to retain the last 30 daily records and exclude daily records that dated back to more than 30 days from the exit interview.

With regard to missing data from the questionnaires, 10 cases had missing data, 8 of which omitted just a single item. The absent data appeared random in nature and the decision was made to employ the conservative procedure of group mean substitution for missing questionnaire data.

Although other procedures such as listwise and casewise deletion, as well as multiple imputation were considered in managing missing data, mean-substitution appeared justified given George and Mallery’s (2000) guidelines. These authors suggested that as long as no single variable evidences greater than 15% missing data, this technique has little influence on the outcome of the analyses. In the present study, missing data was in line with this recommendation.

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CHAPTER 4

RESULTS

Overview

Analyses were conducted on 100 participants. Prior to analysis, all socio-demographic, dependent and independent variables were examined for accuracy in data entry, missing values, and fit between assumptions of multivariate analyses and variable distributions.

The statistical method for identifying and excluding univariate outliers included locating cases with scores on a variable above three standard deviations from the mean, as well as examining graphical depictions (i.e., boxplots) of all variables, as suggested by Tabachnick and Fidell (2001). Mahalanobis distance procedure for identifying multivariate outliers was employed. Neither univariate nor multivariate outliers were present in the data. It is important to note that in an attempt to assess and control for outliers, the main analysis was also conducted utilizing only those in the sample who were within two standard deviations from the mean. However, no differences emerged in the results when including either two or three standard deviations. Thus, in order to improve power, and because we are particularly interested in those women with significant overestimation of pain at recall, we retained all subjects within three standard deviations of the mean.
The presentation of results will be conducted in the following order. Initially, we will review the significant associations between the demographic background variables and our predictor and predicted variables. We will then present the distribution of pain recall and describe the accuracy with which women recalled their pain. A review of the findings of our discriminant function analysis will then follow. Because passive coping did demonstrate a significant association with pain recall accuracy, we conducted further analyses to determine the specific subscales of passive coping that comprised the association; these analyses will be presented next. Finally, we will present an exploratory analysis intended to investigate whether any particular time of the month (i.e. initial, middle or final) was more influential in pain recall accuracy.

Covariation Between Background Variables and Pain Recall Accuracy

Analyses were conducted to determine whether there were significant associations between any of the background characteristics of our sample and pain recall accuracy, including whether the interviews had been conducted in person or on the phone. To compensate for the increased Type 1 error emanating from conducting multiple tests of significance (i.e. ANOVAs, correlations), only relationships at the .01 level of significance were considered. No background variables were significantly related to the dependent measure, pain recall accuracy, thus the analyses did not include covariates.
Description of Pain Recall Accuracy

With regard to the distribution of pain recall accuracy (i.e. ratio score), a large proportion of the sample was relatively accurate, with most of the inaccuracy skewed in the direction of high estimation (see Figure 1 for the distribution of pain recall accuracy). Noting that perfect accuracy would translate into a ratio score of 1.0, and that deviation from this standard in either direction (positive or negative) represents inaccuracy, the mean for this variable was 1.68, the median 1.27, and the standard deviation 1.20. Ratio scores ranged from .10 (low estimation) to 5.83 (high estimation). When the ratio score was evaluated as any deviation from 1.0, 25% of the sample had a ratio score below 1.0 and 75% of the sample had a ratio score of above 1.0. No single individual demonstrated perfect accuracy in recall.

Prediction of Pain Recall Accuracy

Table 2 presents the means and standard deviations of pain recall accuracy and all other measures. As previously mentioned, accuracy of pain recall was relatively good with a tendency toward high estimation. Actual pain ratings over the diary period were not very high and current pain at time of recall was also relatively low.

Table 3 presents the intercorrelations between all predictor variables to be entered into the discriminant function analysis. A Bonferroni-Holm correction was applied to control for Type 1 error, and yielded one significant correlation. Current pain recall was strongly and positively associated with actual pain during the 30-day diary period. There were no other significant associations.
Recall accuracy was dichotomized by a median-split into two groups; groups were subsequently labeled low estimation and high estimation. An independent samples t-test was conducted between the ratio score of the low estimation \((M = .92, SD = .29)\) and that of the high estimation \((M = 2.44, SD = 1.28)\) groups to ensure that the two were significantly different; this analysis was significant, \(t(98) = 50.06, p < .001\). Several tests of significance, including chi square and ANOVA, were conducted to ensure that the two groups did not differ with respect to any demographic background variables; none of these tests reached statistical significance.

A direct discriminant function analysis was performed using five psychosocial variables (current pain at recall, psychological wellbeing, active coping, passive coping, fertility distress) as predictors of membership in one of two pain recall accuracy groups (low estimation and high estimation). Because of a high degree of association between current pain and actual pain \((r = .66, p < .001)\), a decision was made to limit entry into the equation to one of these two variables. Because current pain at the time of recall has consistently been found to be related to recall (Bolton, 1999; Bryant, 1993; Holroyd, France, Nash, & Hursey, 1993; Lefebvre & Keefe, 2002), we chose to exclude actual pain as a predictor variable. Additionally, no relationship emerged between actual pain and our predictor variables of interest.

SPSS DISCRIMINANT was utilized for the following analyses, and all variables entered into the equation at once. The assumptions inherent to discriminant function analysis, including multivariate normality, homogeneity of variance-covariance matrices, multicollinearity, and exclusion of univariate and multivariate outliers were evaluated and determined to be robust. One discriminant function was calculated, with a \(\chi^2 (5, N = \ldots\)
The function maximally separated the low estimation from the high estimation group, and accounted for 100% of the between-group variability. The loading matrix of correlations between predictors the discriminant function, as seen in Table 4, suggested that the best predictors for distinguishing between the two groups were passive coping and current pain at time of recall. Although no consensus currently exists regarding the minimum correlation in the loading matrix required for interpretability, convention dictates that those in excess of .33 may be considered eligible while those falling below this standard are not (Tabachnick & Fidell, 2001). Thus, all correlations in the loading matrix above .33 were considered in the present study. The high estimation group endorsed utilizing significantly more passive coping strategies than the low estimation group and recorded significantly lower levels of current pain at time of recall than the low estimation group (see Table 5). With regard to classification of the total sample of 100 women, 64 (64%) were classified correctly, compared to 50 (50%) who would be correctly classified by chance alone (see Table 6).

Passive Coping as a Predictor of Pain Recall Accuracy

To further explore the predictive power of passive coping as a function of pain recall accuracy, a breakdown of passive coping into its subscales, catastrophizing and praying/hoping, was conducted. Given that catastrophizing and praying/hoping were moderately correlated at a level acceptable for MANOVA ($r = .44, p < .05$), a decision was made to conduct a one-way between-groups multivariate analysis of variance to investigate differences in utilization of passive coping strategies between the high estimation and low estimation groups. As required, a Bonferroni correction was applied...
(.05/2 = .025) and only predictor variables with a significance level of .025 or lower were considered. Table 7 presents the means and standard deviations of the high estimation and low estimation groups on catastrophizing and praying/hoping scores. Preliminary assumption testing was conducted to assess normality, linearity, univariate and multivariate outliers, homogeneity of variance-covariance matrices and multicollinearity. One violation was noted; homogeneity of variance in the two groups was not equal. However, this issue was irrelevant given that the omnibus test for MANOVA was not significant (see Table 8). Considering that the alphas for the omnibus test for MANOVA and for the univariate test on praying/hoping were .06 and .03, respectively, it is possible that a larger sample (increased power) would have yielded significant results.

Exploratory Analyses

To determine whether pain recorded during certain times of the month was more salient and influential while recalling pain, an exploratory analysis was conducted. The 30 days of the pain record were broken up into three distinct time periods, the first ten days of the month, second ten days of the month and third ten days of the month (i.e. initial, middle and final) and the mean of each was computed, in addition to the intercorrelations between pain recall and actual pain at each of the 3 time periods (see Table 9). To investigate the unique contribution of actual pain at the 3 time periods in predicting pain recall, a single standard multiple regression analysis was conducted with actual pain at the 3 time periods entered into the equation simultaneously. In predicting pain recall using the entire sample, the regression equation was significant $F (3, 96) = 57.42, p < .01$, and accounted for 64% of the variance (see Table 10). Only the first and
second 10-day periods appeared to be unique predictors of pain recall; however, considering the high correlations between pain recall and all of the time periods (see Table 9), it appears that the last 10 days were only slightly less predictive of pain recall and not particularly worthy of note.

To further explore the determinants of coping, we ran two sets of moderated multiple regressions, one set for passive coping and one set for active coping. In each set, there were 5 moderated multiple regressions in which we first entered the pain recall accuracy variable, followed by one of the 5 independent variables (fertility distress, psychological wellbeing, current pain at recall, actual pain and active or passive coping), and finally, the interaction of pain recall accuracy and the independent variables in question. Each analysis was run separately without correcting for Type 1 error because they were exploratory in nature and for ease of interpretation. Results of these analyses did not yield any clinically significant findings.

As to passive coping, pain recall accuracy accounted for only 2% of the variance. Passive coping was not predicted significantly by active coping or current pain at recall, nor by the interactions of these variables with pain recall accuracy. Passive coping was predicted by actual pain ($R^2 = 0.09$), fertility distress ($R^2 = 0.09$), and psychological wellbeing ($R^2 = 0.09$), however, none of these three variables accounted for more than 9% of the variance. Interactions between these variables and pain recall accuracy only produced $R^2$ changes ranging from 0.01 to 0.03; although these interactions are statistically significant, they account for a negligible proportion of the variance and are not worthy of interpretation (see Table 11 to Table 13).
As to active coping, pain recall accuracy accounted for only 1% of the variance. Active coping was not predicted by current pain at recall, fertility distress, passive coping, or psychological wellbeing, nor by the interactions of these variables with pain recall accuracy. Active coping was predicted solely by actual pain ($R^2 = 0.06$), however, this variable accounted for only 6% of the variance. Again, the interaction between active coping and actual pain was statistically significant, but accounted for a negligible proportion of the variance (see Table 14).
CHAPTER 5

DISCUSSION

Overview of Findings

Women in this study were generally quite accurate in their recall of pain experienced over the previous 30 days, with inaccuracy most often skewed in the direction of overestimation. Overall, the findings from the discriminant function analysis supported only one of our hypotheses. The first hypothesis, that decreased psychological wellbeing would be a significant predictor of high pain estimation (i.e. overestimation), was not confirmed. Likewise, the second and third hypotheses were not supported; neither fertility distress nor active coping were significant predictors of recall accuracy. Only passive coping emerged as a predictor in the expected direction (our fourth hypothesis); women who endorsed a more passive coping style tended to recall higher levels of pain than they had actually experienced (overestimation). Our fifth hypothesis, that current pain at time of recall would be positively associated with overestimation of pain, was more than disconfirmed – findings were significant in the direction contrary to that expected; women who endorsed higher current pain at time of recall tended to recall lower pain levels over the 30 days prior. Our exploratory analyses, aimed at identifying whether any particular time of the month (initial, middle or final 10 days) was more predictive of pain recall accuracy, yielded non-significant findings. No pain-recording period was any more predictive than any other. Similarly, our exploratory analyses
utilizing moderated multiple regression suggested that none of our variables were strongly predictive of passive or active coping, and that pain recall accuracy was not a strong moderator for any variable. Interpretation of the aforementioned findings will be undertaken below.

Pain Recall Accuracy

Given that the majority of participants were relatively accurate in their recall of pain, one could surmise that physicians can rest assured that the report of pain they are obtaining from the *majority* of patients is a relatively accurate representation of the actual pain experienced. In the subset of women where inaccuracy did occur, it was most often skewed in the direction of overestimation, a finding corroborating those of prior studies (Erskine et al., 1990; Feine et al., 1998; Jamison et al., 1989; Kent, 1985; Linton, 1991; Stone et al., 2004). The overall recall accuracy found in this study echoes findings of a number of previous investigations of various pain conditions, including: labor pain, head pain, chronic back and myofacial pain, experimentally induced pain (i.e., via tourniquet) and acute pain due to clinical procedures such as dentistry (Beese & Morley, 1993; Erskine et al., 1990; Hunter, Philips & Rachman, 1979; Niven & Murphy-Black, 2000; Salovey et al., 1993; Rofe & Algom, 1985; Von Korff, 2001). The findings in this sample suggest that the current clinical practice of asking women with endometriosis to retrospectively report their pain might be more reliable than some health professionals may think.

Alternately, it may be that the monitoring of pain itself promoted more accuracy in our sample than might be found in a real-world clinical setting. The potential for
reactivity during self-monitoring of a number of phenomena, including frequency of face touching (Nelson, Boykin, & Hayes, 1982; Hayes & Nelson, 1983), pain-related variables (Aaron, Turner, Mancl, Brister, & Sawchuk, 2005), verbal nonfluencies (Bornstein, 1978; Mace & Kratochwill, 1985), positive and negative classroom verbalizations/behaviors (Baskett, 2001; Nelson, Hay, & Hay, 1977) and verbal and motor behavior (Willis & Nelson, 1982) is well recognized. In fact, Nelson and Hayes (1981) provided a thorough review of several leading theoretical explanations for reactivity in self-monitoring. Reactivity has been defined as a change in the phenomenon of interest directly attributable to assessment (i.e., self-monitoring/self-recording) of the construct (Aaron, Turner, Mancl, Brister & Sawchuk, 2005), or in other words, an alteration in response frequency (Nelson & Hayes, 1981). Interestingly, most studies that have evaluated reactivity due to pain diary methodology have consistently failed to find that changes over time in the pain levels recorded are attributable to self-monitoring, and have thus concluded that reactivity is not a significant factor in the self-monitoring of pain (Aaron et al., 2005; Cruise, Broderick, Porter, Kaell, & Stone, 1996; Kerns, Finn, Haythornthwaite, 1988; Peters, Sorbi, Kruise, Kerssens, Verhaak, & Bensing 2000, Stone, Broderick, Schwartz, Shiffman, & Litcher-Kelly, 2003). However, the focus in these studies was the reactivity of pain recording, rather than pain recall, as a function of monitoring.

A legitimate concern is that self-monitoring and its attendant awareness of pain may result in inflated accuracy in comparison to reports in clinical settings, where recalled-pain with no previous monitoring is a standard practice. One study provides evidence that this may not be the case. Salovey, Smith, Turk, Jobe, and Willis (1993)
assigned 107 chronic pain patients to one of 4 experimental conditions: 1) daily monitoring of pain intensity, 2) daily monitoring of pain related behavior, 3) daily monitoring of pain intensity and pain behavior, or 4) a no-monitoring control group. At the end of the 30-day recording period, all subjects were asked to recall their pain and pain-related behaviors over the past month. Two findings are of relevance here: 1) in the two groups in which pain intensity was both recorded and recalled, women were highly accurate in their recall and 2) there were no significant differences in recalled pain levels across the 4 groups. Thus, employing a pain diary did not influence subsequent recollection of pain, as the control group did not differ in their recalled pain levels from the accurate self-monitoring groups. This finding should be of some comfort to those investigators and clinicians relying on self-monitoring in pain assessment.

Despite our results supporting the accuracy of pain recall, there is little question that most research indicates a natural fallibility of memory in retrospective recall, especially the more remote the moment of recall is from the actual experience of events. Kent (1985) has addressed the issue of retrospective recall fallibility specifically within the context of pain, stating that although pain recall has been cited as accurate when the recalled time span is short (i.e., 5 days), it is a flawed assumption that patients can access and accurately recall frequency and intensity of past pain over long time spans (i.e., several months). The findings of our study provide evidence to question Kent's diminutive time span, as an entire month had transpired prior to recall. However, the idea that shorter duration between pain-experience and recall improves accuracy is aligned with the model recently proposed by Gedney and Logan (2004). This model offers a theoretical explanation for the inconsistency present in the pain recall-accuracy literature.
by suggesting that when recall period is of short duration (defined as a month or less), actual pain is most predictive of recall, whereas other factors mediate this relationship as recall period is extended. This model was in part based on the findings of Hunter, Phillips, and Rachman (1979) and Singer, Kowalska, and Thode (2001), who determined that acute pain was accurately recalled after a period of weeks, and that of Everts, Karlson, Wahrborg, Abdon, Herlitz and Hedner (1999) who suggested that pain recollection is exaggerated when recall-period extends into months. Additional support for the model includes the findings of McGorry et al. (1999), Porzelius (1995), and Gedney, Logan and Baron (2003), who reported short-term recall of pain or pain relief as accurate, and long-term recall as more problematic and inconsistent. The present study’s findings remain consistent with the model insofar as the recall period was limited to one month and actual pain was highly predictive of recall.

In an attempt to combat memory distortion due to delay in recall, in-the-moment assessment is utilized with increasing frequency in medical and research settings specifically to address health conditions, a methodology referred to as real-time reporting or ecological momentary assessment (EMA; Shiffman, Hufford, Hickcox, & Paty, 1997; Shiffman, Paty, Gnys, Kassel, Hickcox, 1996; Shiffman & Stone, 1997; Stone, Schwartz, Neale, Shiffman, Marco, Hickcox et al., 1998). Such research is now employing portable electronic or palm pilot devices to reduce recall errors. The reality of medical settings, however, makes it unlikely that momentary assessment will be widely adopted clinically in the assessment of pain.

Although the current study did not address the issue specifically, it is possible that women who engage in high pain estimation are most likely to vociferously complain
of pain to physicians. Our study conducted a rudimentary assessment along these lines by asking women to endorse type of pain-management treatment undergone as a result of endometriosis (i.e., hormonal, surgical, pain, and/or psychological), and conducting analyses comparing underestimators and overestimators on type of treatment. We did not find significant differences, but our assessment of health care utilization was not detailed enough to seriously address this specific question. Chronic and persistent complaint of pain has been highlighted as the strongest indication for surgical treatment (Fliegner and Umstad, 1991; Koninckx, Timmermans, Meuleman, Penninckx, 1996; Treloar, Do, O’Connor, O’Connor, Yeo, & Martin, 1999). Thus, if it is the case that overestimators present with pain to their physicians with greater frequency than underestimators or accurate recallers, health professionals may be choosing invasive treatment based on inaccurate pain representation in a certain proportion of women.

Although the results of the current study do not support the recommendation that all women with endometriosis monitor their pain in diary form, it appears both feasible and clinically prudent for the physician to mandate a standardized pain-recording period in all patients under consideration for invasive treatment. Such recommendation aligns itself well with current literature addressing problems with autobiographical memory in medical settings. Stone, Broderick, Shiffman and Schwartz (2004) elucidated the pitfalls of pain assessment based on office visit to office visit, suggesting that such common medical practices as asking patients to make judgments describing relief, no change, or a worsening of pain from one period to the next (often with a considerable time-interval between visits) is a challenging task and may lead to biased data. Dell (2004) recently bemoaned retrospective recall of premenstrual symptoms as “notoriously unreliable,”
advocating daily symptom-recording to allow for accurate diagnosis and improved patient involvement in self-management of the condition.

Predictors of Pain Recall Accuracy

*Psychological Wellbeing and Fertility Distress*

Our finding that psychosocial distress level was not predictive of recall accuracy is not the first of its kind. Linton (1991) found that depression was not correlated with pain recall accuracy. Tasmuth, Estlanderb and Kalso (1996) found that depression and anxiety were unrelated to recalled pain and Zonneveld, McGrath, Reid, and Sorbi (1997) found anxiety unrelated to accuracy of recalled pain intensity in a child population. The finding that both physiological and psychological distress levels were unrelated to recall accuracy in a sample of low back pain patients was reported by McGorry, Webster, Snook, and Hsiang (1999). Haas, Nyiendo and Aickin (2002) compared actual relief in pain (i.e., the difference between patient’s baseline and current pain levels) to recalled relief in pain (which necessitates some component of pain-memory), reporting that stress and wellbeing were not predictive in pain relief recall. However, other investigations have suggested a relationship *does* exist between distress and recall accuracy, with greater distress linked to overestimation of pain at recall (Bryant, 1993; Eli, Baht, Kozlovsky & Simon, 2000; Everts, Karlson, Wahrborg, Abdon, Herlitz & Hedner, 1999; Gedney & Logan, 2004; Hunter, Philips, & Rachman, 1979; Jamison et al., 1989; Kent, 1985; Lander, Hodgins, & Fowler-Kerry, 1992). To complicate matters further, one study demonstrated no relationship between accuracy and distress at 1-week recall, but results supported a relationship at 18 month-recall (Gedney, Logan & Baron, 2003). One
potential explanation for the contradictory results relates directly to the aforementioned model by Gedney and Logan (2004). The authors specifically highlighted affective state (depression and anxiety) as particularly predictive of long-term recall of pain experienced. Thus, it is possible that because our study employed a relatively short recall-duration (i.e., one month), distress was not a significant predictor of accuracy. Had the time span between pain recording and recall been extended, a relationship may have emerged.

An additional factor to consider includes the wide variety of distress measures (some potentially problematic) utilized across studies. Ballweg (1997) admonished the use of measures designed for “physically healthy people” to assess those with medical problems, especially when such standard psychological scales incorporate symptoms of endometriosis. Several of the studies evaluating the relationship between pain recall accuracy and distress level employed the Beck Depression Inventory (BDI). Although the BDI is frequently utilized within health settings, its heavy emphasis on somatic aspects of depression has led some researchers to question whether false-positives among patients with physical problems (especially pain) might occur (Williams & Richardson, 1993; McDowell & Newell, 1996). Sensitive to this measurement problem, the current study enlisted the Rand Mental Health Inventory, which is a measure of psychological symptomatology designed for use with medical populations. Interestingly, the other study in the literature employing a distress measure designed and normed with medical populations (Haas et al., 2002) also found distress to be unrelated to pain recall accuracy. Alternatively, it is possible that our recruitment methods did not tap into the most distressed or painful cases of endometriosis. The majority of participants in our study

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were recruited from the community rather than medical settings, so it stands to reason that the cases most troubled by their illness might be more prevalent within the latter setting.

As was the case with general psychological distress, fertility-specific distress was not sufficiently predictive of pain recall accuracy, although the trend was in the expected direction and the correlation between fertility distress and the structure matrix was relatively high. Given this trend toward significance, as well as the fact that our sample size was relatively small, a greater number of subjects may have yielded a significant relationship, and future studies in this area are encouraged.

Coping

Active Coping was not a significant factor in predicting pain recall accuracy, a finding that corroborates prior research into memory for and adjustment to chronic pain. Zonneveld, McGrath, Reid, and Sorbi (1997) found that pain coping strategies in general were not related to accuracy of recalled pain intensity in a child population. Evers, Kraaimaat, Geenen, and Bijlsma (1998) found that increased passive coping was associated with increasingly negative functional status, while active coping was not predictive of functional status whatsoever. The finding in the present study may be due, in part, to the fact that the strategies emphasized by the active coping measure may be unfamiliar to women with chronic pain. A number of women in the current study denied ever having used strategies such as dissociation from the pain experience (e.g., the “reinterpreting the pain sensations” subscale of the CSQ, one of the five subscales of active coping). Some participants even went so far as to suggest in person to the principle investigator that such statements seemed strange, silly, and ineffective when
pain control is the intention. When evaluating frequency of coping strategy-employment in our study, active coping was limited. For example, the mean of active coping for the entire sample in the current study was 68, even though the ceiling for the subscale was 180. In those women who did employ active strategies, such skills appear to be indiscriminant with regard to recall accuracy.

Passive coping, however, did reach significance in predicting pain recall accuracy. Those who exhibited greater passive coping style also tended to recall pain as greater than what was recorded on a daily basis over the prior month. Importantly, passive coping demonstrated the greatest correlation of all predictors to the structure matrix. Passive coping clearly appears to tap into a mechanism strongly associated with overestimation of pain. From a face-validity standpoint, items keyed in the passive direction appear to have a tendency to maximize pain. Such items include, “When I feel pain, it is terrible and I feel it is never going to get better” and “When I feel pain it is awful and I feel that it overwhelms me.” Such maximization of pain may carry over into a general approach toward pain that, in effect, colors one’s memories of past pain as well as impacts one’s approach to pain in the moment. Given that passive coping is currently viewed as dispositional in nature (Brown & Nacassio, 1987; Walker, Smith, Garber & Claar, 2005), a pervasive tendency to amplify pain may also have trait-like qualities.

The passive scale on the CSQ is comprised of items from two distinct subscales, catastrophizing and praying/hoping. It appears that although neither subscale reached clinical significance when examined individually, there was a trend indicating that both subscales were associated with pain overestimation. Interestingly enough, previous studies have not evaluated the influence of passive coping on the accuracy of pain recall,
but instead, have simply evaluated the effects of catastrophization strategies in isolation. It appears that passive coping as a combined construct rather than catastrophization alone might prove a more worthwhile predictor of accuracy.

The importance of passive coping as it relates to high pain estimation and poor pain adjustment is clear, and voluminous support of this relationship currently exists. Smith, Lumley and Longo (2002) found that passive coping strategies were positively related to negative affect, and associated with greater pain, impairment, and depression. The finding that passive coping was positively associated with depression and physical disability, and negatively associated with physical functioning was observed by Beugnot (2002). In a chronic back pain population, Hellstrom and Anderberg (2003) found that women with a high frequency of pain reported utilizing passive coping more often and endorsed more catastrophizing thoughts. Mercado (2004) highlighted the maladaptive nature of passive coping, and identified such coping as a risk factor for development of disabling pain, as well as a poor prognostic factor in recovery from low back pain due to motor-accident. Bishop and Warr (2003) found that passive coping explained unique variance in self-reported disability status in a breast-cancer population; specifically, passive coping was associated with greater disability. The authors suggested that passive coping and catastrophizing might contribute to negative pain outcome and may be important targets for psychological intervention.

Clearly, passive coping is maladaptive in nature, and direct attempts to decrease this coping style are recommended in the patient with endometriosis. In fact, given that some studies have found adaptive pain outcome to demonstrate a greater association with reduced catastrophization/passive coping rather than increased active/adaptive coping,
some researchers have exclusively emphasized targeting catastrophization in pain treatment (Bishop & Warr, 2003). These findings might also help explain why passive rather than active coping was a significant predictor of pain recall accuracy in the current study, (if one considers accuracy in recall to be indicative of pain adaptation, a link that has yet to be demonstrated in the literature).

**Current Pain at Time of Recall**

Current pain at time of recall was significant in predicting underestimation of recorded pain, a finding that ran counter to our hypothesis and to much of the existing literature which finds high current pain to be strongly associated with overestimation (Bolton, 1999; Bryant, 1993; Eich, Reeves, Jaeger, & Graff-Radford, 1985; Holroyd, France, Nash and Hursey, 1993; Lefebvre & Keefe, 2002; Rocha, 2004; Salovey, Smith, Turk, Jobe and Willis, 1993; Smith & Safer, 1993). A state-dependent theory has been offered to explain the more commonly found positive association between current pain at time of recall and recalled pain. If current pain at time of recall is high, then it colors memory for past pain and elevates the recall closer to the current experience. Interestingly, one study found that high current pain at recall was related to a general inaccuracy in pain-intensity recall, not necessarily an overestimation (Lefebvre & Keefe, 2002). Investigators had asked participants to chart pain for 30 days, and to subsequently recall pain for each of the 30 days to the best of their ability. They found that as pain at time of recall increased, a significant decrease in accuracy of pain-intensity recall was observed. The authors posited a cognitive explanation, suggesting that pain at time of recall interferes with the retrieval of pain experiences in memory due to the high level of cognitive resources that momentary pain demands.
Given that, in our study, the women reporting high levels of pain at recall were inaccurate primarily in one direction (underestimation), Lefebvre & Keefe's explanation of retrieval interference does not quite explain what happened with our sample. An alternative interpretation for our finding also borrows heavily from the cognitive literature. The theory that an individual's quantitative estimate or judgment can be influenced by an arbitrary starting value, referred to as an anchoring effect (Jacowitz & Kahneman, 1995), may shed some light on our results. In fact, such an argument was put forth by Salovey, Sieber, Jobe, and Willis (1994), as authors suggested that participants may utilize current pain as a springboard or anchor for evaluating past pain. Given that participants were asked to rate their current level of pain prior to making retrospective ratings of pain, current level of pain might have operated as an anchor by which previous pain was judged. But why did that anchor result in underestimation? It could be that the saliency of current high pain may have made past pain seem low in comparison (i.e., current pain overshadowed pain in the past), thus yielding underestimation of pain in those with high levels of current-pain. However, given that this finding runs counterintuitive to the majority of research on this subject, a perplexing picture of the potential mediators of pain recall accuracy remains.

Recording Stages as Predictive of Pain Recall

The findings of our exploratory analysis indicate that no particular time of the month or recording stage (i.e., initial, middle, and final 10 days of the pain diary) was more salient at pain recall than any other. Not surprisingly, the overall regression analysis examining the three times of the month was significantly predictive of pain...
recall (accounting for 64% of the variance), again resounding the fact that recall in this study was based in large part on the actual pain recorded. It is interesting to note that the final 10 days of the month were least predictive of pain recall. This seems a counterintuitive finding when one considers the large body of literature substantiating the recency-effect as it applies to short-term recall, and the emerging evidence for a long-term recency-effect (Da costa Pinto & Baddeley, 1991; Davelaar, Goshen-Gottstein, Ashkenazi, Haarmann & Usher, 2005). Clearly this was not the case in the present study.

Again, this latter finding lends additional support to the idea that memory for pain is a highly complicated issue and presents with numerous cognitive influences, a view that has emerged forcefully in the current literature. Turk and Okifuji (2002) argued that current conceptualization of pain patients as a homogenous group has impeded advancements in pain-treatment. Other researchers have proffered the ensemble theory of pain, a concept acknowledging the individual’s genetic, molecular, physiological, emotional and sociocultural factors at play in pain perception/experience. Many of these researchers have suggested treatment-matching to patient characteristics, and advocate an individualized or more prescriptive therapeutic approach to pain control (Dell, 2004; Godown & Stephenson, 1998/1999; Turk & Okifuji, 2002).

Limitations

When interpreting our findings, several caveats are in order. In the present study, diagnosis of endometriosis was self-reported and remained unverified by medical records. Although this is clearly an issue worthy of comment, there is no reason to believe that women would perfidiously present as suffering from endometriosis.
Participants were requested to provide the date of diagnosis, and it was highlighted within the informed consent, as well as verbally in the initial interview, that a medical diagnosis of endometriosis was required. Additionally, most women who do not suffer from endometriosis remain generally unaware of the disease and its symptoms. An additional limitation is the potential for pain-diary review. It is possible that although 1) women were asked to place completed diary pages into an envelope after each entry and seal the contents within, and 2) they did not know they would be asked to recall pain, participants still may have reviewed their recordings at some point over the 30 days, thereby influencing recall toward the direction of greater accuracy. This is, however, unlikely in the current study as it would not explain why we evidenced a strong association between pain at time of recall and underestimation, unless one posits the improbable theory that those in most pain at time of recall were the least likely to review their monthly ratings. In any case, a solution to this dilemma for future studies may be to employ an electronic pain record (i.e., patients submit their daily pain records online via email or an online website) or utilize current technology for pain recording such as palm-held computers, therein bypassing the possibility that review of data will occur.

A more diffuse caveat currently impacting all pain research is the lack of an ideal pain assessment modality. Although VAS pain diaries assist researchers and the medical community alike in assessing pain closer to its occurrence and are considered the most valid and reliable of the measures available, VAS is hardly considered ideal. There is simply no measure in existence that is a purely objective measure of pain; as Whelan (2003) has pointed out in an article specifically targeting pain assessment in the endometriosis population, the primary limitation to such measurement is the subjectivity

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of self-evaluation. A final limitation in the current study centers on the statistical power afforded by our sample size. Given that the study was conducted on a clinical population, access to potential participants was limited and sample size was insufficient for the discriminant function analysis. It would be interesting to witness the performance of fertility distress, psychological wellbeing and active coping in future studies securing larger sample sizes.

**Future Research**

Future research may be wise to explore whether accurate pain recall or inaccuracy in the direction of pain minimization is indicative of better pain adjustment, a potentially philosophical debate. Questions such as, “Is pain minimization adaptive, or a form of denial?” might be researched. Along this vein, it may prove fruitful to determine the degree to which pain recall accuracy is linked to other objective indices of adaptive pain adjustment, such as decreased disability, increased functional level, adequate pain control and increased behavioral activation. As touched upon previously, another interesting and potentially useful avenue of inquiry would be to investigate the extent to which pain recall accuracy is related to health care utilization and treatment seeking. Many women with endometriosis have long histories of multiple treatments, some of which are quite invasive and carry significant risks. Attempts to determine whether patterns of health care utilization are related to pain perception could lead to pain management interventions that could reduce the unnecessarily invasive and ineffectual surgeries that burden both individual women and the health care system as a whole.
Pain management outcome studies focusing on the efficacy of interventions to decrease passive coping might also prove clinically useful. Considering the now acknowledged multi-dimensionality of the pain experience in general and the low correspondence between physical pathology and pain in endometriosis, it seems fruitful to turn our sights on psychological strategies to help women cope with this condition while providing medical treatments as appropriate.

Finally, from a cognitive perspective, future research may want to examine the strategies women are utilizing to remember their pain. Are estimates of pain based on heuristic strategies, such as a subject recalling that only half of the days in her diary record were above the 50% mark, and thus deciding that the recall for the 30 days could not logically result in estimation above this midway point? Or are other strategies at play? Judging from our exploratory analysis of pain recall and time of the month, the heuristic may be substantially more complicated than one would predict with recency or primacy effects. The extent to which women could access these heuristics is an empirical question at this point, but investigations into these questions could inform important questions about the cognitive processing of pain and its mediators.

Implications

The findings of this study have direct application to the medical community and to women who suffer from this disease. Outside of the select subset of women who were inaccurate in the direction of pain overestimation, the majority was accurate. Thus, a reconceptualization of patient self-report of pain within the medical community might be warranted. A primary complaint of women with endometriosis is that health providers
discount or tend to downplay their pain experience. As a result, many feel their communication with physicians regarding pain is strained and ultimately, unsatisfactory. Cox, Henderson, Anderson, Cagliarini, and Ski (2003) documented this well. Their study of endometriosis patients in focus groups found that participants 1) lamented health professionals who were dismissive of or trivialized their symptoms, 2) expressed low self-esteem due to a general disbelief within the medical-community regarding their symptoms, and 3) engaged in “doctor-trials,” consisting of year(s)-long search for attentive and empathetic doctors who “listened and believed” their pain. Many participants reported experiencing emotional damage due to interactions with a number of doctors within the medical community who exhibited a lack of endometriosis-specific knowledge and a poor attitude. Kennedy (1991) corroborated this, suggesting that one of the major stumbling blocks to diagnosis and effective disease management relates to the attitude of the doctor, in part dictated by medical education, which sometimes cultivates misconceptions about the disease and its epidemiology. It is important to note that not all women have experienced such negative interactions with the medical community, and Montague and Wood (1997) found that negative attitude and lack of empathy were exhibited in only a minority of physicians. Overall, our results suggest that physicians may benefit from giving greater credence to women’s reports of pain, given that the pain recalled seems, on the whole, to be a reliable account. For the endometriosis patient who feels that her physician fails to validate her pain, or that interfacing with the medical community has been unsuccessful, a self-initiated pain diary might be a useful strategy to pursue more effective interaction, as she can present “documentation” of the pain experienced. Hopefully, the current study will assist in more effective communication
between the patient and the medical community, in that physicians can allow themselves
to have more faith in their patients’ report of pain.

An additional clinical application concerns those women who are employing non-
adaptive coping strategies. Physicians can quickly screen for patients who frequently
engage in passive coping via use of the praying/hoping and catastrophization subscales of
the CSQ. Physicians can request such patients to keep a daily diary of pain to maximize
the likelihood that daily pain report (rather than recalled pain) dictates treatment. When
identified, such patients may benefit greatly from referral to cognitive behavioral
treatment centering on their attaining a more realistic appraisal of pain. Such a referral
may assist in circumventing more drastic medical procedures if women can attain
improved pain control. Additionally, physicians may wish to institute a mandatory pain-
recording phase for all women who are currently under consideration for extensive and
radical surgery, simply to ensure that treatment is based on the most valid pain report.

Chronic pain is a multi-determined and multi-dimensional construct. Great
strides have occurred within recent years regarding the general pain paradigm, as we have
seen a shift away from the dualistic model of pain to the current biopsychosocial theories
that have revolutionized the field. The complexities of the human pain experience
require that researchers and clinicians continue to consider a variety of mediating
variables rather than concentrating on a single dimension of pain. Given the
multidisciplinary pain approach becoming more widely adopted, as well as the U.S.
Congress’s appointment of the years 2001-2011 as the “Decade of Pain Control and
Research” (Turk & Okifuji, 2002), the future holds promise for significant progress in the
research and treatment of pain.
Table 1

Characteristics of Participants (N = 100)

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>M</th>
<th>SD</th>
<th>n (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age at time of survey (years)</td>
<td>29.36</td>
<td>5.94</td>
<td></td>
</tr>
<tr>
<td>Racial background</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Caucasian</td>
<td>80</td>
<td></td>
<td></td>
</tr>
<tr>
<td>African American</td>
<td>8</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hispanic</td>
<td>8</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Asian</td>
<td>2</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Other</td>
<td>2</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Highest education level completed</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Less than high school</td>
<td>1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>High school</td>
<td>6</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Some college</td>
<td>41</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Undergraduate school</td>
<td>35</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Graduate school</td>
<td>17</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Marital status</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Single</td>
<td>38</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Married</td>
<td>53</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Divorced</td>
<td>8</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Separated</td>
<td>1</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

(Table 1 continues)
(Table 1 continued)

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>M</th>
<th>SD</th>
<th>n (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of children</td>
<td>.53</td>
<td>1.04</td>
<td></td>
</tr>
<tr>
<td>Duration since first diagnosis (in months)</td>
<td>57.57</td>
<td>55.85</td>
<td></td>
</tr>
<tr>
<td>Duration of pelvic pain (in months)</td>
<td>133.75</td>
<td>88.64</td>
<td></td>
</tr>
<tr>
<td>Number of Ob/Gyn visits per year</td>
<td>4.26</td>
<td>4.27</td>
<td></td>
</tr>
</tbody>
</table>

Endorsed utilizing the following endometriosis treatments*:

- Hormonal: 72
- Prescription pain medication: 87
- Surgical procedures: 81
- Fertility procedures: 14
- Psychological treatment for fertility and/or pain: 15

Referral source

- Community flyer: 27
- College campus flyer: 16
- Word of mouth: 7
- Ob/Gyn Clinic: 5
- Psychology 101 subject pool: 3
- Internet site: 42

Note. * Participants could endorse more than one treatment.
Table 2

Means and Standard Deviations of Pain Recall Accuracy and all Dependent Measures

<table>
<thead>
<tr>
<th>Variable</th>
<th>M</th>
<th>SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>(Range of Measure)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pain recall accuracy</td>
<td>1.68</td>
<td>1.20</td>
</tr>
<tr>
<td>(.01 - 100)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Actual pain</td>
<td>28.26</td>
<td>21.01</td>
</tr>
<tr>
<td>(0 - 100)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Current pain</td>
<td>21.25</td>
<td>26.18</td>
</tr>
<tr>
<td>(0 - 100)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fertility Distress</td>
<td>22.38</td>
<td>9.76</td>
</tr>
<tr>
<td>(9 - 45)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Psychological Wellbeing</td>
<td>76.19</td>
<td>14.76</td>
</tr>
<tr>
<td>(18 - 108)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Passive Coping</td>
<td>28.83</td>
<td>14.65</td>
</tr>
<tr>
<td>(0 - 72)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Active Coping</td>
<td>68.48</td>
<td>27.28</td>
</tr>
<tr>
<td>(0 - 180)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
# Table 3

### Intercorrelations for all Measures

<table>
<thead>
<tr>
<th>Measure</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Actual Pain Over 30 days</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2. Current Pain at Recall</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3. Fertility Distress</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4. Psychological Wellbeing</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5. Passive Coping</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6. Active Coping</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Note. * $p < .01$. 

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Table 4

Correlation of Predictor Variables With Discriminant Function (Function Structure Matrix) and Standard Discriminant Function Coefficients

<table>
<thead>
<tr>
<th>Predictor variable</th>
<th>Correlation with discriminant function 1</th>
<th>Standardized discriminant function coefficients</th>
</tr>
</thead>
<tbody>
<tr>
<td>Passive coping</td>
<td>.599*</td>
<td>.77</td>
</tr>
<tr>
<td>Current Pain at recall</td>
<td>-.564*</td>
<td>-.73</td>
</tr>
<tr>
<td>Fertility distress</td>
<td>.289</td>
<td>.26</td>
</tr>
<tr>
<td>Psychological wellbeing</td>
<td>.127</td>
<td>.42</td>
</tr>
<tr>
<td>Active coping</td>
<td>-.041</td>
<td>-.08</td>
</tr>
</tbody>
</table>

Note. * Correlations in excess of .33 were considered eligible for interpretation.
Table 5

Means and Standard Deviations of Variables as a Function of Pain Accuracy at Recall

<table>
<thead>
<tr>
<th>Predictor variable</th>
<th>High Estimation</th>
<th>Low Estimation</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$M$</td>
<td>$SD$</td>
</tr>
<tr>
<td>Passive coping</td>
<td>32.22* 15.10</td>
<td>25.44 13.49</td>
</tr>
<tr>
<td>Current pain at recall</td>
<td>15.52* 24.43</td>
<td>26.98 26.86</td>
</tr>
<tr>
<td>Fertility distress</td>
<td>23.49 10.08</td>
<td>21.26 9.40</td>
</tr>
<tr>
<td>Psychological wellbeing</td>
<td>76.93 15.14</td>
<td>75.45 14.48</td>
</tr>
<tr>
<td>Active coping</td>
<td>68.04 30.32</td>
<td>68.92 24.17</td>
</tr>
</tbody>
</table>

Note. Means of the high estimation group with an * differ significantly at $p < .05$ from means of the low estimation group.
Table 6

Classification Analysis for Pain Recall Accuracy

<table>
<thead>
<tr>
<th>Actual group membership</th>
<th>Predicted group membership</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>High Estimation</td>
<td>Low Estimation</td>
</tr>
<tr>
<td></td>
<td>$n$</td>
<td>$n$</td>
<td>%</td>
</tr>
<tr>
<td>High Estimation</td>
<td>50</td>
<td>31</td>
<td>62.0</td>
</tr>
<tr>
<td>Low Estimation</td>
<td>50</td>
<td>17</td>
<td>34.0</td>
</tr>
</tbody>
</table>

Note. Overall percentage of correctly classified cases = 64.0%.
Table 7

Mean Scores and Standard Deviations for Measures of Passive Coping Strategies as a Function of Pain Recall Accuracy

| Group          | Passive Coping Strategies |\hline
|                | Catastrophization | Praying/Hoping |\hline
|                 | \( M \) | \( SD \) | \( M \) | \( SD \) |
| High Estimation | 13.78   | 9.22     | 18.44   | 8.97    |
| Low Estimation  | 10.82   | 7.70     | 14.62   | 8.07    |

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Table 8

Multivariate and Univariate Analyses of Variance for Passive Coping

<table>
<thead>
<tr>
<th>Source</th>
<th>MANOVA</th>
<th>Catastrophization</th>
<th>Praying/Hoping</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$F(2, 97)$</td>
<td>$F(1, 98)$</td>
<td>$F(1, 98)$</td>
</tr>
<tr>
<td>Estimation Group</td>
<td>2.88</td>
<td>3.04</td>
<td>5.01</td>
</tr>
<tr>
<td>(High or Low Estimation)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Significance Level</td>
<td>.06</td>
<td>.08</td>
<td>.03</td>
</tr>
</tbody>
</table>

Note. Multivariate $F$ ratios were generated from Wilks' Lambda statistic.
Table 9

Means, Standard Deviations, and Intercorrelations for Pain Recall and Initial, Middle, and Last Pain Recordings of the Month

<table>
<thead>
<tr>
<th>Variable</th>
<th>M</th>
<th>SD</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Pain Recall</td>
<td>37.83</td>
<td>23.57</td>
<td>--</td>
<td>.74*</td>
<td>.70*</td>
<td>.69*</td>
</tr>
<tr>
<td>2. Initial 10 days</td>
<td>28.11</td>
<td>22.18</td>
<td>--</td>
<td>.64*</td>
<td>.79*</td>
<td></td>
</tr>
<tr>
<td>3. Middle 10 days</td>
<td>29.36</td>
<td>23.65</td>
<td>--</td>
<td></td>
<td>.68*</td>
<td></td>
</tr>
<tr>
<td>4. Final 10 days</td>
<td>27.30</td>
<td>24.73</td>
<td>--</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Note. *p < .01.
Table 10

Regression Analysis Summary for Initial, Middle, and Final Pain Recordings of the Month Predicting Pain Recall

<table>
<thead>
<tr>
<th>Variable</th>
<th>$B$</th>
<th>$SEB$</th>
<th>$\beta$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Initial 10 Days</td>
<td>.46</td>
<td>.11</td>
<td>.43*</td>
</tr>
<tr>
<td>Middle 10 Days</td>
<td>.36</td>
<td>.09</td>
<td>.36*</td>
</tr>
<tr>
<td>Final 10 Days</td>
<td>.10</td>
<td>.10</td>
<td>.11</td>
</tr>
</tbody>
</table>

Note. $R^2 = .64$ ($N = 100, p < .01$).

*p < .01.
Table 11

Hierarchical Moderated Multiple Regression Analysis for Effects of Recall Accuracy, Actual Pain, and the Interaction Term on Passive Coping

<table>
<thead>
<tr>
<th>Possible Moderator</th>
<th>Controlling for</th>
<th>$R^2$</th>
<th>Change in $R^2$</th>
<th>Error df</th>
</tr>
</thead>
<tbody>
<tr>
<td>Recall Accuracy</td>
<td>---</td>
<td>0.02</td>
<td>---</td>
<td>---</td>
</tr>
<tr>
<td>Actual Pain **</td>
<td>Recall Accuracy</td>
<td>0.09</td>
<td>0.07</td>
<td>97</td>
</tr>
<tr>
<td>Recall Accuracy</td>
<td>Recall Accuracy, Actual Pain</td>
<td>0.11</td>
<td>0.01</td>
<td>96</td>
</tr>
</tbody>
</table>

Note. *p < .05. **p < .01.
Table 12

Hierarchical Moderated Multiple Regression Analysis for Effects of Recall Accuracy, Fertility Distress, and the Interaction Term on Passive Coping

<table>
<thead>
<tr>
<th>Possible Moderator</th>
<th>Controlling for:</th>
<th>$R^2$</th>
<th>Change in $R^2$</th>
<th>Error df</th>
</tr>
</thead>
<tbody>
<tr>
<td>Recall Accuracy</td>
<td>---</td>
<td>0.02</td>
<td>---</td>
<td>---</td>
</tr>
<tr>
<td>Fertility Distress**</td>
<td>Recall Accuracy</td>
<td>0.09</td>
<td>0.07</td>
<td>97</td>
</tr>
<tr>
<td>Recall Accuracy X Fertility Distress**</td>
<td>Recall Accuracy, Fertility Distress</td>
<td>0.12</td>
<td>0.03</td>
<td>96</td>
</tr>
</tbody>
</table>

Note. ** $p < .01$. 

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Table 13

Hierarchical Moderated Multiple Regression Analysis for Effects of Recall Accuracy, Psychological Wellbeing, and the Interaction Term on Passive Coping

<table>
<thead>
<tr>
<th>Possible Moderator</th>
<th>Controlling for:</th>
<th>( R^2 )</th>
<th>Change in ( R^2 )</th>
<th>Error df</th>
</tr>
</thead>
<tbody>
<tr>
<td>Recall Accuracy</td>
<td>---</td>
<td>0.02</td>
<td>---</td>
<td>---</td>
</tr>
<tr>
<td>Psychological Wellbeing*</td>
<td>Recall Accuracy</td>
<td>0.09</td>
<td>0.07</td>
<td>97</td>
</tr>
<tr>
<td>Recall Accuracy</td>
<td>Recall Accuracy, Psychological Wellbeing*</td>
<td>0.11</td>
<td>0.02</td>
<td>96</td>
</tr>
</tbody>
</table>

Note. * \( p < .05 \).
Table 14

Hierarchical Moderated Multiple Regression Analysis for Effects of Recall Accuracy, Actual Pain, and the Interaction Term on Active Coping

<table>
<thead>
<tr>
<th>Possible Moderator</th>
<th>Controlling for:</th>
<th>$R^2$</th>
<th>Change in $R^2$</th>
<th>Error df</th>
</tr>
</thead>
<tbody>
<tr>
<td>Recall Accuracy</td>
<td>---</td>
<td>0.01</td>
<td>---</td>
<td>---</td>
</tr>
<tr>
<td>Actual Pain</td>
<td>Recall Accuracy</td>
<td>0.06</td>
<td>0.04</td>
<td>97</td>
</tr>
<tr>
<td>Recall Accuracy X Actual Pain*</td>
<td>Recall Accuracy, Actual Pain</td>
<td>0.09</td>
<td>0.03</td>
<td>96</td>
</tr>
</tbody>
</table>

Note. * $p < .05$. 

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Figure 1

Accuracy of Pain Recall Distribution
(M = 1.68, SD = 1.20, N = 100)
REFERENCES


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