ILLNESS SELF-SCHEMA IN

SYSTEMIC LUPUS ERYTHEMATOSUS

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Many thanks to all those who have helped with my research, which has resulted in this long-awaited thesis.

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Systemic lupus erythematosus (SLE) is a relatively rare autoimmune disease with no known aetiology or cure. In addition to numerous physical symptoms, those living with SLE have also been shown to experience significant emotional and psychosocial difficulties. There has been little psychological research into SLE despite the rapidly increasing interest in health psychology and quality of life issues over the last two decades. One such issue that has commanded particular attention is that of cognitive bias in individuals with chronic pain and/or chronic illness. Cognitive bias toward illness-related information is theorised to indicate the presence of an illness self-schema, and is a valuable tool of investigation as it permits access to a level of cognitive structure that is inaccessible via self-report instruments.

The primary focus of the present study is to investigate recall bias for pain- and illness-related words in SLE patients. This bias is explored relative to the recall of neutral words and depression-related words, and also relative to the responses of rheumatoid arthritis (RA) patients and healthy controls. Two hypotheses are proposed: firstly, that bias is related to disease activity; and secondly, that bias is related to the combination of illness and depression.

The findings provide support for the second hypothesis, with the additional caveat that the nature of the pain/illness stimuli used is important in determining the presence of cognitive bias. No recall bias for illness-related words as a whole was found in any of the groups, nor was there evidence of a recall bias in the SLE and RA patients when they were divided according to depression status. However, when the illness words were examined separately according to “sensory pain” and “disability-related” words, a clear bias for disability words was found in the depressed patient group. It is concluded that there is a relationship between
depression in chronically ill individuals, and the way in which such individuals process disability-related words. In accordance with the *schema-enmeshment model* (Pincus & Morley, 2001), it is suggested that both a pain-schema and an illness-schema exist, and it is when these two schemas become enmeshed with the self-schema that depression occurs in chronic pain/chronically ill patients.

The cognitive bias assessment paradigm adopted in this study-one that is typically used in similar investigations—is lengthy, requires sophisticated equipment and can be difficult to interpret on an individual level. The present study investigates the relationship between cognitive biases in SLE patients and a recently-developed task, PRISM, which appears to symbolise the enmeshment of illness-, pain- and self-schemas. Analyses confirmed that recall of negative illness words was the only independent predictor of PRISM scores. This suggests that PRISM, a quick and easy task to administer, may have considerable usefulness as a clinical tool to assess information relevant to the enmeshment of illness- and self-schema. A greater understanding of schema and the processing styles of chronically ill patients will allow for more effective psychological treatment such that quality of life can be improved.
SECTION I:

LITERATURE REVIEW
1. **SYSTEMIC LUPUS ERYTHEMATOSUS**

1a. *Introduction*

Systemic lupus erythematosus (SLE) is a chronic autoimmune disease with no known aetiology or cure. SLE typically affects multiple organ systems such that they become inflamed and sometimes damaged. Common symptoms include extreme fatigue, arthritis, fever, anaemia, pleurisy, facial skin rash and hair loss. SLE is potentially life-threatening when it affects essential organs such as the heart, central nervous system or kidneys (McCracken et al, 1995).

SLE is rare, with prevalence rates in Caucasian populations estimated at between 12 and 50 per 100,000, but in non-Caucasian populations the prevalence rate is three times as high. Non-Caucasian race is also a risk factor for death from SLE (Wallace, 1995). SLE is far more common in females, occurring eight times more often in women than in men (McAlindon, 2000).

The disease tends to follow an unpredictable and chronic course with alternating exacerbations (flares) and remissions of symptoms (Segui et al, 2000). Typically, remissions refer to a reduction in symptoms such that the person is able to live a relatively normal lifestyle. Symptoms can range from mild to severe from person to person, or from flare to flare within the same person. New symptoms may continue to appear years after the initial diagnosis (National Institute of Health (NIH), 2001). Diagnosis is often difficult, as many of the symptoms of SLE are similar to those of other illnesses. To complicate diagnosis further, symptoms are often not present concurrently, with different symptoms presenting at different times during the illness. Newly diagnosed patients have been experiencing symptoms such as extreme fatigue, weakness and frequent infections for an average of three years (Wallace, 1995). Treatments for partial relief of the
symptoms of SLE include corticosteroids and non-steroidal anti-inflammatory medications, as well as immunosuppressant medication (NIH, 2001). However, none of these treatments are able to provide a cure for the illness and are more aimed at preventing the development of different symptoms.

1b. Neuropsychiatric Manifestations of SLE

"Neuropsychiatric" is a collective term for both the neurological and psychiatric problems known to be associated with SLE. Such problems can indicate that there is central nervous system (CNS) involvement. That is, just as SLE can manifest in inflammation of the kidneys or lungs, it can also result in inflammation of various parts of the brain. Common neuropsychiatric sequelae range from overt manifestations such as seizure disorders, strokes, psychosis and mood disorders, to headaches or subtle abnormalities in cognitive functioning (Hanly & Liang, 1997; Iverson, 1995).

The neuropsychiatric manifestation of SLE has been the focus of the largest amount of psychological research in the SLE field, however, still relatively little is known about the frequency of neuropsychiatric presentations and the underlying mechanisms that account for their development. Estimations of frequency vary from 14 to 75 percent (Hanly & Liang, 1997). These discrepancies in estimated prevalence are likely to be due to methodological differences in patient selection, classification and definition, and attribution of cause (Kozora et al, 1996). Some studies, for example, classify depression as a neuropsychiatric manifestation of SLE, whilst other studies do not, considering depression to be an understandable psychological reaction to a potentially debilitating illness. To complicate matters further, corticosteroid medication (a standard treatment for SLE) is known to provoke disturbances in mood and cognition in some patients (Alpay & Cassem,
1. Systemic Lupus Erythematosus

2000). Thus, considerable controversy exists as to the extent and aetiology of the numerous neuropsychiatric manifestations of SLE. It is often difficult to differentiate the primary symptoms of disease from the psychological consequences of living with a chronic illness. Of the neuropsychiatric manifestations, there are two areas of particular interest to the psychological functioning of patients with SLE: cognitive dysfunction and psychiatric morbidity.

1b i) Cognitive Dysfunction

The most common of the neurological complications found in SLE is cognitive dysfunction (Ainiala et al., 2001; Grant et al., 1997). Once again, the estimated prevalence varies widely from study to study (12 to 87 percent) (Harrison & Ravdin, 2002). However, suffice to say that in all studies a sizeable proportion of SLE patients demonstrate cognitive problems. Deficits are most frequently observed in the areas of memory, simple attention, visuospatial processing and psychomotor speed (Ainiala et al., 2001).

Cognitive problems are more common in SLE patients with prior or current overt neuropsychiatric (CNS) involvement, but have also been found to occur in patients without overt CNS involvement at a higher rate than that found in healthy controls (Monastero et al., 2001; Denburg et al., 1997). In an attempt to delineate the relationship between these factors, Kozora and colleagues (1996) compared the cognitive and psychological functioning of SLE patients with no known CNS involvement with that of rheumatoid arthritis (RA) patients and healthy controls. One-third of the SLE patients were found to have cognitive abnormalities, indicating that overt CNS disease involvement is not the only cause of cognitive deficits in SLE. However, a similar incidence (31%) of cognitive impairment was found in the RA group. RA is an autoimmune disease causing joint inflammation,
but is not typically associated with CNS involvement. These findings suggest that the mechanisms underlying the cognitive deficits may not be specific to SLE, but rather may be associated with typical chronic illness symptoms such as pain, fatigue, mood disturbance and medication effects. All of these factors are known to affect cognitive processing. However, one marked difference between the two patient groups was in learning efficiency. SLE patients exhibited significantly more difficulty in encoding novel material than did the RA patients. It is conceivable that an SLE-specific process may mediate this aspect of cognitive dysfunction.

Research to date has demonstrated that cognitive impairment is common in SLE, but more research is required in order to identify the underlying mechanisms.

**1b ii) Psychiatric Morbidity**

Point prevalence of psychiatric disorders in SLE is estimated to be approximately 20%, although some studies have found prevalence rates to be as high as 70% (Hugo et al, 1996; Hay et al, 1994; Wekking, 1993). An accurate estimate of the occurrence of psychiatric disorders is complicated by methodological differences between studies. Studies relying on questionnaire assessments typically overestimate the occurrence of depression, due to the preponderance of somatic items that are influenced by current physical symptomatology (such as aches or fatigue). Moreover, some of the documented psychiatric morbidity can be attributed to a primary manifestation of neuropsychiatric involvement (such as psychosis). Nonetheless, a considerable number of psychiatric disorders cannot be explained by a direct expression of the disease (such as depression in non-CNS SLE patients) and more likely reflect psychological consequences of living with a chronic illness (Denburg et al, 1997; Kozora et al, 1996).
1c. Psychosocial Difficulties Associated with SLE

In addition to the psychiatric morbidity typically attributed to CNS involvement, a growing body of evidence indicates that SLE is associated with a number of psychological and emotional difficulties. In a study of nearly 400 Australian women with SLE, the most commonly reported problems included depressed mood, stress and changes in body image (Bauman et al, 1990). Iverson (1995) reported that SLE is associated with psychological problems including depression, low self-esteem, sleep disturbance, fear of death, emotional lability and marital discord. Indeed, a recent study comparing cancer patients and SLE patients found that cancer patients were less demoralised, less emotionally distressed, in less pain, and reported more benefit-finding associated with their illness (Katz et al, 2001). These data indicate that it is often difficult to cope with living with SLE, such that it frequently has emotional consequences that can lead to high levels of distress.

Whilst some research has been conducted into the psychological factors associated with SLE, the area remains relatively unexplored when compared with such research in other chronic illness populations. Rheumatoid arthritis (RA), for example, is an autoimmune disease that is similar to SLE in its mechanisms, symptoms and treatment. Presumably because it is more common (occurring in approximately 1% of the population), RA has commanded considerable attention in both the medical and psychological literature. A consistent finding has been that RA patients exhibit a higher rate of depression than the general population (Alpay & Cassem, 2000; Katz & Yelin, 1993; Creed, 1990). Other findings include the importance of active coping strategies, social support, realistic disease interpretations and self-efficacy in minimising impairment and disability (see Young, 1992 for a review).
Whilst patients with RA have been shown to experience considerably more psychological distress than healthy controls, evidence suggests that the level of distress is at least as high or higher in SLE patients. Burckhardt and colleagues (1993) found that SLE and RA patients reported similar levels of psychosocial impact of disease and perceived health status. Non-CNS SLE patients were found to experience greater emotional and psychological distress (42%) than RA patients (7%) or controls (6%) (Kozora et al, 1996). Wekking and colleagues (1991) found that the number and intensity of daily stressors were more strongly related to physical well-being in SLE than in RA.

Considering the pain and lifestyle limitations that RA and SLE impose on patients, it is not surprising that depression rates are elevated. In many ways it makes intuitive sense that the chronically ill with particularly severe pain and discomfort will be the most depressed. Indeed, it has been assumed that disease severity plays the most significant role in patients’ emotional distress. However, over the last two decades the evidence from the “quality of life” literature has challenged this assumption.
2. Psychological Factors & Chronic Illness

2a. Quality of Life in Chronic Illness

“Quality of life” has become a very popular concept in both the medical and psychological literature in recent years (Wood-Dauphinee, 1999). Although there is contention over its specific definition, quality of life (QOL) broadly refers to the patient’s subjective account of his/her health, incorporating variables such as physical mobility, emotional well-being, social life, and overall well-being (Jenkinson et al, 1993). Whilst laboratory and clinical indicators of disease were once considered to provide a complete picture of health status, health professionals are now conceding that the patient’s perspective is also of substantial significance (Wood-Dauphinee, 1999). This shift has been necessitated by the growing body of evidence indicating that QOL and disease variables often have little or no association with one another (Persson & Sahlberg, 2002; Stoll et al, 2001; Wang et al, 2001; Gladman et al, 1996). That is, a more severe disease process does not necessarily imply a poorer QOL. It follows then, that improving physical symptoms through medical treatment should not be the only consideration when dealing with chronically ill patients, as this may not necessarily improve a patient’s QOL (Muldoon et al, 1998). A greater understanding of the psychosocial factors contributing to the way in which people endure chronic illness is therefore of great importance in determining how to best improve QOL.

2b. Psychological Factors & the Disease Process

Investigation into the psychological factors associated with chronic illness is not only important in terms of alleviating the distress experienced by these patients: the evidence indicates that psychological processes and emotional states can actually influence the progression of disease. In reviewing the literature in RA, for
example, Young (1992) found that psychological measures have been shown to be more important determinants of disability than have disease variables. In particular, catastrophising has been convincingly shown to influence pain experiences. The tendency to magnify or exaggerate the threat value or seriousness of pain is associated with an increase in disability, pain behaviour and use of analgesic medication (Sullivan et al, 2001).

In the SLE literature, a finding of particular interest indicates that SLE patients differ from healthy controls in their immunological response to acute psychological stress (Pawlak et al, 1999). Anecdotal evidence supporting this finding is the clinical observation that stress often precedes an increase in disease activity in SLE patients. Da Costa and colleagues (1999) found that the major short-term determinants of functional disability in SLE patients were not demographic- or disease-related factors, but rather stress associated with negative life events. There is considerable evidence demonstrating that there is a link between the way people think, feel and behave, and how well they tend to withstand illness and poor health (Turk & Okifuji, 2002; Baum & Posluszny, 1999; Compas et al, 1998). Thus, investigation of attitudes and behaviours should lead not only to an improvement in the emotional well-being of patients, but also in their physical well-being.

2c. Attitudes & Illness

Evidence indicates that some patients have significantly higher rates of distress than others with the same chronic illness and the same severity of symptoms. But what is the origin of these inflated rates of distress? In SLE, it cannot be fully explained by disease severity or CNS involvement, so another factor must contribute to the high levels of distress observed. Self-regulation theory suggests
that attitudes towards illness are one important determinant of emotional adjustment to illness (Leventhal et al, 1984; Nerenz & Leventhal, 1983). That is, the way in which someone perceives his or her illness will affect the way in which he or she copes with that illness, which will in turn affect the level of psychological distress experienced. The self-regulation model proposes that a patient’s representation of his or her illness has five primary components - identity, cause, time-line, consequences and cure - as depicted in Table 1 below. These components are proposed to reflect a patient’s cognitive response to symptoms and illness, whilst emotional responses are processed in parallel. The components are not necessarily independent factors, but rather are likely to be inter-related.

**Table 1:** The five components of illness representation according to self-regulation theory.

<table>
<thead>
<tr>
<th>Component</th>
<th>Relates to:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Identity</td>
<td>the nature and symptoms of one’s illness</td>
</tr>
<tr>
<td>Cause</td>
<td>the patient’s beliefs about the likely cause(s) of the condition</td>
</tr>
<tr>
<td>Time-line</td>
<td>perceptions regarding the likely duration and pattern of illness</td>
</tr>
<tr>
<td>Consequences</td>
<td>beliefs regarding illness severity and likely impact on functioning</td>
</tr>
<tr>
<td>Cure</td>
<td>extent to which the patient considers the illness to be amenable to cure or control</td>
</tr>
</tbody>
</table>

*Adapted from Nerenz & Leventhal (1983)*

Evidence supporting this model has been found in numerous patient populations, including RA. Smith and colleagues (1988) demonstrated that RA patients who displayed negative or exaggerated interpretations of their illness (that is, perceived excessively negative consequences) were significantly more depressed and disabled than were patients whose interpretations were less extreme. A similar
finding indicated that pain in RA patients is as equally and independently predicted by negative illness cognitions as it is by impairment (Persson & Sahlberg, 2002). In a recent prospective study, Sharpe, Sensky & Allard (2001) found that beliefs about the consequences of arthritis predicted depression in recent-onset RA patients over a 21-month period.

The primary method by which illness attitudes have been explored is self-report measures. Indeed, a questionnaire has been developed (based on the illness representation components outlined in self-regulation theory) to specifically measure illness perceptions (the Illness Perception Questionnaire; IPQ) (Weinman et al, 1996). Questionnaire measures are useful in that they assess the content of one’s beliefs, and provide information as to the degree that individuals endorse (in a strategic manner) certain statements as applying to their situation. However a disadvantage of using questionnaires is that they cannot test the proposition that cognitive representations (or sets of beliefs) of illness affect the way in which future information (for example, symptoms of illness) is processed and understood. Such a proposition has been put forward in the literature pertaining to self-schemas and cognitive bias.
3. Self-Schemas

Markus (1977) was one of the first to articulate and test out the hypothesised construct of self-schemas. She defined self-schemas as "cognitive generalisations about the self, derived from past experience, that organise and guide the processing of self-related information contained in the individual's social experiences" (Markus, 1977; p64). Self-schemas represent the way in which the self has been articulated and differentiated in memory, and have properties relating to both content and function. The content of the structure is a list of general and specific terms characteristic of the individual that have been derived from a lifetime of experience with personal data. The content of self-schemas is typically assessed through questionnaires that ask patients to endorse beliefs about themselves. The self-schema functions to organise the processing and retention of self-related information (Derry & Kuiper, 1981). That is, self-schemas determine the importance given to experiences and the subsequent interpretation and processing of those experiences. Individuals will be more attentive to, and take ownership of, experiences that are deemed to be self-relevant; that is, consistent with their self-schema (Wiginton, 1999; Rojahn & Pettigrew, 1992).

As mentioned earlier, beliefs have been typically assessed via questionnaires. In the self-schema model, questionnaires assess the schema content. Yet they cannot assess the automatic processing resulting from the presence of schema (schema function). Assessment of this schema aspect necessitates tasks that require the individual to process information automatically. These cognitive processing tasks (discussed in detail below) complement the questionnaire method, as they each assess different aspects of self-schemas.

Each person is thought to possess a number of self-schemas pertaining to various
content domains. That is, from each individual's unique experiences, one develops cognitive generalisations about oneself with regard to many aspects of life. For example, researchers have identified self-schemas in the domains of depression, Type A behaviour, age, gender, and one’s body (Schwoebel et al., 2001; Clemmey & Nicassio, 1997; Derry & Kuiper, 1981; Beck, 1976).

It is hypothesised, given the nature of self-schemas and the likely existence of domain-specific schemas, that an illness self-schema also exists. Self-regulation theory’s illness representation that was described earlier is almost identical to what we understand an illness self-schema to be. The patient is described as actively constructing a representation of his or her illness from current illness episodes, memories of prior experiences of illness, and lay understandings of illness (Nerenz & Leventhal, 1983). This representation is described as directing and regulating action, in particular, coping responses. The illness representation is described as flexible and changeable, depending on the nature of current situational stimuli. Self-regulation theory postulates that patients who have been labelled chronically ill will search their present and past concrete experiences for validating signs or symptoms, due to their cognitive representations of chronic illness.

3a. Identifying & Measuring Self-Schemas:

Cognitive Bias

Attempts to investigate the effects (functions) of self-schemas has largely focused on the area of information processing and the presence of cognitive biases towards relevant information. Such a focus makes intuitive sense, as the inevitable consequence of a structure that functions to organise and process information is to bias and distort information (Clemmey & Nicassio, 1997). Calfas and colleagues
3. Self-Schemas

(1997) state that:

“Individuals will process most thoroughly, and hence later recall, information that is consistent
with their active self-referent cognitive structures…..Recall patterns thus allow inferences
as to the presence, composition and/or operation of cognitive structures.” (p577)

Studies have demonstrated this, in that the existence of self-schemas in a given content domain affects the processing of information consistent with that domain. Most commonly, studies have investigated either selective attention (using the Stroop task or the dot probe task) (Grisart & Plaghki, 1999; Riemann & McNally, 1995), or memory bias (Pauli & Alpers, 2002; Koutantji et al, 1999; Edwards et al, 1992; Bradley & Mathews, 1988). Differential response times to schema-consistent stimuli relative to schema-inconsistent stimuli have also been investigated (Koutantji et al, 2000).

3a i) Cognitive Bias in Psychological Disorders

Much of the early research into cognitive bias was conducted in the area of psychological disorders, primarily depression and anxiety. Patients suffering from a variety of anxiety disorders (such as obsessive compulsive disorder or specific phobias) have been shown to selectively attend to, and recall more of, stimuli related to their fears (Mogg & Bradley, 1998; Williams et al, 1996). Cognitive bias in this population has become a well-established phenomenon. Although not as robustly as in anxious patients, cognitive bias has also been demonstrated in depressed populations (for a review, see Mathews, 1997). An investigation by Bradley & Mathews (1988) is representative of a typical study in this area. They examined the memory biases of depressed patients, and found that depressed patients exhibited a bias for negative material (words such as ‘ashamed’ or
’miserable’), whilst controls recalled more positive material (words such as ‘talented’ or ‘delighted’). As has been demonstrated in numerous other studies (for example, Pincus et al, 1993), the recall bias for self-referent material was significantly greater than that for other-referent stimuli. This is consistent with a self-schema model, in that information directly related to the self and consistent with core beliefs is more readily processed (Koutantji et al, 1999).

3a ii) Cognitive Bias in Chronic Pain & Chronic Illness

It is only in the last decade and a half that the information processing paradigm has been extended to pain populations. If anxious patients selectively process anxious material, and depressed patients selectively process negative material, then perhaps pain patients selectively process pain material. Research to date regarding cognitive bias for pain-related stimuli in pain patients has been somewhat inconsistent (see Pincus & Morley, 2001 for a comprehensive review).

One important reason why the literature may be inconsistent is the differing definitions of “pain” and “chronic pain”. The International Association for the Study of Pain (IASP) defines pain as “an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage” (IASP Sub Committee on Taxonomy, 1979; p250). This definition of pain is widely used, however the definition of chronic pain is more contentious. Chronic pain in its strict sense refers to physical pain that has been ongoing for a number of months for which physical explanations do not fully account for the reported severity (Birket-Smith, 2001). Typically, this refers to individuals who have sustained an injury (for example, to their lower back), and the pain has continued despite the overt injury itself having healed. This definition is not consistently applied in the chronic pain and cognitive bias literature, where “chronic pain” can refer to either this situation, or (for example) to patients who
suffer pain chronically as a direct result of a chronic illness (for example, arthritis). This issue in and of itself has been the subject of much research and debate, and therefore falls beyond the scope and aim of the present study. Suffice to say that we consider this distinction to important, as the process through which individuals experience pain and illness is different (Dersch et al, 2002). For clarity, in the present study we will use the term “chronic pain” in its strict sense unless otherwise stated.

**Attention Bias**

One of the first studies to demonstrate pain bias was conducted by Pearce & Morley (1989). Using a modified Stroop task, they found that chronic pain patients were slower to name the colours of sensory pain words (for example, ‘throbbing’ or ‘stabbing’) and affective pain words (for example, ‘sickening’ or ‘unbearable’) compared to neutral words. This attention bias was not mediated by mood, and no such bias was observed in pain-free subjects. In the only study to replicate this attention bias for all pain stimuli in chronic pain patients as a whole, Snider and colleagues (2000) only found a bias after controlling for depression. Other studies have failed to find an overall bias towards pain-related material in pain populations, and have only found attention biases in subgroups of pain patients divided according to depression, anxiety and fear of pain (Crombez et al, 2000; Pincus et al, 1998; Asmundson et al, 1997; Dehghani et al, submitted), or more specific biases according to the type of pain stimuli used (Crombez et al, 2000; Dehghani et al, submitted).

**Recall Bias**

Similar complexities and apparent inconsistencies have been found in the memory bias literature. Pearce and colleagues (1990) demonstrated memory bias for sensory pain words in chronic pain patients relative to pain-free controls, however
there are a number of reasons why this study cannot be easily compared to subsequent studies. Participants were warned of the recall task prior to encoding the words (intentional memory), which is in contrast to the incidental memory paradigm employed in other studies. Hence, this task was unlikely to have represented an automatic level of processing because it required individuals to purposefully attempt to recall information at a strategic level. The mechanisms of strategic versus automatic processing are argued to be different (Watkins et al, 2000; Richardson-Klavehn & Gardiner, 1998; Fastenau et al, 1997), and it is incidental memory that has traditionally been utilised to reflect cognitive bias at an automatic level of processing (Pincus & Morley, 2001). In addition, in Pearce and colleagues’ study (1990), the encoding stage was conducted as an auditory task, as opposed to a visual task in other recall bias studies. This methodological difference may also have contributed to conflicting results. Finally, many of the chronic pain patients were depressed. The observed recall bias may have reflected the patients' depressed mood rather than their pain per se. Indeed, Edwards and colleagues (1992) demonstrated that depressed chronic pain patients had selective recall for sensory pain-related words and affective pain-related words, whilst non-depressed chronic pain patients showed selective recall for sensory pain-related words only.

As in the original Pearce & Morley (1989) study, the pain-sensory and pain-affect contrast was adopted by two studies comparing arthritis patients and pain-free controls (Koutantji et al, 1999; Pincus et al, 1993). Both found a recall bias for sensory pain words when compared to pain affective and neutral words (self-referent encoding) for the pain groups, however, Koutantji and colleagues (1999) found that sensory words were better recalled overall, irrespective of group. The authors suggest that this bias may be as a result of pain-sensory stimuli having a self-preservation role for all human beings.
Two studies examined an alternative contrast, investigating recall patterns with regard to pain/illness words (negative illness) and pain-free/healthy words (positive illness) in depressed and non-depressed arthritis patients (Clemmey & Nicassio, 1997; Pincus et al, 1995). For further comparison, one of the studies (Pincus et al, 1995) also included positive and negative depression words, and positive and negative control words. Results indicated that whilst depressed arthritis patients selectively endorsed and recalled self-referent negative illness stimuli, but not depression-related stimuli, no such bias existed in the non-depressed arthritis patients. These findings are consistent with those of the other study, in which depressed and non-depressed RA patients were compared to depressed and non-depressed healthy controls (Clemmey & Nicassio, 1997). Depressed RA patients exhibited endorsement and recall biases towards negative illness-related information, whilst non-depressed RA patients demonstrated the opposite bias towards positive illness-related information. No bias was detected in depressed and non-depressed controls for illness related information. The authors propose that their findings provide evidence for the presence of illness self-schema in RA patients. They argue that the illness schema is associated with mood disturbance, and its content is distinct from a depressive self-schema in healthy but depressed individuals. The results of these two studies are consistent with much of the selective attention literature, in which affective distress appears to be a mediating factor in the cognitive bias of pain patients to at least some types of pain-related stimuli.

*Illness Schema*

Despite being discussed as a hypothesised construct a number of years ago (Nerenz & Leventhal, 1983), the term ‘illness schema’ was only first used in the context of cognitive bias research in the aforementioned study by Clemmey & Nicassio (1997). Although other studies may have alluded to illness schema, it has
not been specifically mentioned. Subsequent to Clemmey & Nicassio’s (1997) paper, the term was not explicitly discussed until Pincus & Morley (2001) reviewed the cognitive bias literature and proposed a model for bias in pain patients. Thus, illness schema remains a relatively under-researched area.

3a iii) Cognitive Bias for Pain Stimuli in Healthy Populations

Cognitive biases for pain-related information have been studied in populations other than chronic illness and/or chronic pain. Keogh and colleagues (2001) conducted an attentional bias study in which healthy individuals were classified according to their fear of pain (high, medium or low). Participants completed a dot-probe task in which the experimental stimuli were pain sensory words, positive words, or words related to socially threatening situations. Each word category had a list of neutral words matched for frequency and length. Healthy individuals with a high fear of pain were found to demonstrate a clear attentional bias for pain sensory words relative to individuals with a low fear of pain. This bias was specific for pain material and was not found for social threat or control words. The importance of this finding is that if a pain-specific attentional bias exists in healthy individuals, the pain bias seen in chronic pain populations is not necessarily simply a product of chronic pain states.

Koutantji and colleagues (2000) investigated memory and processing time biases for pain-related words in a group of otherwise healthy students with high or low frequencies of pain episodes. Relative to participants reporting a low number, students reporting a high number of pain episodes recalled significantly more pain-sensory words and processed such words significantly faster. These findings indicate that pain-specific cognitive biases can develop alongside multiple, discrete pain episodes in the absence of either depressed mood or chronic pain.
3a iv) Cognitive Bias for Pain Stimuli: A Summary

It is clear that no firm conclusions can be drawn regarding the specific nature of cognitive bias in pain patients. Direct comparison of studies is difficult due to differing methodologies and samples. Nevertheless, it can be said that selective attention and recall bias for sensory pain words appears to exist in at least a subgroup of patients experiencing chronic pain. Although a small number of studies have found a cognitive bias in chronic pain patients as a whole group, the majority of studies have not. The relative contributions of the pain population (chronic pain versus chronic illness), depression, anxiety, fear of pain, and type of pain stimuli, are still somewhat unclear.

Such complexity is not surprising, however, given the heterogeneity of patients with chronic pain and/or chronic illness. Cognitive bias has been most clearly established in populations where affective distress is the identifying feature (for example, in depressed or anxious populations; see Section 3a i)). In pain patients however, affective distress and overall level of functioning differs considerably from patient to patient and from illness to illness. If it were the combination of affective state and chronic pain that determined cognitive bias, such bias would not be apparent in every individual with chronic pain. A further consequence of the heterogeneity of the populations is that the personal relevance of pain stimuli will vary between individuals, depending upon the nature of their pain. Using the same pain words for groups of pain patients with different histories and different health problems may not reflect the personal schemas of each individual. Clearly, these are difficult issues to address in standardised studies, however, more research will indeed assist in untangling this complex area.
4. A MODEL FOR COGNITIVE BIAS IN CHRONIC PAIN/CHRONIC ILLNESS

Despite the complexities, Pincus and Morley (2001) have recently reviewed the literature and have proposed a model to account for the various pain-related biases in patients with chronic pain and/or chronic illness. They suggest that each individual has a self-schema, one or more illness schemas, and a pain schema. The authors define the self and illness schemas similarly to those described previously (see Section 3), whilst the pain schema is defined as incorporating the sensory-intensity, spatial, and temporal features of pain. Pincus & Morley (2001) acknowledge that pain and illness schemas are closely interrelated, yet argue that they should be considered independently for two main reasons. Firstly, some illnesses do not necessarily involve pain as a symptom (for example, diabetes), and secondly, some pain does not imply illness (for example, broken bones). This model is the first to differentiate illness schema from pain schema.\(^1\)

The authors propose that it is the interaction of three schemas-self, illness and pain-which accounts for the presence or absence of cognitive bias in pain patients. The *schema enmeshment model* asserts that when elements from illness and pain schemas are frequently activated at the same time as elements from one’s self-schema, the content of the three schemas can become incorporated into one another, or enmeshed. Thus, the individual’s view of himself or herself is disrupted (see Figure 1). Pincus & Morley propose that it is the enmeshment of these schemas that underlies cognitive bias for sensory and affective pain stimuli. Depression, or affective distress, is offered as the primary mediating factor of the extent of the enmeshment, however, whether depression is the cause or consequence remains unknown.

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\(^1\) It is noteworthy that this model, and hence the pain schema/illness schema distinction, was published after the current study had begun.
The schema enmeshment model predicts that pain patients who are depressed will show cognitive bias towards sensory and affective pain words (and potentially depressive words if the patient has a history of depression), whilst non-depressed pain patients will show a pain-sensory bias only. These two situations are represented by Figures 1C and 1B respectively. Pain-free individuals who are depressed will be biased towards depressive stimuli, whilst non-depressed pain-free individuals will demonstrate cognitive bias towards neutral and/or positive stimuli. Both of these situations are represented by Figure 1A. The differing biases between depressed pain patients and depressed healthy individuals suggest that the nature of depression in pain patients is often distinct from depression in the absence of illness. This distinction has found support in studies by Pincus and colleagues (1995) and Clemmey & Nicassio (1997).
4a. The Pictorial Representation of Illness &

Self Measure (PRISM)

When examining Pincus & Morley’s graphical representation of the schema enmeshment model, a recently developed measure of the impact of illness comes to mind. Originally conceived as a possible summary measure of adjustment to illness, the Pictorial Representation of Illness and Self-Measure (PRISM) (Buchi et al, 1998) appears to be conceptually related to the schema enmeshment model. PRISM is a task in which "subjects are asked to imagine that a small board represents his/her life and a fixed disc on the board represents his/her 'self'. The task is to place another disc (illness) on the board to represent the current importance of illness in the patient's life." (Buchi et al, 1998; p222). The outcome measure is the distance between the centres of the two discs (see Figure 2). That is, PRISM measures the extent to which patients perceive their illness to be part of their sense of self. Conceptually, this is very similar to the schema enmeshment described by Pincus & Morley (2001). Naturally, a self-report instrument such as PRISM requires strategic processing, whereas the function of schemas and the resulting cognitive bias is automatic. Nevertheless, due to their superficial similarity, the relationship between so-called schema enmeshment and PRISM may prove to be interesting.
Buchi and colleagues (1998) investigated the utility of PRISM in a sample of RA patients, and found that the closer patients placed the illness disc to the self disc, the more pain, functional impairment and depression they experienced. In addition, PRISM distance was related to coping resources and perceived control over illness. Comparable results were found in a sample of SLE patients (Buchi et al, 2000). More detailed information about PRISM was gained from a validation study involving over 700 patients with a variety of physical illnesses (Buchi et al, 2002). From the results, the authors argue that PRISM is a potentially useful means of rapidly assessing what they term, “burden of suffering due to illness”, where a smaller distance between the illness and self discs indicates a greater burden of suffering for the individual. To a large extent this finding was derived from a qualitative component of the validation study, in which patients were asked to describe their reasons for their placement of the illness disk. However, without a quantitative measure of burden of suffering to function as a ‘gold standard’, it is difficult to establish the validity of PRISM as a measure of suffering.

Although preliminary PRISM results are encouraging, it remains unclear as to precisely what PRISM is measuring. It is possible that PRISM merely provides a
fairly crude indication of how significant an individual’s illness is to him or her. Furthermore, there are other measures (for example, quality of life or disability questionnaires) that seem to measure a similar concept. However, given the conceptual similarities between PRISM and the schema enmeshment model, it seems worthwhile to investigate the relationship between the two. It is possible that PRISM can provide an indication of the extent of enmeshment of self and illness schemas. It may be a valuable adjunct to other illness self-schema measures, which have typically relied on more complex and lengthy information processing paradigms.
If we were to understand the specifics of cognitive bias in chronically ill patients, in what way, and for whom, would that be beneficial? The basis for this answer resides in the application of cognitive therapy to pain patients. Cognitive theory states that it is an individual’s thoughts and interpretation of an event that influence mood and behaviour in response to that event (Beck et al, 1979). Cognitive therapy aims to modify unrealistically negative and dysfunctional thoughts in order to improve mood and behaviour. Schemas underlie automatic thoughts and influence thinking patterns and information processing. As discussed earlier, if patients (or a subset of) with chronic illnesses and/or chronic pain demonstrate cognitive bias for illness- and/or pain-related stimuli, this is argued to indicate the presence of negative illness-related schema or schema enmeshment.

A greater understanding of the nature of cognitive bias and thinking patterns in pain patients has the potential to assist our understanding of underlying belief systems. It is well-established in cognitive therapy literature that such an understanding allows for more effective modification of dysfunctional thoughts and schemas. Such modification is likely to reduce affective distress and improve QOL. McCracken & Turk (2002) reviewed behavioural and cognitive-behavioural treatment (CBT) programs for chronic pain, and found them to be generally effective. They identified initial distress levels, beliefs about pain and illness, and self-efficacy as moderators of treatment outcome. In a review and meta-analysis of randomised controlled trials of CBT for chronic pain patients, Morley and colleagues (1999) also found this treatment to be effective. In the long-term, if cognitive bias knowledge was to assist clinicians in the administration of CBT, treatment outcome could be maximised.
To our knowledge, all but one of the studies to date that have investigated cognitive bias for pain stimuli have done so using chronic pain patients or arthritis patients (RA and osteoarthritis). The exception is a recent investigation of the impact of diagnosis on cognitive bias in patients with ankylosing spondylitis, an autoimmune disease causing severe, chronic back pain (Wells et al., 2003). This narrow focus is surprising given the numerous other chronic illnesses in which chronic pain is a significant symptom. One such illness is SLE. Various elements of illness have been implicated as contributing to cognitive bias in pain; repeated interruption of daily activities as a result of the illness, degradation of performance in comparison to desired standards, frustration, and unattained goals (Pincus & Morley, 2001; Nerenz & Leventhal, 1983). As all of these elements are present in SLE, it is reasonable to anticipate some evidence of cognitive bias in such patients.

As discussed earlier, the literature on the cognitive, emotional and psychological sequelae of SLE is limited despite the substantial impact of the illness on the lives of its patients. The majority of the literature has focused on self-report measures and correlational analyses. For example, Baker & Wiginton (1997) interviewed 38 women with SLE to explore their “commonsense representations” of their illness. In a related study, Wiginton (1999) utilised a cognitive mapping strategy to investigate how 20 women with SLE viewed their illness. The conclusion of both of these studies was that SLE is a complex illness and is represented differently by different patients. Whilst qualitative and quantitative research based on self-report methodologies is invaluable as a means of exploring the content of schemas, the methodological limitations associated with such research need to be considered. For example, questionnaires are susceptible to response bias, and can only investigate explicit cognitive structures at a strategic level of processing. A major
advantage of the information processing paradigm is that it allows researchers to
gain access to levels of cognitive structure not accessible through self-report
formats (Pincus & Morley, 2001). That is, information processing paradigms allow
the function of underlying schemas to be identified.

The investigation of cognitive bias in SLE patients may be all the more interesting
as a result of the fluctuating course of the illness. As mentioned earlier, SLE
patients typically experience periods of symptom exacerbation (flares) and of
symptom improvement (non-flares) from the time of diagnosis. During non-flares
patients can be virtually symptom-free with few interruptions to their daily routine.
Surprisingly, the majority of SLE studies have not accounted for this flare/non-
However, even during non-flares the continued threat of illness remains, as in most
cases the symptoms of SLE will recur. This creates the situation where individuals
with the same chronic illness can be tested, in which a proportion are in pain and
ill, and the remainder are not in pain, are not overtly ‘ill’, but still live with the
threat of ‘illness’. This is not the case in arthritis or chronic pain, where although
symptom severity fluctuates, some symptoms are commonly present at all times.
Thus, a ‘full remission’ cannot reliably be said to exist in these patients.

Symptom severity has been found to not necessarily be associated with cognitive
bias in pain patients (Pincus et al, 1995), however symptom absence or presence
has not been investigated. Schemas are known to be dynamic structures, and are
hypothesised to change and develop according to the accumulation of life
experiences. They can also remain dormant for periods of time, and can then be
activated by particular situations. It is plausible that such activation may occur
with the onset of symptoms (change of status from non-flare to flare). Edwards and
colleagues (1995) investigated cognitive bias in a small group of chronic pain
patients who were about to undergo surgery to ameliorate their pain complaint. Although the findings did not reach significance, there was a trend that chronic pain patients exhibited a bias for pain-related words, whilst recovered chronic pain patients (six months post-surgery) did not. This concept has been investigated in parallel circumstances by Bradley & Mathews (1988). They compared the memory biases of depressed patients to those who had recovered from depression in an effort to establish whether memory bias results from the depressed state itself, or from more persistent individual cognitive differences. The findings evidenced that depressed patients, but not controls or patients who had recovered, demonstrate a memory bias for negative self-referent material. Results consistent with this investigation have been found in numerous studies, for example, Dohr and colleagues (1989). These studies suggest that schemas are not static, but rather depend to some extent on current schema-related experiences.

As discussed earlier, the schema enmeshment model proposes that it is depression that accounts for the activation of illness schema (or enmeshment of illness and self schemas) (Pincus & Morley, 2001). That is, schemas relating to illness are considered to be dynamic structures whose interaction and activation are influenced by the depressed mood of the patient.
7. THE PRESENT STUDY

Due to their potential influence upon information processing and behaviour, a greater understanding of illness schemas in SLE may contribute to our knowledge of the adaptive and maladaptive ways in which patients attempt to deal with this illness. Such an understanding is particularly important in SLE due to the documented levels of distress experienced by patients with this illness.

The present investigation seeks to examine illness schemas via cognitive biases in SLE patients. Due in part to the absence of any prior research in this area, two main hypotheses are proposed. The first relates to the disease status of the SLE patients, and will be referred to as the Flare Hypothesis. If illness schemas are activated by the re-emergence of disease symptoms, then dichotomising SLE patients according to flare status should result in a difference between groups, such that patients in a flare will demonstrate cognitive bias for illness words. The second hypothesis (Depression Hypothesis) arises from the evidence in other patient populations that depression is the mediating factor in cognitive bias. If illness schemas are invoked as part of a process through which ill individuals become depressed, then dividing the SLE patients according to depression status should result in a difference between the groups, where depressed patients demonstrate a bias but non-depressed patients do not.

A clinical comparison group of RA patients is included in the present study. As RA has been the most common patient population that has been tested for cognitive bias, and RA and SLE are both autoimmune, rheumatological diseases, such a comparison was thought to be valuable in this investigation. For the purposes of the depression hypothesis the depressed SLE patients and the
depressed RA patients will be compared as a group (depressed ill) to the non-depressed patients (non-depressed ill). A healthy control group is also included.

In accordance with self-schema research under the information processing paradigm, the current study measures illness schema via endorsement and recall of positive and negative illness-related words. The methodology has its basis in the work of Pincus and colleagues (1995). Additional positive and negative control words are included in the information processing task in order to determine whether any affectively valenced bias is specifically related to illness-related words, or merely represents a bias in general (Pincus et al, 1995). Positive and negative depression-related words are also included in order to ascertain the nature of depressive schema in depressed, ill patients. In keeping with previous findings (Clemmey & Nicassio, 1997; Pincus et al, 1995), it is hypothesised that neither the patient groups (SLE/RA or depressed ill/non-depressed ill) nor the control group will exhibit a bias for the depression or control words.

To complement the experimental paradigm, PRISM (Buchi et al, 1998) is also administered. It is predicted that the distance that patients place the ‘illness’ from the ‘self’ in the PRISM task will be inversely related to the degree to which patients demonstrate cognitive bias towards negative illness-related words. Support for this hypothesis would confirm the proposed relationship between the PRISM task and the concept of illness self-schema (or enmeshment of illness and self schemas).

In addition to these primary aims and hypotheses, the current investigation aims to explore the relationships between illness self-schema, disease activity, and a number of other psychosocial aspects of SLE and RA. These will include
7. The Present Study

depression, quality of life, functional impairment, and illness perception. All of these constructs have been implicated in the psychological and physical well-being of rheumatology patients (Da Costa et al, 2000; Abu-Shakara et al, 1999; Denburg et al, 1997; Bauman et al, 1990), and will be measured via self-report instruments.
SECTION II:

COGNITIVE BIAS IN SYSTEMIC LUPUS ERYTHEMATOSUS

The following section is based on a manuscript prepared for publication:

1. INTRODUCTION

Evidence of cognitive bias in depression and anxiety has sparked an increasing interest in the potential for pain-related bias in patients suffering from chronic pain and/or illness. Cognitive biases are thought to arise from the activation of cognitive structures, or schemas, which function to organise and prioritise stimuli (Derry & Kuiper, 1981). Individuals possess a number of these schemas pertaining to various content domains, different combinations of which will be activated at different times. For example, depressed patients demonstrate memory bias for negative self-referent material (Mathews, 1997; Bradley & Mathews, 1988), and anxious patients show selective attention for anxiety-related stimuli (Keogh et al, 2001; Williams et al, 1997). It makes intuitive sense that individuals who have endured pain or illness for an extended period of time may demonstrate pain/illness-congruent biases, thereby implicating the presence of active pain/illness schema.

Results to date in this area have yielded somewhat conflicting results (see Pincus & Morley, 2001 for a comprehensive review). In the selective attention literature, one of the first studies to demonstrate pain bias found that, relative to pain-free controls, chronic pain patients showed selective attention for pain-affective words (e.g. ‘unbearable’) and pain-sensory words (e.g. ‘throbbing’) (Pearce & Morley, 1989). In the only study to replicate this attention bias for all pain stimuli in chronic pain patients as a whole, Snider and colleagues (2000) only found a bias after controlling for depression. Other studies have only found attention biases in subgroups of pain patients categorised according to depression, anxiety and fear of pain (Keogh et al, 2001; Crombez et al, 2000; Pincus et al, 1998; Asmundson et al,
1997; Dehghani et al, submitted), or more specific biases according to the type of pain stimuli used (Crombez et al, 2000; Dehghani et al, submitted).

In keeping with the findings from the selective attention literature, investigation of pain-related memory bias has implicated two main mediating variables: the nature of the pain stimuli used and the mood status of the pain patient. With reference to the former, all studies have found a recall bias for sensory pain words in pain patients as a whole (Koutantji et al, 1999; Pincus et al, 1993; Edwards et al, 1992; Pearce et al, 1990). Only two of these, however, employed a pain-free control group, one of which found a sensory pain bias irrespective of pain status (Koutantji et al, 1999). Recall bias for non-sensory pain words has generally only been demonstrated in depressed pain patients (Clemmey & Nicassio, 1997; Pincus et al, 1995; Edwards et al, 1992). This bias is relative to non-depressed pain patients, and depressed and non-depressed control groups (Clemmey & Nicassio, 1997), and is specific for pain words (Pincus et al, 1995). Pincus & Morley (2001) argue that the results indicate that depression is the key determinant of pain-related bias in pain patients.

The apparent complexity in this area is not entirely surprising, given the heterogeneity of patients with chronic pain and/or chronic illness. Cognitive bias was originally, and has been most clearly, established in populations where affective distress is the identifying feature (that is, in depression and anxiety). In pain patients, affective distress and overall level of functioning differs considerably from patient to patient and from illness to illness. If it were the combination of affective state and chronic pain that determines cognitive bias,
such bias would not be apparent in every individual with chronic pain. A further consequence of the heterogeneity of this population is that the personal relevance of pain stimuli will vary between individuals. Using the same pain words for groups of pain patients with different histories and different health problems may not reflect the personal schemas of each individual.

All of the cognitive bias research in pain patients to date (save one study, Wells et al, 2003) has been conducted on just two patient populations: rheumatoid arthritis (and arthritis) and chronic pain\(^2\) patients. This narrow scope is somewhat surprising, given the numerous other conditions in which persisting pain is a significant symptom. One such condition is the autoimmune disease, systemic lupus erythematosus (SLE). Similar to rheumatoid arthritis (RA) in many ways, SLE is a chronic, autoimmune disease with no known aetiology or cure. SLE typically affects multiple organ systems such that they become inflamed and sometimes damaged. Common symptoms include extreme fatigue, arthralgia and joint pain, fever, anaemia, pleurisy, facial skin rash and hair loss. SLE is potentially life-threatening when it affects essential organs such as the heart, central nervous system or kidneys (McCracken et al, 1995). Diagnosis is often difficult, as many of the symptoms of SLE are similar to those of other illnesses and are often not present concurrently. Newly diagnosed patients have been experiencing symptoms such as extreme fatigue, weakness and frequent infections for an average of three years (Wallace, 1995). The disease tends to follow an

\(^2\) We use the term “chronic pain” to refer to physical pain that has been ongoing for a number of months in which physical explanations do not fully account for the reported severity (Birket-Smith, 2001). Typically, this refers to individuals who have sustained an injury (for example, to their lower back), and the pain has continued despite the injury itself having healed.
unpredictable and chronic course with alternating exacerbations (flares) and remission of symptoms.

It is partly because of this last characteristic of SLE (alternating flares and remissions) that the investigation of cognitive bias in SLE patients may be particularly interesting. During remission patients can be virtually symptom-free, however, the threat of illness remains as in most cases the symptoms of SLE will recur. This creates the situation where individuals with the same chronic illness can be tested in which a proportion are in pain and ill, and the remainder are not in pain, are not overtly ‘ill’, but still live with the threat of ‘illness’. This is not the case in arthritis or chronic pain, where although symptom severity fluctuates, some symptoms are commonly present at all times. Whilst symptom severity is not necessarily associated with cognitive bias in pain patients (Pincus et al, 1995), symptom absence or presence has not yet been investigated. Since we know that schemas are dynamic structures that can be dormant for periods of time and then activated by particular situations (Clemmey & Nicassio, 1997; Markus, 1977), it is plausible that such activation may occur with the onset of symptoms (change of status from remission to flare). Evidence for this mechanism in terms of de-activation has been found in comparisons of cognitive bias in chronic pain patients pre- and post-surgery (Edwards et al, 1995), and depressed patients compared with those who have recovered from depression (Bradley & Mathews, 1988; Dohr et al, 1989). Alternatively, it may be the mood status of the patient that determines schema activation (and hence, cognitive bias), as Pincus & Morley (2001) have proposed. They argue that the evidence to date implicates depression as the determining factor of cognitive bias in pain patients. That is, regardless of the presence or absence of symptoms, those patients who are depressed will demonstrate bias for illness- and/or pain-related stimuli.
Thus, due to their potential influence upon information processing and behaviour, a greater understanding of illness schemas in SLE is likely to contribute to our knowledge of the adaptive and maladaptive ways in which patients attempt to deal with this condition. Such an understanding is particularly important in SLE due to the documented levels of distress experienced by patients with this illness (Katz et al., 2001; Iverson, 1995). Furthermore, the cognitive bias pain literature would benefit by broadening its focus beyond that of arthritis and chronic pain.

The present investigation has two main aims: to investigate illness schemas in a previously neglected population of SLE patients, and to explore the nature of the schemas with regard to flare status and depression as described above. The present study aims to test two competing hypotheses related to cognitive biases. Firstly, if illness schemas are activated by the re-emergence of disease symptoms, then dichotomising SLE patients according to flare status should result in a difference between groups, such that patients in a flare will demonstrate cognitive bias for illness words (flare hypothesis). Alternatively, if illness schemas are invoked as part of a process through which ill individuals become depressed when living with illness or threat of illness, then dividing the patient groups according to depression status should result in a difference between the groups, where depressed patients demonstrate a bias in comparison to their non-depressed counterparts (depression hypothesis).

A clinical comparison group of RA patients is included in the present study. As RA is the patient population that has been most commonly tested for cognitive bias, and RA and SLE are both autoimmune diseases, such a comparison was thought to be valuable in this investigation. For the purposes of the depression hypothesis the depressed SLE patients and the depressed RA patients will be compared as a group (depressed ill) to the non-depressed patients (non-depressed
ill). A healthy control group is also included.

In accordance with self-schema research under the information processing paradigm, the current study measures illness schema via recall and endorsement of negative illness-related words relative to positive illness words. The methodology has its basis in the work of Pincus and colleagues (1995). Positive and negative depression-related words are included in order to ascertain the nature of depressive schema. Additional positive and negative control words are included to determine whether any affectively valenced bias is specifically related to illness-related words, or merely represents a bias in general (Pincus et al, 1995). In keeping with previous findings (Clemmey & Nicassio, 1997; Pincus et al, 1995), it is hypothesised that neither the patient groups (SLE/RA or depressed ill/non-depressed ill) nor the control group will exhibit a valence bias for the depression or control words.
2. METHOD

2a. Participants

The present study used a convenience sample. RA patients and approximately half of the SLE patients were recruited from the rheumatology department at Royal North Shore Hospital, Sydney. Eligible patients were informed of the study by their rheumatologist during their routine medical consultation, all of whom agreed to be contacted by the researcher for further information. Of these, four patients declined to proceed with participation.

The remaining half of the SLE patients were recruited via advertisement in the newsletter of the NSW Lupus Association. Of those who expressed interest, 5 patients decided not to participate. A letter confirming the diagnosis was sought from their treating rheumatologist. Patients over the age of 18 who fulfilled American College of Rheumatology (ACR) criteria for SLE or RA were eligible for inclusion in the study.

Sixteen RA patients and 45 SLE patients agreed to participate in the study, after which two SLE patients were excluded due to their doctor being unable to confirm the diagnosis. For some analyses the SLE patients were further divided according to disease status, as measured by the Systemic Lupus Erythematosus Disease Activity Index (SLEDAI). The SLEDAI has been shown to be a valid measure of clinicians’ global assessment of disease activity in SLE (Bombardier et al, 1992). Patients rated at 6 or higher were considered to be in a flare (Gladman, 1995).
Twenty-two control participants were recruited via local advertisement. Exclusion criteria were non-fluent English or a history of chronic illness. Analysis revealed that there were no differences between SLE, RA or controls with regard to male to female ratio (p=0.275). SLE and control subjects were not significantly different in age (F<1), but RA patients were significantly older than the other two groups (F(2,78)=8.26, p<0.01). This reflects the increasing prevalence of RA with increasing age. An age-matched sample was likely to have been an unusual sample and not representative of the population of patients with RA. Pain levels, functional impairment, depression and anxiety were assessed in all participants (please see section 2f for a full description).

2b. Procedure

Two measures of cognitive bias were employed; an endorsement task followed by a free recall task. These two tasks were separated by an interference task, PRISM (see Section III). Memory was then tested by the Rey Auditory Verbal Learning Test (RAVLT), after which participants completed a series of self-report measures. The assessment took a total of 90 minutes.

2c. Design

2c. i) Flare Hypothesis

A 4 x 3 x 2 x 2 design, with one between-group factor, illness (SLE flare, SLE non-flare, RA, healthy control): and three within-subject factors, word type (illness, depression, control), valence (positive and negative), and reference (self and other). The two dependent variables measuring self-schema were word
endorsement and word recall.

**2c. ii) Depression Hypothesis**

A 4 x 3 x 2 x 2 design, with one between-group factor, ill/depression (depressed ill, non-depressed ill, healthy control): and three within-subject factors, word type (illness, depression, control), valence (positive and negative), and reference (self and other). The two dependent variables measuring self-schema were word endorsement and word recall.

**2d. Cognitive Bias Measures**

**2d. i) Endorsement Task**

This task was based on the methodology used by Pincus and colleagues (1995) and was presented to subjects on a portable laptop P.C. computer. Subjects were exposed to 72 stimulus trials, preceded by 10 practice trials. Each trial consisted of an adjective and a question being presented on the screen. Of the 72 adjectives, 24 belonged to one of three content categories (illness, depression and control) of which 12 were positive and 12 were negative (see Table 2). For each adjective, subjects were asked in alternating order, "Describes you?" (self reference) or "Describes your best friend?" (other reference). The cue questions were followed by a gap of 500 milliseconds and the appearance of the target adjective. Subjects were required to respond by mouse-clicking on buttons on the screen marked 'Yes' or 'No', which were presented in a counter-balanced order from left to right. The response terminates the display, and the next cue question is presented 3.5 seconds later. The computer generated the random order for each subject such that no two
words from the same content category were presented in succession. The 72 stimulus trials were then repeated such that subjects were presented with the opposite reference question for each adjective. Response time to each trial was recorded by the computer. The dependent variable was the number of words endorsed (response of ‘Yes’) in each stimulus category.

Table 2: Stimuli used in the endorsement task

<table>
<thead>
<tr>
<th></th>
<th>Self Reference</th>
<th>Other Reference</th>
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<tbody>
<tr>
<td>Control -ve</td>
<td>Crude, discourteous, nosy, phoney, thoughtless, uncivil</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Disrespectful,immoral, obnoxious, rude, ungrateful, unprincipled</td>
<td></td>
</tr>
<tr>
<td>Control +ve</td>
<td>Congenial, polite, genuine, cooperative, scrupulous, tactful</td>
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<tr>
<td></td>
<td>Amiable, cordial, ethical, honest, mannered, nice</td>
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<tr>
<td>Depression -ve</td>
<td>Inefficient, inadequate, lazy, boring, guilty, withdrawn</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Ineffective, insignificant, lowly, shameful, uninspired, unlikeable</td>
<td></td>
</tr>
<tr>
<td>Depression +ve</td>
<td>Lovable, motivated, outgoing, valuable, worthy, potent</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Ambitious, eager, praiseworthy, enthusiastic, attractive, pleasant</td>
<td></td>
</tr>
<tr>
<td>Illness -ve</td>
<td>Hurting, vulnerable, agonised, suffering, ill, uncomfortable</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Aching, dependent, sore, tortured, disabled, stiff</td>
<td></td>
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<tr>
<td>Illness +ve</td>
<td>Healthy, well, self-sufficient, active, healing, flexible</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Strong, lively, assertive, athletic, wholesome, comfortable</td>
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</table>

2d. ii) Recall Task

In the recall task, subjects were given a blank piece of paper by the experimenter and asked, "Please write down as many of the words as you can remember from the computer task you completed a few minutes ago". Order and spelling were de-
emphasised. Subjects were given a maximum of five minutes to complete the task. The dependent variable was the proportion of words in each stimulus category that were recalled out of the total number of words recalled.

2e. Cognitive Measure

2e. i) Rey Auditory-Verbal Learning Test (RAVLT)

Due to the documented cognitive deficits in some SLE and RA patients (Denburg et al, 1997), and the fact that one of the primary dependent variables in the present study was recall, the Rey Auditory-Verbal Learning Test (RAVLT) was administered in order to control for learning and memory. The RAVLT has Australian norms (Geffen et al, 1990) and has been found to be a sensitive measure of cognitive involvement in patients with SLE (Kozora et al, 1996).

2f. Self-Report Measures

2f. i) Medical Outcomes Survey 36-Item Short Form Health Survey (SF-36)

Functional impairment was measured using the SF-36 (Ware & Sherbourne, 1992), a widely used questionnaire for assessing issues related to quality of life in SLE (Stoll et al, 2001). The SF-36 is designed to measure physical and psychological functioning, and has been widely used as a health outcome measure in clinical trials in rheumatic diseases (for a review, see Molenaar et al, 2000). When compared to two other health measures, the SF-36 was found to be the most responsive to changes in perceived general health in arthritis patients (Husted et al,
1998). In a study of 150 SLE patients, Stoll and colleagues (1997) reported that the SF-36 has good internal consistency (Cronbach’s alpha > 0.71) and good discriminatory validity. The Bodily Pain scale was used as a measure of pain. Whilst this scale is not a comprehensive assessment of pain levels, as pain is only one symptom of SLE and RA it was deemed satisfactory for the purposes of the present study. It was not feasible to independently assess each symptom of the two illnesses.

2f. ii) Hospital Anxiety and Depression Scale (HADS)

The Hospital Anxiety and Depression Scale (HADS) (Zigmond & Snaith, 1983) was used to assess depressive symptomatology for each subject. Research has demonstrated that depression is a significant factor in SLE (Klopchin, 2002; Da Costa et al, 1999), RA (Sharpe, 2001; Clemmey & Nicassio, 1997) and memory bias in self-schema (Pincus & Morley, 2001; Clemmey & Nicassio, 1997; Derry & Kuiper, 1981). The HADS was chosen for its minimal use of items based on somatic complaints, an important factor when sampling a population of chronically ill patients. In his comprehensive review of more than 200 published studies that have utilised the HADS, Herrmann (1997) reports good internal consistencies for both the anxiety and depression scales (Cronbach alphas between 0.8 and 0.93). Sensitivity and specificity of the scales are also good at 0.8 or higher.

2g. Statistical Analysis

Repeated measures ANOVAs were conducted on the endorsement data and the recall data. In the cases where the data was not normally distributed, variances
were examined for homogeneity. In each case the largest variance was less than four times the smallest and so statistical analysis proceeded.
3. Results

The average time since diagnosis was 10 years for SLE patients and 13.4 years for RA patients, where the former were judged on average as having a moderate degree of disease activity (mean SLEDAI=5.96). All RA and SLE patients were taking some form of medication to control the disease. Both SLE and RA patients rated their physical impairment as high (mean SF-36 Physical=37; 31 respectively), and their bodily pain as high (SF-36 Bodily Pain=41; 37 respectively). These scores are below average relative to SF-36 norms for RA patients in general, indicating that this sample of rheumatology patients falls at the more severe end. Consistent with prior research, average anxiety and depression levels were mild to moderate in both SLE and RA patients. Surprisingly, short-term memory in the two patient groups was not significantly below average (SLE RAVLT z-score= -0.046), (RA RAVLT z-score= -0.39). Healthy controls demonstrated significantly above average short-term memory (RAVLT z-score=1.06; p<0.001).

3a. Self-Report Measures

Endorsement of self-referential negative illness words was significantly correlated with SF-Physical, SF-Mental, Pain Rating, HADS Depression and HADS Anxiety (all p<0.001). Endorsement of positive illness words was also correlated with these measures (in the opposite direction) (all p<0.001). Recall of illness words was not significantly related to any of self-report measures, whilst RAVLT z-score was highly correlated with total recall (r = 0.54, p<0.000).

3b. Flare Hypothesis

The characteristics of the four groups after SLE patients were divided according to
flare status are shown in Table 3. The control group significantly differed from the other three groups on all variables, and significant differences between the patient groups are marked with $^b$.

**Table 3:** Means (standard deviations) of demographics, disease and mood characteristics across flare groups

<table>
<thead>
<tr>
<th></th>
<th>SLEnonflare</th>
<th>SLE flare</th>
<th>RA</th>
<th>Controls</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>N</strong></td>
<td>17</td>
<td>26</td>
<td>16</td>
<td>22</td>
</tr>
<tr>
<td><strong>Male: Female</strong></td>
<td>1:16</td>
<td>0:26</td>
<td>2:14</td>
<td>1:21</td>
</tr>
<tr>
<td><strong>Age$^b$</strong></td>
<td>50.8 (9.7)</td>
<td>40.2 (12.1)</td>
<td>58.7 (11.8)</td>
<td>44.0 (13.8)</td>
</tr>
<tr>
<td><strong>Marital Status (% married)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Years of Education</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Work Status (% working at least part-time)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>SLEDAI$^b$</strong></td>
<td>1.3 (1.8)</td>
<td>9.0 (4.2)</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Years since diagnosis$^b$</strong></td>
<td>14.1 (10.5)</td>
<td>7.4 (5.5)</td>
<td>13.4 (8.1)</td>
<td></td>
</tr>
<tr>
<td><strong>SF-36 Physical$^a$</strong></td>
<td>37.1 (10.2)</td>
<td>37.0 (13.3)</td>
<td>31.5 (7.5)</td>
<td>55.2 (5.4)</td>
</tr>
<tr>
<td><strong>SF-36 Mental$^{ab}$</strong></td>
<td>52.7 (7.6)</td>
<td>44.3 (10.5)</td>
<td>51.2 (9.6)</td>
<td>54.5 (6.9)</td>
</tr>
<tr>
<td><strong>Pain Rating$^b$</strong></td>
<td>44.5 (9.6)</td>
<td>39.6 (12.4)</td>
<td>36.3 (9.6)</td>
<td>54.3 (6.6)</td>
</tr>
<tr>
<td><strong>HADS Depression$^b$</strong></td>
<td>4.0 (2.5)</td>
<td>5.7 (3.5)</td>
<td>7.0 (3.8)</td>
<td>1.6 (1.8)</td>
</tr>
<tr>
<td><strong>HADS Anxiety$^b$</strong></td>
<td>5.4 (2.2)</td>
<td>8.8 (2.8)</td>
<td>9.0 (4.5)</td>
<td>5.0 (2.0)</td>
</tr>
<tr>
<td><strong>RAVLT Z-score</strong></td>
<td>0.27</td>
<td>-0.25</td>
<td>-0.39</td>
<td>1.06</td>
</tr>
</tbody>
</table>

SLEDAI = Systemic Lupus Erythematosus Disease Activity Index
HADS = Hospital Anxiety and Depression Scale
SF-36 = Short Form-36 Health Survey
RAVLT = Rey Auditory Verbal Learning Test

$^a$ Lower score indicates poorer functioning/more pain. General population mean=50 (s.d.=10)
(Ware & Kosinski, 1994)

$^b$p<0.05 comparing patient groups
As a result of these significant differences between patient groups, the use of covariates was closely considered. Whilst higher pain ratings and SLEDAI scores in symptomatic patients are predicted and valid disparities, differences in anxiety, depression, general mental health, years since diagnosis and age have the potential to confound the results of any group comparisons on the variables of interest. Anxiety, depression and SF-Mental Health scores were highly correlated with each other (r>0.512, p<0.000), as were age and time since diagnosis (r=0.37, p=0.004). Due to these high intercorrelations, entering all variables as covariates is statistically problematic. Therefore, depression and age were initially both entered as covariates, however, as age did not alter the outcome it was removed. Depression is included as a covariate in all analyses for the flare hypothesis.

3b. i) Endorsement

A repeated measures analysis of variance (ANOVA) was conducted on the number of words endorsed as descriptive. The four-way interaction between group, word type, valence and reference (4 x 3 x 2 x 2) was significant (F(3,76)=5.84, p=0.001). Two separate three-way repeated measure ANOVAs were conducted in each of the reference conditions, ‘self’ and ‘other’. The four groups did not differ from each other in the words they endorsed as describing their best friend (F(3,76)=0.29, p=0.83), however, the interaction was significant for the ‘self’ reference (F(3,76)=3.8, p=0.013). Accordingly, all further analyses were carried out on the endorsement scores in the condition of self-reference only. Within this condition there was no significant main effect for group (F(3,76)=0.83, p=0.48). A group by valence analysis in each word type domain revealed that groups differed
in their responses to illness words (F(3,76)=3.08, p=0.03), but not depression words (F(3,76)=1.6, p=0.21) or control words (F(3,76)=0.59, p=0.63). Post-hoc analyses using Bonferroni correction revealed that each of the patient groups endorsed more negative illness words and fewer positive illness words than healthy controls (SLEflare vs HC, p=0.006 (-ve), p=0.003 (+ve), SLEnonflare vs HC, p=0.000 (-ve), p=0.000 (+ve), RA vs HC, p=0.001 (-ve), p=0.000 (+ve)), but did not differ from each other (p >0.149). Means are presented in Table 4.

**Table 4**: Means (and standard deviation) of number of words self endorsed across flare groups

<table>
<thead>
<tr>
<th>Word Type</th>
<th>SLEnonflare</th>
<th>SLEflare</th>
<th>RA</th>
<th>Controls</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control neg.</td>
<td>0.53 (0.62)</td>
<td>0.50 (0.58)</td>
<td>0.63 (1.15)</td>
<td>0.36 (0.58)</td>
</tr>
<tr>
<td>Control pos.</td>
<td>11.24 (0.66)</td>
<td>11.04 (1.11)</td>
<td>10.94 (1.18)</td>
<td>11.41 (0.73)</td>
</tr>
<tr>
<td>Depression neg.</td>
<td>0.76 (1.09)</td>
<td>1.50 (1.73)</td>
<td>2.06 (2.69)</td>
<td>0.95 (1.56)</td>
</tr>
<tr>
<td>Depression pos.</td>
<td>9.53 (1.91)</td>
<td>9.19 (2.42)</td>
<td>7.63 (3.48)</td>
<td>10.41 (2.11)</td>
</tr>
<tr>
<td>Illness neg.</td>
<td>4.47 (2.65)</td>
<td>6.42 (3.02)</td>
<td>6.63 (3.65)</td>
<td>1.45 (1.3)</td>
</tr>
<tr>
<td>Illness pos.</td>
<td>7.29 (3.14)</td>
<td>7.19 (2.12)</td>
<td>7.00 (3.48)</td>
<td>10.27 (1.55)</td>
</tr>
</tbody>
</table>

**3b. ii) Recall**

As previous studies have found that words are more likely to be recalled if they have been endorsed in the encoding stage, correlations of endorsement and recall in each word category were calculated. No significant correlations were revealed in any of the word categories, thereby avoiding any concerns regarding the analysis of the recall data (r <0.16, p>0.17).
As there was such variation in the total number of words recalled (range 1-35), the dependent variable of choice was the proportion of total recall (Koutantji et al, 1999; Calfas et al, 1997; Edwards et al, 1992). The three-way interaction between group, word type and valence (4 x 3 x 2) was not significant ($F(3,76)= 0.91, p=0.44$). There was a main effect for word type ($F(1,76)=20.45, p<0.000$) and valence ($F(1,76)=8.04, p=0.006$), indicating that all groups recalled more illness words and more positive words overall. Means are presented in Table 5.

<table>
<thead>
<tr>
<th>Word Type</th>
<th>SLEnonflare</th>
<th>SLEflare</th>
<th>RA</th>
<th>Controls</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control neg.</td>
<td>0.12 (0.12)</td>
<td>0.09 (0.08)</td>
<td>0.16 (0.10)</td>
<td>0.14 (0.10)</td>
</tr>
<tr>
<td>Control pos.</td>
<td>0.12 (0.08)</td>
<td>0.17 (0.13)</td>
<td>0.16 (0.15)</td>
<td>0.19 (0.10)</td>
</tr>
<tr>
<td>Depression neg.</td>
<td>0.08 (0.07)</td>
<td>0.09 (0.08)</td>
<td>0.11 (0.16)</td>
<td>0.09 (0.09)</td>
</tr>
<tr>
<td>Depression pos.</td>
<td>0.21 (0.13)</td>
<td>0.20 (0.11)</td>
<td>0.23 (0.24)</td>
<td>0.18 (0.10)</td>
</tr>
<tr>
<td>Illness neg.</td>
<td>0.23 (0.30)</td>
<td>0.21 (0.10)</td>
<td>0.19 (0.13)</td>
<td>0.22 (0.10)</td>
</tr>
<tr>
<td>Illness pos.</td>
<td>0.25 (0.14)</td>
<td>0.24 (0.10)</td>
<td>0.20 (0.11)</td>
<td>0.20 (0.10)</td>
</tr>
</tbody>
</table>

### 4. Depression Hypothesis

An insufficient number of SLE or RA patients scored above the cut-off point for depression to analyse the data separately for each illness group. Therefore, for the purposes of the depression hypothesis, all SLE and RA patients were combined and then re-divided according to depression status. Those with a score of 8 or higher on the HADS were classed as depressed (Zigmond & Snaith, 1983). None
of the healthy control group were depressed and therefore that group remained the same. The characteristics of the three resultant groups are shown in Table 6.

**Table 6:** Means (standard deviations) of disease and mood characteristics across depression groups

<table>
<thead>
<tr>
<th></th>
<th>Ill-depressed</th>
<th>Ill-non-depressed</th>
<th>Controls</th>
</tr>
</thead>
<tbody>
<tr>
<td>N</td>
<td>18</td>
<td>41</td>
<td>22</td>
</tr>
<tr>
<td>Male: Female</td>
<td>1:17</td>
<td>2:39</td>
<td>1:21</td>
</tr>
<tr>
<td>Age</td>
<td>47.9 (15.4)</td>
<td>48.5 (13.0)</td>
<td>44.0 (13.8)</td>
</tr>
<tr>
<td>SLEDAI&lt;sup&gt;b&lt;/sup&gt;</td>
<td>8.5 (4.3)</td>
<td>6.1 (5.8)</td>
<td></td>
</tr>
<tr>
<td>Years since diagnosis</td>
<td>9.0 (6.8)</td>
<td>11.8 (8.8)</td>
<td></td>
</tr>
<tr>
<td>SF-36 Physical&lt;sup&gt;ªb&lt;/sup&gt;</td>
<td>30.5 (10.7)</td>
<td>37.7 (10.8)</td>
<td>55.2 (5.4)</td>
</tr>
<tr>
<td>SF-36 Mental&lt;sup&gt;ªb&lt;/sup&gt;</td>
<td>39.8 (10.0)</td>
<td>52.4 (7.5)</td>
<td>54.5 (6.9)</td>
</tr>
<tr>
<td>Pain Rating&lt;sup&gt;ªb&lt;/sup&gt;</td>
<td>34.3 (12.6)</td>
<td>42.6 (9.6)</td>
<td>54.3 (6.6)</td>
</tr>
<tr>
<td>HADS Depression&lt;sup&gt;b&lt;/sup&gt;</td>
<td>9.7 (2.2)</td>
<td>3.8 (2.1)</td>
<td>1.6 (1.8)</td>
</tr>
<tr>
<td>HADS Anxiety&lt;sup&gt;b&lt;/sup&gt;</td>
<td>10.7 (3.9)</td>
<td>6.7 (2.7)</td>
<td>5.0 (2.0)</td>
</tr>
<tr>
<td>RAVLT Z-score&lt;sup&gt;c&lt;/sup&gt;</td>
<td>-0.5 (1.4)</td>
<td>0.0 (1.3)</td>
<td>1.1 (1.0)</td>
</tr>
</tbody>
</table>

SLEDAI = Systemic Lupus Erythematosus Disease Activity Index
HADS = Hospital Anxiety and Depression Scale
SF-36 = Short Form-36 Health Survey
RAVLT = Rey Auditory Verbal Learning Test
<sup>ª</sup>Lower score indicates poorer functioning/more pain. General population mean=50 (s.d.=10) (Ware & Kosinski, 1994)
<sup>b</sup>Patient groups differed from each other and controls, p<.01
<sup>c</sup>Patient groups did not differ from each other but differed from controls, p<0.01

The statistical analyses conducted for the flare hypothesis were replicated for the depression hypothesis for word endorsement and word recall, except SF-36 Physical scores were entered as a covariate in the endorsement analyses. This was
deemed necessary as a result of the differences between the patient groups on SF-36 Physical, and the SF-36 Physical’s correlation with endorsement of negative illness words (r=-0.717, p<0.000).

4a. Endorsement

The four-way ANOVA between group, word type, valence and reference (3 x 3 x 2 x 2) with SF-36 Physical as a covariate was significant (F(2,77)=3.813, p=0.026). As before, the groups only differed from each other in the words they endorsed as being self-descriptive (F(2,77)=5.618, p=0.005), therefore all further analyses were carried out on the self-reference endorsement scores only. There was no significant main effect for group (F(2,77)=0.95, p=0.39), but the interaction effect of group by valence (F(2,77)=11.155, p=0.00) was significant. Means are presented in Table 7.

Table 7: Means (standard deviation) of number of words self endorsed across depression groups

<table>
<thead>
<tr>
<th>Word Type</th>
<th>Ill-depressed</th>
<th>Ill-non-depressed</th>
<th>Controls</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control neg.</td>
<td>0.94 (1.06)</td>
<td>0.37 (0.54)</td>
<td>0.36 (0.58)</td>
</tr>
<tr>
<td>Control pos.</td>
<td>10.78 (1.17)</td>
<td>11.2 (0.93)</td>
<td>11.41 (0.73)</td>
</tr>
<tr>
<td>Depression neg.</td>
<td>2.72 (2.47)</td>
<td>0.88 (1.33)</td>
<td>0.95 (1.56)</td>
</tr>
<tr>
<td>Depression pos.</td>
<td>7.94 (3.1)</td>
<td>9.27 (2.43)</td>
<td>10.41 (2.11)</td>
</tr>
<tr>
<td>Illness neg.</td>
<td>7.89 (2.89)</td>
<td>5.05 (2.95)</td>
<td>1.45 (1.30)</td>
</tr>
<tr>
<td>Illness pos.</td>
<td>5.17 (2.20)</td>
<td>8.05 (2.58)</td>
<td>10.27 (1.55)</td>
</tr>
</tbody>
</table>

Post-hoc analyses, using Bonferroni adjustment, revealed that ill-depressed patients endorsed more negative illness words than ill-non-depressed patients (p=0.000), who endorsed more than controls (p=0.000). The reverse pattern existed for the positive illness words (ill-depressed vs HC, p=0.000, ill-depressed vs ill-
nondepressed, p=0.001). Ill-depressed patients endorsed significantly fewer positive depression words than control subjects (p=0.008) although differences between ill depressed and non-depressed subjects failed to reach significance (p=0.2). Ill-depressed patients endorsed more negative depression words (vs HC, p=0.005; vs. ill-nondepressed, p=0.001) and more negative control words than either of the other groups (vs HC, p=0.031; vs. ill-nondepressed, p=0.013), who did not differ from each other (p=1). There were no significant differences between any of the groups in terms of positive control words (p>0.113).

4b. Recall

There was a main effect for word type (F(1,78)=26.6, p<0.000) and valence (F(1,78)=20.3, p<0.000), indicating that all groups recalled more illness words and positive words overall. The three-way interaction between group, word type and valence (3 x 3 x 2) was also significant (F(2,78)=3.13, p<0.05). However, post-hoc analyses revealed that the only difference between groups was due to the ill-depressed group recalling fewer positive control words than the ill-non-depressed group (p = 0.019). Means are presented in Table 8.
Table 8: Means (and standard deviation) of proportion of words recalled across depression groups

<table>
<thead>
<tr>
<th>Word Type</th>
<th>Ill-depressed</th>
<th>Ill-non-depressed</th>
<th>Controls</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control neg.</td>
<td>0.093 (0.11)</td>
<td>0.115 (0.09)</td>
<td>0.138 (0.10)</td>
</tr>
<tr>
<td>Control pos.</td>
<td>0.212 (0.17)</td>
<td>0.124 (0.08)</td>
<td>0.188 (0.09)</td>
</tr>
<tr>
<td>Depression neg.</td>
<td>0.099 (0.16)</td>
<td>0.086 (0.07)</td>
<td>0.086 (0.06)</td>
</tr>
<tr>
<td>Depression pos.</td>
<td>0.166 (0.14)</td>
<td>0.231 (0.16)</td>
<td>0.176 (0.10)</td>
</tr>
<tr>
<td>Illness neg.</td>
<td>0.223 (0.11)</td>
<td>0.203 (0.12)</td>
<td>0.216 (0.10)</td>
</tr>
<tr>
<td>Illness pos.</td>
<td>0.207 (0.12)</td>
<td>0.241 (0.11)</td>
<td>0.198 (0.10)</td>
</tr>
</tbody>
</table>

5. Post-Hoc Analyses

In their recently-proposed model of cognitive bias in pain, Pincus & Morley (2001) were the first to suggest that pain schemas and illness schemas could be conceptualised separately. They propose that active pain schemas are reflected in cognitive bias for sensory pain stimuli (for example, 'throbbing', 'aching'), and that active illness schemas are reflected in cognitive bias for non-sensory pain stimuli, or pain affective and disability words (for example, 'dependent', 'suffering'). We examined the stimuli used in the current study, and found that ten of the twelve negative illness words could be divided into either sensory pain words or disability words. We performed post-hoc analyses on the self-referential endorsement and recall of these two types of words, firstly dividing the patients according to the flare hypothesis, and then according to the depression hypothesis. Covariates remained as before.

ANOVAAs were conducted for sensory and disability words with group as the between subjects factor. The dependent variable used was the proportion of the
total number of negative illness words recalled. Analyses revealed a difference in recall patterns only when participants were divided according to the depression hypothesis. For endorsement, there was main effect for group (F(2,78)=37.35, p=0.000) and a significant interaction effect (F(2,78)=3.62, p=0.03). Post-hoc comparisons, using Bonferroni adjustments, revealed that depressed patients endorsed more disability words than non-depressed patients (p=0.000), who endorsed more than controls (p=0.002). There was no significant difference between the two ill groups as to the number of sensory words endorsed (p=0.109). Similar results were found for the recall data. The main effect for group was not significant (F(2,78)=1.368, p=0.246), however, there was a significant interaction effect (F(2,78)=3.95, p=0.023). Ill-depressed patients recalled significantly more disability illness words relative to the non-depressed patients (p=0.01) and the control subjects (p=0.041), who did not differ from one another (p=1). There were no differences between the three groups for recall of sensory information (p>0.29) (see Figure 3).

Figure 3: Mean sensory and disability words recalled as a proportion of total negative illness words recalled, across depression groups
4. Discussion

The aim of the present study was to determine whether the presence of current symptomatology (the flare hypothesis) or depression (the depression hypothesis) better accounted for cognitive biases in chronically ill patients. The flare hypothesis did not receive support from either the recall data or the endorsement data. This suggests that symptomatology does not affect endorsement or recall of negative illness-related information. On the other hand, the depression hypothesis was supported by the endorsement data but not the recall data. That is, when compared to non-depressed patients and healthy controls, depressed SLE/RA patients endorsed more negative illness words and fewer positive illness words, but did not differ in their recall of such words. However, when in post-hoc analyses words were divided as to whether they were related to pain or illness schema, differences in recall patterns did emerge that support the depression hypothesis. That is, depressed patients recalled and endorsed a significantly greater proportion of illness words related to disability than either of the other groups, whereas no differences were observed for sensory words.

These results differ from those reported by Pincus and colleagues (1995) in their study of RA patients. Differences are unlikely to be due to methodological considerations, as the methodology for the present study was based on their work. It is possible that the discrepancy is due to inherent differences between SLE and RA patients. Firstly, SLE patients have been shown to have poorer cognitive functioning (and thus, memory) than the general population and poorer learning ability than RA patients (Kozora et al, 1996). Hence, the recall bias may have been less sensitive due to an overall floor effect. This explanation seems unlikely though, as there were no differences between the RA and SLE patients on the RAVLT. Yet as a full cognitive assessment was not conducted, this explanation
cannot be excluded. Secondly, it has been repeatedly proposed in the literature that depression in SLE is part of a neuropsychiatric presentation of the illness. It may be that for those SLE patients where the depressive symptomatology reflects an underlying disease process, self-schema has little relevance or relationship to the depression. For this reason, general group effects in SLE may be less reliable than in samples of RA patients. Again, the lack of difference between groups does not support this interpretation, but in the absence of any effort to determine the source of depressed mood, this interpretation cannot be excluded.

In examining the comparison between our results and those of Pincus and colleagues (1995), it is noteworthy that of the eight published studies investigating pain-related recall biases in chronic pain/chronic illness, the majority have not found biases in a heterogenous group of patients towards heterogenous pain stimuli (Pincus & Morley, 2001). Rather, two studies found that only depressed pain patients demonstrated a recall bias for pain-related words (Clemmey & Nicassio, 1997; Pincus et al, 1995). Other studies have demonstrated that non-depressed patients show bias towards sensory words in preference to illness or disability words (Wells et al, 2003; Koutantji et al.; 1999; Edwards et al., 1992; Dehghani et al., submitted). Another found that depressed pain patients show the opposite bias to this mainstream tendency, demonstrating cognitive bias for affective, disability or illness-related words (Edwards et al., 1992). It is this latter effect that was observed in the present study.

Although in the present study patients as a whole recalled more illness words than words in other categories, this was also true of control participants. The presence of a bias towards pain-related information has been observed in other research, for example, a recent study by Wells and colleagues (2003). One explanation that they gave was the make-up of their control group, which was comprised of hospital
staff (who were exposed frequently to pain-related words and hence, this effect may be attributable to a frequency effect) or those attending for immunisations (whose pain schema may have been activated by the immunisation process). This explanation fails to account for the bias in the present study, as the control participants were university employees and students. They are therefore unlikely to have biases not evident in the general public as a result of their work or present circumstances. As has been suggested previously, a sensory bias may be adaptive and have evolutionary advantage, and it may not be specific to patients with chronic painful illnesses (Wells, et al, 2003; Koutantji et al., 1999).

Consistent with the predictions of Pincus and Morley’s (2001) schema enmeshment model, we found that the level of depression accounted for the degree to which patients recalled disability-related words (illness self-schema) in preference to sensory pain-related words (pain self-schema). That is, depressed patients demonstrated a recall bias for disability words at the expense of sensory pain words, whereas the non-depressed patients and healthy controls recalled the two types of words equally. However, no differences were observed between the two patient groups on depression words. This again supports the view that depression in chronic illness is related to a different set of cognitive processes to those observed in depressed but healthy patients (Pincus & Morley, 2001). That is, patients whose illness schema are activated by their experience of chronic pain, are those who are at risk for poor adjustment to illness and hence depression.

There are some limitations of the present study that must be borne in mind in interpreting the results. Firstly, convenience samples were used and therefore it is unclear the degree to which patients are representative of the population of patients with these illnesses. However, the levels of disability, illness duration, depression and age are similar to those reported in other studies of Lupus patients (Boomsma
et al, 2002; Buchi et al, 2000) and RA patients (Buchi et al, 2002; Gilboe et al, 1998). Further, all groups were drawn through convenience sampling and are therefore likely to be subject to the same biases, making this an unlikely reason for the lack of differences. The samples were relatively small and although they were large enough to detect large effect sizes, smaller effect sizes may have been obscured, leading to Type II errors. This is particularly true when comparing flare and non-flare or depressed and non-depressed groups. Nonetheless, the sample sizes here are comparable to others reported in the literature (range n = 12 – 40).

A further limitation worthy of note is the general severity of symptoms in the SLE group. Whilst they could be divided into flare and non-flare on the basis of SLEDAI scores, the mean SF-36 Physical score of each of the two groups reflected a relatively high level of symptomatology. Thus, it is unlikely that the ‘non-flare’ SLE group were in fact truly asymptomatic. This may have decreased the differences on the dependent variables between the two groups. Nonetheless, if symptomatology were the primary mechanism determining cognitive bias, one would expect to see at least a trend toward differences, which was not observed. In addition, as mentioned earlier the Bodily Pain scale of the SF-36 is not a comprehensive assessment of pain levels. Ideally the present study would have incorporated such an assessment. Finally, the effect of depression on responses to disability versus sensory words was based on post-hoc analyses. For these reasons these results should be interpreted with caution.

These limitations aside, the present study is the first to extend the findings of the cognitive bias literature to an illness where patients differ markedly in their current symptoms. The results indicate that the level of symptomatology does not predict cognitive biases to illness-related words. The level of depression, on the other hand, was related to recall biases towards disability related words, but not sensory
pain words. These results suggest that an illness schema activated in some ill individuals may mediate the development of depression in individuals with chronic illnesses, such as SLE and RA. These results have potentially important clinical implications. The findings suggest that cognitive interventions that focus on changing unhelpful self-schema related to illness are likely to be helpful in reducing levels of depression amongst those ill individuals who are concurrently depressed.
SECTION III:

PRISM: ENMESHMENT OF ILLNESS & SELF SCHEMA

The following section is based on a manuscript prepared for publication:

1. **INTRODUCTION**

Quality of life in chronic illness has become a widely researched area over the last two decades. In both the medical and psychological literature there has been a gradual shift away from relying only on laboratory and clinical indicators of disease, and a move towards measures that incorporate the patient’s perspective of their illness (Wood-Dauphinee, 1999). Such a shift has been necessitated by the growing body of evidence indicating that quality of life and disease variables often have little or no association with one another (Stoll et al, 2001; Gladman et al, 1996). It cannot be assumed that the patient with severe illness has a poorer quality of life than the patient with milder illness. Numerous other psychological and emotional factors mediate patients’ experience of chronic illness, including patient-perceived impact of the disease (Edworthy et al, 1998; Burckhardt et al, 1993), depression (Sharpe et al, 2001), learned helplessness (Thumboo et al, 2000), and coping strategies (Da Costa et al, 1999; Dobkin et al, 1999).

Buchi and colleagues (1998) have proposed that a key element of health status is the importance that an individual attaches to his/her illness in everyday life. In an attempt to measure this concept they developed the Pictorial Representation of Illness and Self Measure (PRISM). This novel graphic task requires the patient to position a disc representing their ‘illness’ in proximity to a disc representing their ‘self’. The distance between the two disc centres is the main quantitative measure derived from the task.
Data from a validation study involving over 700 patients with a variety of physical illnesses (Buchi et al, 2002) indicated that the PRISM task is significantly correlated with a number of measures of physical and psychological function. However, the authors argue that PRISM measures a relatively specific notion that they term **burden of suffering due to illness**. According to their work, a smaller distance between the illness and self indicates a greater burden of suffering for the individual. To a large extent this finding was derived from a qualitative component of their study, in which patients were asked to describe their reasons for their placement of the illness disk. A qualitative methodology of this nature has the advantage of allowing investigators to understand the experience of illness for individuals and how this experience influences their responses. Such a methodology allows the generation of hypotheses that can be tested in subsequent larger quantitative studies. However, there are no reports of a quantitative measure of burden of suffering due to illness in the literature. Without such a quantitative measure to function as a ‘gold standard’, establishing the validity of PRISM as a measure of suffering remains difficult. Therefore, it is unclear at this stage as to whether PRISM represents burden of suffering as the authors suggest, or whether it is primarily related to other relevant concepts. One concept that is potentially relevant to this area is illness schema.

Pincus and Morley (2001) have recently reviewed the literature on cognitive bias in chronic pain. They have proposed that the bias toward negative illness-related words observed in this population is a result of the enmeshment of three schemas representing pain, illness, and the self. They suggest that individuals hold sets of beliefs (or schemas) about themselves, their illness and their pain. According to
Pincus and Morley’s model, if elements from schemas related to illness and pain are frequently activated at the same time as elements from one’s self-schema, the content of the three schemas can become incorporated into one another, or enmeshed. Thus, the individual’s view of himself or herself is disrupted. Enmeshment of self and illness schemas is proposed to have an important role in the development of depression in the presence of chronic pain or illness (Pincus & Morley, 2001; Clemmey & Nicassio, 1997).

Studies of cognitive bias represent a significant way in which researchers have been able to gain access to levels of cognition and cognitive processes that are generally inaccessible via standard interviews and questionnaires (Pincus & Morley, 2001). Cognitive bias (for example, attentional bias or memory bias) is hypothesised to indicate the presence of one or more schemas. Schemas represent the way that experiential knowledge has been articulated and differentiated in memory, and function to organise the processing, retention and retrieval of information (Derry & Kuiper, 1981).

PRISM appears to be conceptually related to the schema enmeshment described by Pincus and Morley (2001), in that the task essentially requires patients to nominate how much their illness defines, or intrudes upon, their sense of self. In addition, given the relationship between schema enmeshment and depression, a relationship between enmeshment and PRISM (demonstrated to be related to burden of suffering) is likely.

Thus, the present study seeks to investigate the relationship between the PRISM
task, and self and illness schemas, via measures of cognitive bias. It is hypothesised that the degree to which patients demonstrate a recall bias toward negative illness-related stimuli will be inversely related to the distance between the self and illness discs on the PRISM task. Systemic lupus erythematosus (SLE) was the chosen chronic illness for this study, as patients with this disease were included in the original validation study of PRISM (Buchi et al, 2002). SLE is a chronic, autoimmune disease in which multiple organs become inflamed and sometimes damaged. Symptoms tend to follow an unpredictable course with alternating exacerbations (flares) and remissions. These symptoms include extreme fatigue, painful and/or swollen joints, skin rash and fever (McCracken et al, 1995).
2. METHOD

2a. Sample

Participants were a convenience sample recruited from a rheumatology department at one Sydney hospital, and via advertisement in the regional Lupus Association’s newsletter. Patients over the age of 18 who fulfilled American College of Rheumatology (ACR) criteria for SLE according to their Rheumatologist were eligible for inclusion in the study. For participants who volunteered, a letter confirming their diagnosis was sought from the treating Rheumatologist. Forty-five patients volunteered for the study and two were excluded because their Rheumatologist was unable to confirm an SLE diagnosis.

2b. Measures

2b. i) PRISM Task

The method of administration of this task was identical to that outlined in the original publication of PRISM (Buchi et al, 1998), except that the present study used a computerised version. Subjects viewed a white computer screen with a fixed yellow disc (6cm in diameter) and two moveable discs, one red and one blue (4cm in diameter). Each subject was asked to imagine that the whole screen represents their life as it is currently, that the yellow disc represents their self, and that the red disc represents their illness. Each subject was then asked, "Where would you place the illness (red) disc in your life at the moment?". The subject was required to move the red disc by using a 'click and drag' movement with the computer mouse. The blue disk was available to illustrate the task with respect to

3 Identical sample as used in the previous study that is described in Section II.
work or family if the subject had difficulty understanding the task. The outcome measure was the distance in centimetres between the centres of the self and illness discs, termed Self Illness Separation (SIS). All participants were able to understand and complete the task.

2b. ii) Endorsement Task

This task was based on the methodology used by Pincus and colleagues (1995) and was presented to subjects on a portable laptop P.C. computer. Subjects were exposed to 72 experimental trials twice, preceded by 10 practice trials. Each trial consisted of an adjective and a question being presented on the screen. Of the 72 adjectives, 24 belonged to one of three content categories (illness, depression and control) of which 12 were positive and 12 were negative. For each adjective, subjects were asked in alternating order, "Describes you?" or "Describes your best friend?". Subjects were required to respond by mouse-clicking on buttons on the screen marked 'Yes' or 'No', which were presented in a counter-balanced order from left to right. The computer generated the random order for each subject such that no two words from the same content category were presented in succession. For the purposes of this study, only data pertaining to self-referent ("Describes you?") illness words are reported. A measure of endorsement bias was calculated by subtracting the number of negative illness words (e.g. disabled, aching) from the number of positive illness words (e.g. healthy, active) that were endorsed.

2b. iii) Recall Task

In the recall task, subjects were given a blank piece of paper by the experimenter and asked, "Please write down as many of the words as you can remember from
the computer task you completed a few minutes ago”. Subjects were given a maximum of five minutes to complete the task. A measure of recall bias was calculated by subtracting the number of negative illness words from the number of positive illness words that were recalled (such that the lower the score the greater bias for negative words). It is this recall bias measure that is purported to assess schema (Pincus & Morley, 2001).

2b. iv) Disease Activity

Disease activity was assessed using the Systemic Lupus Erythematosus Disease Activity Index (SLEDAI), a valid measure of clinician’s global assessment of disease activity in SLE (Bombardier et al, 1992). The SLEDAI was completed by the treating Rheumatologist.

2b. v) Impairment and Handicap

Functional impairment was measured using the SF-36 (Ware & Sherbourne, 1992), a widely used questionnaire for assessing issues related to quality of life in SLE (Stoll et al, 2001).

2b. vi) Depression

The Hospital Anxiety and Depression Scale (HADS) (Zigmond & Snaith, 1983) was used to assess depressive symptomatology for each subject. Research has demonstrated that depression is a significant factor in both SLE and memory bias in self-schema (Da Costa et al, 1999; Clemmey & Nicassio, 1997; Edwards et al, 1992; Bradley & Mathews, 1988; Derry & Kuiper, 1981). The HADS was chosen for its minimal use of items based on somatic complaints, an important factor
when sampling a population of chronically ill patients.

2b. vii) Illness Beliefs

Subjects’ attributions and beliefs about SLE were assessed using the Illness Perception Questionnaire (Weinman et al, 1996). This measure assesses beliefs relating to control, consequences and causes of the specified illness.

2b. viii) Cognitive

Due to the documented cognitive deficits in some SLE patients (Denburg et al, 1997), and the fact that one of the primary dependent variables in the present study was recall, the Rey Auditory-Verbal Learning Test (RAVLT) was administered in order to control for learning and memory. The RAVLT has Australian norms (Geffen et al, 1990) and has been found to be a sensitive measure of cognitive involvement in patients with SLE (Kozora et al, 1996).

2c. Statistical Analysis

Bivariate correlations with SIS were calculated using Pearson’s correlation coefficient. A multiple regression analysis was then conducted with a subset of the variables that were significantly associated with SIS to investigate independent predictors of SIS. Although the risk of Type I errors increases with this number of correlations, as this analysis is primarily exploratory it is not necessary for it to be overly conservative.
3. RESULTS

Of the 43 participants in the sample, 42 were female (98%) and the mean age was 44 years (range 22-70). Duration of illness was measured from the time of first diagnosis, and was on average a period of 10 years (range 1-42). Table 9 outlines characteristics of the sample. Aside from the female to male ratio in which men are under-represented, the characteristics of this sample are comparable to SLE outpatients in other studies (Buchi et al, 2000).

Table 9: Characteristics of the study sample (n = 43)

<p>| | | |</p>
<table>
<thead>
<tr>
<th></th>
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</thead>
<tbody>
<tr>
<td>Age</td>
<td>44</td>
<td>(range 22-70)</td>
</tr>
<tr>
<td>Female: Male ratio</td>
<td>42:1</td>
<td></td>
</tr>
<tr>
<td>Duration of Illness (years)</td>
<td>10</td>
<td>(range 1-42)</td>
</tr>
<tr>
<td>Disease Activity (SLEDAI)</td>
<td>6.8</td>
<td>(range 0-16)</td>
</tr>
<tr>
<td>SF-36 Physical Component Scale</td>
<td>37.0</td>
<td>(12.03)</td>
</tr>
<tr>
<td>SF-36 Mental Component Scale</td>
<td>47.8</td>
<td>(10.2)</td>
</tr>
<tr>
<td>HADS; Depression subscale</td>
<td>5.05</td>
<td>(3.24)</td>
</tr>
<tr>
<td>HADS; Anxiety subscale</td>
<td>7.4</td>
<td>(3.1)</td>
</tr>
<tr>
<td>RAVLT-Total score</td>
<td>51.4</td>
<td>(7.9)</td>
</tr>
<tr>
<td>IPQ Consequences</td>
<td>3.50</td>
<td>(0.7)</td>
</tr>
<tr>
<td>IPQ Cure Control</td>
<td>3.48</td>
<td>(0.5)</td>
</tr>
<tr>
<td>IPQ Timeline</td>
<td>4.08</td>
<td>(0.9)</td>
</tr>
<tr>
<td>IPQ Identity</td>
<td>1.38</td>
<td>(0.4)</td>
</tr>
</tbody>
</table>

* All values are means with standard deviations in brackets, unless stated otherwise.
SLEDAI = Systemic Lupus Erythematosus Disease Activity Index
SF-36 = Short Form 36 of Medical Outcomes Study
HADS = Hospital Anxiety and Depression Scale
RAVLT = Rey Auditory Verbal Learning Test
IPQ = Illness Perception Questionnaire
The mean SIS was 6 cm with a standard deviation of 5 cm, and ranged from 0 cm to 21.4 cm. These data were transformed to make them comparable with the available range on the original manual version of PRISM (that is, 0-27cm). Our data are equivalent with a mean of 7.5 cm, a standard deviation of 7.1 cm, and a range of 0-26.9 cm. SIS showed significant correlations with both endorsement bias ($r = 0.34$, $p < 0.05$) and recall bias of illness words ($r = 0.37$, $p < 0.05$), and IPQ Consequences subscale ($r = -0.43$, $p < 0.01$) (Table 10). The correlation between SIS and SF-36 physical component scale approached significance ($r = 0.29$, $p < 0.057$).

**Table 10: Correlations with Self Illness Separation (SIS)**

<table>
<thead>
<tr>
<th>Correlation</th>
<th>$r$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Recall bias of illness words</td>
<td>0.37*</td>
</tr>
<tr>
<td>Endorsement bias of illness words</td>
<td>0.34*</td>
</tr>
<tr>
<td>Age</td>
<td>0.00</td>
</tr>
<tr>
<td>Duration of illness</td>
<td>0.06</td>
</tr>
<tr>
<td>Disease Activity (SLEDAI)</td>
<td>—</td>
</tr>
<tr>
<td>SF-36 Mental Component Scale</td>
<td>0.05</td>
</tr>
<tr>
<td>SF-36 Physical Component Scale</td>
<td>0.29</td>
</tr>
<tr>
<td>Depression (HADS subscale)</td>
<td>—</td>
</tr>
<tr>
<td>Anxiety (HADS subscale)</td>
<td>—</td>
</tr>
<tr>
<td>IPQ Consequences</td>
<td>—</td>
</tr>
<tr>
<td>IPQ Identity</td>
<td>0.07</td>
</tr>
<tr>
<td>IPQ Timeline</td>
<td>—</td>
</tr>
<tr>
<td>IPQ Cure control</td>
<td>—</td>
</tr>
<tr>
<td>Memory and Learning (RAVLT)</td>
<td>0.01</td>
</tr>
</tbody>
</table>

* $p < 0.05$; ** $p < 0.01$  
Abbreviations as in Table 9
Variables were chosen for the multiple regression equation on the basis of their correlation with SIS (endorsement bias, recall bias, IPQ Consequences scale) or their theoretical interest (SF-36 PCS, HADS-Depression Scale). The multiple regression equation significantly predicted the SIS score derived from PRISM ($F(5,37) = 3.743, p < .01$). However, of those variables entered into the multiple regression equation, only recall bias made an independent contribution to SIS ($t(5,37) = 2.522, p < 0.05$) (Table 11).

Table 11: Multiple regression analysis of Self Illness Separation (SIS)

<table>
<thead>
<tr>
<th></th>
<th>Beta</th>
<th>T</th>
<th>Significance</th>
</tr>
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<tbody>
<tr>
<td>Recall bias</td>
<td>0.367</td>
<td>2.522</td>
<td>0.016</td>
</tr>
<tr>
<td>Endorsement bias</td>
<td>0.319</td>
<td>1.640</td>
<td>0.109</td>
</tr>
<tr>
<td>HADS-Depression</td>
<td>0.026</td>
<td>0.147</td>
<td>0.884</td>
</tr>
<tr>
<td>SF-36 Physical</td>
<td>-0.062</td>
<td>-0.345</td>
<td>0.732</td>
</tr>
<tr>
<td>IPQ Consequences</td>
<td>-0.270</td>
<td>-1.762</td>
<td>0.086</td>
</tr>
<tr>
<td>Adjusted r square</td>
<td>25%</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
### 4. Discussion

The results confirm the hypothesised relationship between the PRISM task and cognitive bias. The closer that SLE patients placed the ‘illness’ disk to the ‘self’ disk, the more negative illness words they recalled relative to positive illness words. Recall biases of this nature have been widely used to indicate the presence of schema (Pincus & Morley, 2001).

The findings of this study are generally consistent with those of the SLE component of the PRISM validation study (Buchi et al, 2002). SIS was not correlated with disease activity, depression or SF-36 MCS in either study, whilst both studies found at least a near-significant correlation between SIS and SF-PCS. Three themes were identified in the qualitative validation study as contributing to an individual’s placement of the illness disk: intrusiveness, controllability, and interference with salient aspects of everyday living. This is consistent with the finding in the present study of a significant correlation of SIS with the IPQ Consequences subscale. This subscale addresses the extent to which an individual perceives that their illness has had far-reaching consequences upon their life. This concept is clearly related to the three features listed above.

Buchi and colleagues’ (2002) interpretation of their findings leads them to the conclusion that PRISM is a measure of burden of suffering. Whilst their results are indeed consistent with what one might expect from a measure of suffering, we propose that this interpretation fails to encompass the full extent of the nature of PRISM.
Based on our findings, we propose that the PRISM task is assessing the enmeshment of an individual’s self and illness schemas, as suggested by Pincus and Morley’s model (2001). Pincus and Morley argue that in some patients with chronic pain and/or illness, their view of themselves becomes affected by their beliefs about the illness and pain. The more that their illness beliefs influence their self-schema, the more that their self and illness schema are said to become enmeshed. Pincus and Morley (2001) propose that the enmeshment of self and illness schemas is important in the disturbance to mood that commonly occurs in chronically ill patients. They suggest that cognitive bias for negative illness related information found in chronic pain patients measures the degree of schema enmeshment. The enmeshment described by Pincus and Morley appears to be similar to a notion outlined in the self-regulation model of illness (Nerenz & Leventhal, 1983). This model suggests that in some patients “the self is the disease, and the disease is the self” (p28).

Buchi and colleagues (2002) argue that PRISM embodies a number of concepts that are central to suffering, namely Cassell’s notion of a threat to the individual self (Cassell, 1982), and a loss of meaning, purpose and autonomy. We suggest that such concepts are central to the enmeshment of illness and self schemas, as a result of which suffering ensues. Their comment that “resolution of suffering is said to depend on reformulation of the self” (Buchi et al, 2002; p16), is a near-perfect description of the disentangling of the self and illness schemas that would be predicted to lead to a greater sense of autonomy and control.

Schema enmeshment and suffering need not be mutually exclusive in terms of
what PRISM is measuring. Rather, we suggest that perceived burden of suffering is a manifestation of schema enmeshment. In this study, recall bias of illness words was the only significant predictor of where SLE patients placed the illness disc. This suggests that the observed cognitive bias is closely linked to the PRISM task. However, as we have not measured burden of suffering it is possible that this would also contribute to the variance in PRISM.

It is important to note the limitations of this study. Firstly, the extent to which these results can be generalised to other chronic illnesses is unknown, as only patients with SLE were included in the present study. The sample of SLE patients in the PRISM validation study (Buchi et al, 2002) differed from the other illness groups, in that the relationship between depression and PRISM was non-significant. Considering the established relationship between depression and cognitive bias (Pincus & Morley, 2001), it is unclear whether the relationships observed between negative illness self-schema and PRISM would be the same for other chronic illness samples. Further, participants were not a consecutive sample of patients and therefore may differ from the population of patients with SLE. Future research will be needed to investigate the generalisability of these results.

Secondly, whilst the sample size in the present study is sufficiently large to detect medium to large effect sizes, smaller effect sizes may not reach significance. For example, the Consequences subscale of the IPQ approached significance as an independent predictor of PRISM (p = 0.086). In a larger sample, it is likely that this relationship would have reached significance. Despite the limitations to power observed in the present study, in comparison to the literature on cognitive biases,
this is a large sample. Sample sizes in the cognitive bias and chronic pain literature have relied on samples of between 12 and 40 participants (for a review, see Pincus & Morley, 2001). Hence, it is likely that the sample size is sufficient to detect clinically meaningful results.

Notwithstanding these limitations, the results of our study strongly support the view that PRISM measures the concept of self and illness schema enmeshment. This is a potentially important finding because schema enmeshment is thought to be an important process in adjustment to illness and hence, its measurement has great potential clinical utility. To date, studies that have purported to measure negative illness self-schema have done so using complex computerised programs that take over half an hour to administer. While such tasks are theoretically interesting, their clinical usefulness is limited because of their cost-inefficiency. Although PRISM was administered in this study by computer, the original task was developed as a manual task. The task can be easily adapted for routine clinical administration and takes less than five minutes to complete. More importantly, even in this sample of patients with SLE, many of whom had cognitive deficits associated with their illness, PRISM was easily understood by all participants. We believe that these results demonstrate that PRISM may be a useful tool to assess the enmeshment of self and illness schema, and potentially has wide clinical and research applicability.
SECTION IV:

GENERAL DISCUSSION
The primary aim of this investigation was to explore illness self-schema in SLE via a cognitive bias paradigm. As no cognitive bias research has been conducted in SLE patients to date, two sets of hypotheses were proposed, partly generated on the basis of the biases that have been demonstrated in other patient populations. Firstly, it was suggested that the flare status of SLE patients may determine the cognitive bias (flare hypothesis), and secondly, that the determining factor is the presence of depression (depression hypothesis).

The results provide partial support for the depression hypothesis, however the results are not clear-cut. It was predicted that depressed SLE and RA patients would demonstrate endorsement and recall bias for negative illness-related words relative to positive illness words, whilst non-depressed patients and healthy controls would not demonstrate such a bias. This prediction was in line with the findings of Pincus and colleagues (1995), whose study the present investigation was based upon. They found that depressed pain patients endorsed and recalled more negative pain words and fewer positive pain words than non-depressed pain patients and controls (who did not differ from each other), whilst all groups endorsed and recalled depression and control words similarly. Despite using a similar task and the same stimuli, the present investigation could not replicate these findings. Although depressed SLE/RA patients endorsed more negative illness words than the other two groups, they also endorsed more negative depression words and negative control words, indicating a non-specific negative bias. In terms of recall of illness, depression and control words (positive and negative), the groups did not differ from each other in any meaningful fashion.

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4 Pincus and colleagues (1995) refer to the stimuli as 'pain' words whereas we have referred to them as 'illness' words. They are however, the same stimuli.
The most interesting results surfaced when the data was analysed in accordance with the structure of the *schema enmeshment model*, which distinguishes between pain-schema and illness-schema. Whilst no recall bias was found for negative illness words in general, when these stimuli were divided into sensory-pain and disability-related words the depressed patients demonstrated a clear bias for disability words. Non-depressed patients and healthy controls recalled the two types of words equally. So too with the patterns of endorsement; depressed patients endorsed significantly more disability words—but not sensory words—than non-depressed patients. Thus, our results support the depression hypothesis that it is the presence of depression in SLE and RA patients that determines cognitive bias, however the nature of the bias was at variance with our initial expectations.

Why then did we find different results to those found by Pincus and colleagues' (1995)? Clearly, the differences are unlikely to be due to methodological considerations as the present study replicated the endorsement and recall tasks and used the same stimuli. However, one way in which the current study differed methodologically from Pincus’ was in the use of PRISM as an interference task between the endorsement and recall sections of the cognitive bias task. It is possible that PRISM may have primed participants for the recall, given the illness component of the PRISM task. Whilst differences between groups is minimised because all participants completed the same series of tasks, this does represent a limitation of this study.

As mentioned earlier the patient populations are indeed different, and this may be the source of the discrepancy. The cerebral aspect of SLE can be responsible for poor cognitive functioning and neuropsychiatric presentations such as depression, which may have made general cognitive bias effects in our SLE sample less reliable than in samples of RA patients. Our RAVLT results and the absence of
any differences between the RA and SLE patients do not support this explanation, however as this is the first cognitive bias study to include SLE patients we cannot rule it out. Further research is certainly required before any generalisations can be made.

Thus, it is not entirely clear why our results differ from those found by Pincus and colleagues (1995). However, it is noteworthy that our results are not inconsistent with the literature in general. As mentioned earlier, the majority of published studies investigating recall biases in chronic pain have not found biases in a heterogenous group of pain patients towards heterogenous pain stimuli (Pincus & Morley, 2001). Rather, two studies found that only depressed pain patients demonstrated a recall bias for pain-related words (Clemmey & Nicassio, 1997; Pincus et al, 1995). Other studies have demonstrated that non-depressed patients show bias towards sensory words in preference to illness or disability words (Wells et al, 2003; Koutantji et al.; 1999; Edwards et al., 1992; Dehghani et al., submitted). Another found that depressed pain patients show the opposite bias to this mainstream tendency, demonstrating cognitive bias for affective, disability or illness-related words (Edwards et al., 1992). It is this latter effect that was observed in the present study.

Whilst Clemmey & Nicassio's (1997) study could be cited as one in support of Pincus and colleagues' (1995) results, on closer inspection the direct comparability of the results is unclear. The reason is that we cannot be certain of the stimuli that were used as the authors did not specify their words in the publication. They simply state that words "deemed to be health/illness relevant" were "generated…from dictionaries and a thesaurus" (p277) and validated in a pilot study. As a result, the reader is unable to ascertain the exact nature of the words
with regard to their ‘pain-sensory’ or ‘disability’ quality. Recent developments in the literature - and indeed, our own results - identify that this distinction is likely to be a significant one in terms of the presence of cognitive bias. Clemmey & Nicassio (1997) state their aim as being to identify illness schema, and they refer to their stimuli as being illness-related. One might surmise, therefore, that at least a reasonable proportion of the words are likely to be disability-related. Thus, their results may in fact reflect a more specific bias than at first glance, and may be more consistent with the results of the current study than they appear.

What is becoming increasingly apparent is that whilst our results are not clear-cut, neither is the literature. In many ways it appears that the results of Pincus and colleagues (1995) are the more unusual relative to the literature in general, yet can we discuss the ‘literature in general’ considering its ambiguity? It is the nature of the cognitive bias that is the complex issue to untangle. Whereas it has been established that some chronic pain/chronically ill patients demonstrate a processing bias for some stimuli related to pain and illness issues, the specific nature of that bias has not been established. Different studies have operationalised the concept of cognitive bias in different ways. Of those studies investigating recall bias (as opposed to attentional bias or interpretational bias), a different combination of stimuli has been used in each of them. For example, pain-sensory versus neutral versus general negative (Pearce et al, 1990), pain-sensory versus pain-affective versus neutral (Edwards et al, 1992), positive pain/illness versus negative pain/illness (Clemmey & Nicassio, 1997; Pincus et al, 1995) to name a few. The differing types of stimuli is a source of variance over and above the use of different actual stimuli (that is, words). In addition, the choice for dependent variable is different in different studies. Whilst the majority have used the proportion of words recalled (Wells et al, 2003; Koutantji et al, 1999; Calfas et al, 1997; 5 We contacted the authors in an attempt to obtain a list of the stimuli, however we received no response.}
Edwards et al, 1992), others have simply used the number of words recalled (Pincus et al, 1995; Pearce et al, 1990), and one study only considered recalled words that had been endorsed (Clemmey & Nicassio, 1997). These numerous sources of methodological variability are likely to contribute substantially to the apparent inconsistencies within the literature.

One consequence of the confusion as to how to operationalise cognitive bias, is uncertainty as to the precise relationship between cognitive bias and schema. Both Pincus and colleagues (1995) and Clemmey & Nicassio (1997) assert that endorsement bias and recall bias reflect self-schema. In both of these studies the endorsement data show the same bias as the recall data, and are therefore interpreted to be measuring the same construct. The present study, however, found the ill depressed patients demonstrated an endorsement bias for negative illness words, but not a recall bias. Furthermore, endorsement and recall of each word type were not correlated as they have been in numerous other studies (Calfas et al, 1997; Pincus et al, 1995). These results suggest that endorsement and recall are not necessarily one and the same as measures of self-schema. In their most recent investigation, Wells and colleagues (2003) appear to be in agreement with such a suggestion. Once again they utilise the endorsement and recall methodology, but for this study they only report the recall data. This suggests that they perceive the recall data as being a more relevant cognitive bias measure, whilst the endorsement of stimuli serves as a means of encoding.

This issue relates back to the nature of schema, hypothesised to consist of both content and function (Derry & Kuiper, 1981). Just as questionnaires are thought to measure schema content, it appears reasonable that endorsement of words as being self-descriptive would also reflect schema content. It is recall bias (or attention bias or interpretation bias) that represents the schema's function. One possible
explanation for finding endorsement bias but not recall bias relates to the
crudeness of the methodology employed. Endorsement on a 'yes' or 'no' basis is not
a realistic representation of the structure of beliefs, which are inherently non-
dichotomous in nature. Some individuals may endorse stimuli when their strength
of belief is weak, and some may endorse only when their strength of belief is
almost 100%. If the majority of individuals adopt the former approach the results
may indeed indicate an endorsement bias, however it may reflect a weak schema
that is not sufficiently pervasive to affect patterns of recall. The use of a Likert
scale during the endorsement stage is one way in which this issue could be
addressed. Such an approach would more accurately reflect the nature of beliefs, as
individuals would have the opportunity to endorse stimuli on a continuum. Future
research is required in order to empirically investigate the utility of such a
methodology, and whether or not the strength of endorsement is related to recall
bias.

Whilst we may not be certain of the nature of the relationship between
endorsement and recall bias, it is clear that they both relate to self-schema in some
way. A secondary aim of the current study of SLE patients was to investigate the
relationship between such supposed measures of self-schema, and PRISM, a
recently-developed measure of suffering (Buchi et al, 1998). Our results indicate
that PRISM may indeed be measuring schema-related issues in addition to
reflecting the burden of suffering due to illness. A multiple regression analysis
clearly demonstrates that recall bias for negative illness words makes an
independent and significant contribution to PRISM distance. [The fact that
endorsement bias did not make an independent contribution provides further
evidence that endorsement bias and recall bias are not one and the same.] As
discussed earlier, these results are interpreted as providing preliminary evidence
that the PRISM task may reflect the enmeshment of illness and self-schemas, as
outlined in the schema enmeshment model of chronic pain (Pincus & Morley, 2001).

When conducting these PRISM analyses the issue of which cognitive bias measure to use surfaced once again. At this stage it is worth reiterating the point that the distinction between pain schema and illness schema was not made in the literature until after the design of the present study. The initial intention was to use the negative/positive bias of illness-related words (assumed to reflect illness self-schema), however the unexpected findings of the primary investigation (Section II) forced a re-evaluation of the situation. After careful consideration it was decided to continue as planned, and use the difference between the proportion of positive and negative illness words recalled as a single dependent variable. This choice was made primarily as a result of the investigation being exploratory and the first of its kind. Firstly, no cognitive bias study has been conducted in SLE patients, and secondly, the PRISM task has not been explored in relation to any cognitive bias measure in any population. Therefore, it was considered most appropriate for the initial investigation to not be limited by overly specific hypotheses regarding subtypes of pain and illness words. Furthermore, the inclusion of all illness words accounted for the possibility that PRISM represents the enmeshment of illness and self schemas, pain and self schemas, or both. Certainly, future research would benefit from investigating this relationship further. If the PRISM task can be demonstrated to robustly reflect patients' illness or pain schemas, it would be an

For interest's sake, post-hoc analyses were conducted on the PRISM data with the SLE and RA patients grouped as per the depression hypothesis (ill-non-depressed versus ill-depressed). Ill-depressed patients placed the illness and self discs significantly closer together than did the ill-non-depressed (p<0.05). When RA patients were included in a post-hoc replication of the regression analysis, the contribution of recall bias remained the only significant predictor of PRISM distance. Recall of sensory and disability words were not significantly related to PRISM.
invaluable, quick and easy tool to assess schema and hence provide a greater understanding of how a patient is affected by his/her pain or illness.

One finding of the present study that is entirely in keeping with the literature is the lack of support for what we termed the flare hypothesis. There is has been an accumulation of evidence indicating that disease severity has little effect on quality of life, depression and schema-related issues (Persson & Sahlberg, 2002; Stoll et al, 2001; Wang et al, 2001; Wood-Dauphinee, 1999). Our results provide evidence for a similar effect in SLE patients. As mentioned earlier, the SF-36 scores of our SLE-nonflare sample do suggest that they were not entirely asymptomatic, thereby raising the query of whether our flare/nonflare comparison was justifiable. Nonetheless, if disease severity and/or symptomatology was indeed the determining factor for cognitive bias, at the very least a hint of a distinction should have been evident. No such hint was evident in the data.

As opposed to symptomatology determining cognitive bias, our results are more indicative of the specificity of stimuli being important, with particular regard to the sensory-pain or disability-related component of negative illness words. Furthermore, the presence of depression appears to play a role in either the activation of illness schemas or the enmeshment of them with the self-schema. Unfortunately the small sample size of our ill-depressed group (n=18) does represent a limitation of the study, as it reduced the power of detecting the positive-negative bias that Pincus and colleagues (1995) found. The patterns of recall bias for negative illness words were in the expected direction (see Table 8),
but failed to reach significance. This may suggest that the bias is occurring, but only in a subgroup of the stimuli (for example, disability versus sensory words). Our effect size was calculated as being moderate and requiring a sample size of 64 to become statistically significant. Once again it is clear that further research is required, using larger sample sizes and more consistent stimuli, to determine the true effect.

In summary, it appears as though the present investigation raises more questions than it answers. As discussed however, this is in keeping with the literature relating to recall bias in patients with chronic pain and/or chronic illness. When one considers the additional factor that the present study investigated the relatively rare and under-researched illness of SLE, it is not surprising that few firm conclusions can be drawn at this early stage. The results of this study need to be regarded cautiously, as the sample sizes are relatively small and the most interesting findings are based on post-hoc analyses. Naturally, as the majority of the participants were patients with SLE, the extent to which our findings can be generalised to other patient populations is unknown. Nevertheless, it was a major aim of this investigation to explore the nature of cognitive bias and illness schema in SLE in particular. Further research is required to ascertain how representative our results are of this illness in general. SLE has largely been neglected within much of the psychological literature, despite being a potentially debilitating disease with far-reaching consequences for the quality of life of its sufferers. A greater understanding of the belief structures and patterns of cognitive processing of SLE patients-and indeed, patients with any chronic illness or chronic pain issue-will inform the way in which we aim to improve their quality of life. Until pain
and illness are cured, the ultimate goal for psychologists remains to develop effective treatments that minimise the negative effect of chronic illness upon people's lives.
SECTION V:

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SECTION VI:

APPENDICES
APPENDIX A:

QUESTIONNAIRES
APPENDIX B:

STATISTICAL ANALYSES
Re: Section II
RE: SECTION III