Systemic Lupus Erythematosus Information in Physical Therapy Practice in Districts of New York State

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Introduction: Systemic Lupus Erythematosus (SLE) is a chronic autoimmune disease that involves multiple systems of the body affecting both physical and psychological function. Information regarding SLE and physical therapy interventions is scarce in the physical therapy literature. The purpose of this study was to examine the knowledge base of physical therapy clinicians as it pertains to SLE.

Methods: The survey was divided into three main areas: demographic information, methods/settings where information regarding SLE was obtained and questions pertaining to the clinician’s knowledge base as it relates to SLE. The survey was sent to members of the New York Physical Therapy Association in districts of New York State.

Results: Two-hundred and ninety-two surveys were analyzed. The clinicians reported that they obtained information regarding SLE through clinical practice (n=116, 39.7%), personal experience with individual with SLE who was not a patient/client (n=68, 23.3%), continuing education (n=25, 8.6%), classroom/lab education (n=204, 69.9%) and clinical education (n=28, 9.6%). The majority of clinicians indicated their knowledge as “minimal” (n=212, 74.9%), 55 (19.4%) reported adequate knowledge, 14 (14.9%) reported no knowledge pertaining to SLE and 2 respondents (0.7%) rated their knowledge as thorough.

Conclusion: There needs to be a mechanism of delivery of material regarding SLE in both physical therapy curricula and in continuing education for clinicians. Limitations of this study included its ability to be generalized to other areas of the country. Further study is needed into the extent and method of delivery of information regarding SLE in physical therapy curricula and continuing education.
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Systemic Lupus Erythematosus, known as SLE or Lupus, is a chronic autoimmune disease that affects multiple systems of the body. SLE has a varied clinical presentation and can affect individuals across their lifespan. Physical therapists (PTs) may encounter patients with SLE in a variety of clinical settings. As independent practitioners it is important for PTs to be aware of the clinical presentation, recognize the sign/symptoms, and understand the affects of the disease in order to provide appropriate interventions, education and referral to other practitioners.

The pathogenesis of SLE is not entirely understood, but it is thought to be the result of genetic factors that make a person vulnerable to the disease and environmental factors.\(^1\text{-}\text{5}^{1}\) Environmental factors that are related to SLE include drugs, viral infections, ultraviolet exposure, diet, stress, tobacco exposure and hormonal changes.\(^1,\text{3-7}^{1}\) The reported prevalence of SLE in the literature is variable with ranges of 14-51/100,000 adults.\(^1,\text{8,9}^{1}\) The Centers for Disease Control estimate that SLE affects 1.4 million people in the United States.\(^10\) The incidence of SLE has been reported to be 1 in 2500 of the general population with a higher incidence reported in African American women, of 1 in 250.\(^5,\text{7, 9, 11}^{5}\) SLE is also more common in Latin Americans, Native Americans and Asians.\(^8,\text{11-13}^{8}\) Most cases occur in women in their childbearing years, ages 15-45.\(^3,\text{5,11,13}^{3}\)

Uramoto et al used data from the Rochester Epidemiology Project and medical records to determine the incidence of SLE diagnosis between January 1, 1980 – December 31, 1992\(^6\). The average incidence rate for the white population was 5.56 per 100,000 (95%, CI 3.93-7.19) and prevalence of approximately 1.22/1000 (95%, CI 0.97-1.47).\(^6\) In another study,
Ward used data from the Third National Health and Nutrition Examination Survey (NHANES III) to estimate adult prevalence of SLE.\textsuperscript{14} Twenty thousand and fifty adult participants, 17 years or older, were asked if they had been diagnosed with SLE by a physician. Ward reported the prevalence of adult SLE to be higher at 241 per 100,000 (95% CI 130-352).\textsuperscript{14}

**SLE Classification:**

SLE has a varied clinical presentation making diagnosis of this disease difficult. Systems that may be affected include musculoskeletal, constitutional, integumentary, cardiorespiratory, renal, hematologic, gastrointestinal, and the nervous system.\textsuperscript{15-18} The American College of Rheumatology Ad Hoc Committee on Systemic Lupus Erythematosus (ARC Ad Hoc Committee) first established criteria for diagnosis of SLE in 1982 and the criteria were updated in 1997. For a diagnosis of SLE an individual must meet at least 4 of the 11 criteria, however an individual does not have to have the symptoms concurrently.\textsuperscript{19} The criteria include malar rash, discoid rash, photosensitivity, oral ulcers, non-erosive arthritis, pleuritis or pericarditis, renal disorder, neurologic disorder, hematologic disorder, immunologic disorder and positive antinuclear antibody.\textsuperscript{19} A full list of ACR Ad Hoc Committee criteria and the definitions are included in Table 1. If an individual has fewer than 4 of the 11 criteria a diagnosis of SLE may be considered on the basis of clinical judgment, and he/she may be described as having incomplete or latent SLE.\textsuperscript{8,19} It is also important to note that an individual may not initially meet the criteria for diagnosis of SLE but may develop further manifestations throughout the course of their disease.\textsuperscript{9} ACR suggests that SLE should be suspected if a patient presents with involvement of 2 or more organ systems.\textsuperscript{19}
System Involvement:

SLE affects multiple systems of the body. The following sections provide an overview of SLE system involvement and clinical manifestations.

Musculoskeletal:

Arthralgia is the most common manifestation of SLE with 90-95% of individuals with SLE reporting joint pain.\textsuperscript{16, 20-23} It also has been reported to be the most common initial symptom in approximately 50% of individuals with SLE\textsuperscript{16, 22, 24} and the most common reason that individuals seek medical attention.\textsuperscript{17, 25} Knees, wrists, hands and feet are most frequently involved.\textsuperscript{17, 21, 23-27} Both SLE and rheumatoid arthritis (RA) present with morning stiffness, symmetrical presentation and polyarthritis making diagnosis difficult.\textsuperscript{22, 27} Unlike RA, active signs of inflammation are less common in SLE with only mild swelling with or without effusion noted.\textsuperscript{22, 27} Arthritis associated with SLE is non-erosive and there is less synovial thickening.\textsuperscript{22, 23, 25, 27, 28} Analysis of synovial fluid may assist in the differentiation of SLE versus RA.\textsuperscript{27} Both frequently test positive for antinuclear antibodies, however the synovial fluid in SLE has specific complement levels that are associated with it.\textsuperscript{25, 27}

Deforming arthritis is less common in SLE than RA, however there are cases of severe hand deformities.\textsuperscript{27} Jaccoud’s Syndrome is a form of arthritis that presents as a pattern of nonerosive but deforming disease.\textsuperscript{17, 22, 26, 27} In this form there is little inflammation or erosion but the deformities that occur may be the result of ligamentous laxity.\textsuperscript{17, 22, 27}

Tenosynovitis, bursitis and nodule formation can also occur with SLE.\textsuperscript{16, 22} Tendon rupture is less common, but has been documented in SLE, and has been linked to periods of “lupus flare” and corticosteroid therapy.\textsuperscript{22, 27, 29} Myalgia with pain and weakness is a common finding. Zoma reported myalgia to occur in 40-80 % of individuals with SLE.\textsuperscript{22} Myopathy or
myositis has a similar presentation to idiopathic myopathy or polymyositis but is usually less severe in individuals with SLE.\textsuperscript{27} It has been reported to be present in 5-10\% of individuals with SLE.\textsuperscript{22, 27} These inflammatory myopathies may be the result of active disease, corticosteroid therapy, antimalarial therapy and the use of statins.\textsuperscript{22, 27}

Avascular necrosis (AVN) is frequently seen in individuals with SLE with occurrence ranging from 5-40\%.\textsuperscript{22, 27, 30, 31} The hip is most commonly involved, however it is also seen in the knee, shoulder, talus, scaphoid and lunate.\textsuperscript{17, 27} Corticosteriod therapy is thought to be the leading cause of AVN in SLE.\textsuperscript{17, 27, 30} Nagasawa et al\textsuperscript{30} looked at the relationship between corticosteriod use in SLE and the development of necrosis of the femoral head. Forty-five patients were studied who were specially selected on the basis of new diagnosis of SLE and who required a high dose (40mg/day or more) of prednisone. MRI and plain radiography of the femoral head were performed 3 months following the beginning of corticosteriod treatment. The subjects had repeat testing every year for a period of 5 years. Silent Osteonecrosis of Femoral head (ONF) was diagnosed if MRI changes were noted but no symptoms were reported. If the patient had MRI changes in the presence of continuous hip pain and/or radiographic changes then they were said to have Conventional Symptomatic ONF. Forty of the 45 subjects completed the study. All of the 40 subjects continued steroid therapy. Fifteen of the subjects (33\%) developed Silent ONF and 14 of those had evidence of AVN at the 3-month examination. Five of the subjects progressed and developed symptomatic ONF.

Antiphospholipid Antibody Syndrome (APS), abnormalities in bone metabolism, vasospasm and vasculitis may also play a role in the development of AVN.\textsuperscript{27, 30-32} Tektonidou and associates evaluated 79 subjects using MRI.\textsuperscript{33} Subjects included patients with primary
antiphospholipid syndrome (APS) (n=30), patients with SLE (n=19) and 30 healthy individuals. Subjects were matched by age and sex. All subjects had no symptoms of AVN and had no history of corticosteroid therapy. In the study, 20% (n=6) of the 30 patients with primary APS had AVN on MRI evaluation and 3 individuals had early AVN. In contrast to other studies, none of the patients with SLE demonstrated AVN. Mok et al reviewed a cohort of 265 patients receiving long-term follow up in a SLE clinic between 1978 and 1998. Individuals with SLE who had APL were matched with individuals with SLE who did not have APL and with 31 patients chosen at random. Four percent (n=11) demonstrated AVN. There was no significant difference in this group as it related to the presence of antiphospholipid antibodies.

Osteoporosis has been reported in individuals with SLE. Reasons for the development of osteoporosis include chronic steroid therapy, renal failure, long-standing arthritis, ovarian dysfunction, physical inactivity and avoidance of sunshine. Pathogenesis of osteoporosis in SLE is multifactorial. Lee and Ramsey-Golman have reported traditional and SLE related risk factors for development of osteoporosis (Table 2). SLE inflammatory mediators and cytokines may cause accelerated bone remodeling resulting in reduced bone mineral density (BMD). Active SLE has also been linked to ovarian dysfunction and premature menopause. Premature menopause may also be a result of immunosuppressive therapy in individuals with SLE. Individuals with SLE are encouraged to limit sun exposure to prevent “flares” which in turn may result in vitamin D deficiency. Vitamin D production may also be altered by renal dysfunction in individuals with SLE.

Chronic use of corticosteroids is a risk factor for osteoporosis in SLE. Glucocorticoids have been shown to alter normal bone production and remodeling.
Sinigaglia et al.\textsuperscript{35} looked at bone mineral density in 84 premenopausal patients with SLE. All patients were receiving corticosteroids at the time of the study. Osteoporosis was detected in 22.6\% of these patients.\textsuperscript{35} In the study by Pineus et al.,\textsuperscript{39} 205 women with SLE were evaluated for osteoporosis using dual energy X-ray absorptiometry (DEXA). DEXA scan showed that 18\% had osteoporosis, 48.8\% osteopenia and 33.3\% had normal bone mineral density, however there was no association found between corticosteroid use and osteoporosis.\textsuperscript{39} Kipen et al.\textsuperscript{41} measured bone mineral density in 32 premenopausal women with SLE over a 3 year period in an effort to identify factors predictive of bone loss in this population. Of the 32 subjects, 21 had drug therapy utilizing corticosteroids. There was no significant change in BMD of the lumbar spine or femoral neck in the subjects as a whole. A subgroup of patients who were treated with corticosteroids greater than or equal to 7.5mg/day (n=14) had a significant loss (p=0.01) of BMD in the lumbar spine.\textsuperscript{41} They also found that disease activity, severity, or duration were not predictive of bone loss.\textsuperscript{41}

The prevalence of osteoporosis in individuals with SLE is variable across different racial groups.\textsuperscript{37} Lee and Ramsey-Goldman suggested that the variation among different groups may be partially the result of differences in the inherent calcium metabolism, severity of SLE, cumulative exposure to specific disease and therapeutic interventions the individual received.\textsuperscript{37} The prevalence of osteoporosis in their analysis of various racial groups ranged from 1.4-16\%. Yee and associates studied 242 patients of various racial backgrounds and 50.8\% (n=123) demonstrated reduced BMD, having a T score < -1, and 10.3\% (n=25) were found to have osteoporosis with a T score < -2.5.\textsuperscript{36} Fragility fractures occurred in 9.1\% of the subjects. In this study non-Afro-Caribbean race (OR 2.5 (1.2 to 5.4); CI 95\%) and
exposure to predisone >10mg/day (OR= 2.1 (1.1 to 4.2); CI 95%) were associated with reduced BMD.\textsuperscript{36}

The use of anticonvulsant therapy in individuals with SLE has been linked to a lower BMD.\textsuperscript{37} Lee and Ramsey-Goldman state that this may be the result of accelerated vitamin D metabolism resulting in decreased calcium absorption.\textsuperscript{37} The use of prolonged anticoagulant therapy to prevent thrombosis formation in individuals with SLE can also alter bone production and reabsorption resulting in low BMD.\textsuperscript{37}

Musculoskeletal involvement in SLE is varied and the clinical presentation of SLE may mimic other diseases such as rheumatoid arthritis, fibromyalgia, and Sjogren’s syndrome.\textsuperscript{18, 19} It is important to be aware of the possible clinical presentation of SLE and to be able to screen for signs of SLE in physical therapy examinations. The possibility of reduced bone mineral density and AVN should be considered in individuals with SLE when designing physical therapy interventions.

**Constitutional:**

Involvement of the constitutional system include fatigue, fever, malaise, and weight changes (loss or gain).\textsuperscript{12, 16, 17, 25, 28} Gill et al\textsuperscript{12} reported 50-100 % of SLE patients had involvement of the constitutional system. These symptoms, however, are nonspecific and may not be recognized as clinical presentations of SLE, thus delaying diagnosis.\textsuperscript{12} Nass reported that episodic fever was experienced by more than 80 % of SLE patients.\textsuperscript{16} The fever is usually low grade, however it may be high during acute flare up of the disease.\textsuperscript{16} The low grade fever associated with active SLE needs to be distinguished from fever associated with infections.\textsuperscript{44} Infections, common in individuals with SLE, are a major cause of morbidity and mortality.\textsuperscript{45-48} Infections are more common in individuals with SLE due to an impaired
immune system and the use of immunosuppressive drugs to treat SLE.\textsuperscript{44, 49} Individuals with SLE are also at risk for opportunistic infections as a result of active disease, immunosuppressive therapy and corticosteroid treatment. Trager and Ward reported risk factors for serious bacterial infection include immunosuppressive treatment with cytotoxic medications or corticosteroids, proteinuria, renal insufficiency and active SLE.\textsuperscript{45} Physical therapists should be aware of the increased risk of infections and monitor patients with SLE closely.

Extreme fatigue is another commonly reported symptom in individuals with SLE and has been reported to occur in 80-100\% of individuals with SLE.\textsuperscript{17} Reasons cited for this extreme fatigue include active disease, anemia, cardiac involvement, hypertension, depression and deconditioning.\textsuperscript{50} Mckinley and associates looked at fatigue, depression and sleep problems in 48 women with SLE compared to 27 women from the general population.\textsuperscript{50} The SLE group reported greater overall fatigue than did the controls (p<.03). The women in the control group reported fatigue as an “acute, short-lived experience” whereas the women with SLE reported continuous fatigue which increased periodically.\textsuperscript{50} Krupp et al\textsuperscript{51} looked at severity of fatigue using a Likert-type Fatigue Severity Scale (FSS) in 59 individuals with SLE. Fatigue was reported as the most disabling symptom in 53\% of the participants. Fatigue had a low correlation with depression( r=0.46, p<0.001).

\textbf{Integumentary:}

Skin lesions are a common presentation in individuals with SLE. Cutaneous involvement has been reported to occur in 70 - 90 \% of patients with SLE over the course of their disease.\textsuperscript{21, 44} Skin involvement has a wide clinical presentation including rashes, oral or nasophynggeal ulcers, photosensitivity and alopecia.\textsuperscript{16, 17, 20, 23, 26, 52} Hughes et al reported that
70% of patients reported alopecia and 20-40% present with oral lesions typically involving lips, hard palate, buccal mucosa or tongue, petechiae and temporomandibular joint (TMJ) erosion.\textsuperscript{53}

Three forms of skin involvement have been found to be specific to SLE. These include acute, subacute and chronic cutaneous lupus.\textsuperscript{44} Acute Cutaneous Lupus is erythema across the bridge of the nose and face which is commonly referred to as a “butterfly rash”.\textsuperscript{20, 26, 44} These lesions can also occur at other sites, especially sites exposed to sun.\textsuperscript{44} Subacute Cutaneous Lupus occurs in about 10% of individuals with SLE and is characterized by annular erythematous papulosquamous eruptions.\textsuperscript{44} These lesions resemble psoriasis however are non-scarring and have periods of exacerbation and remissions.\textsuperscript{17, 44} Chronic Cutaneous Lupus, or Discoid Lupus, is more severe and results in depigmentation and scarring.\textsuperscript{20, 26} These lesions can occur in individuals with SLE with or without other systemic manifestations. It can also appear in individuals without SLE, which is referred to as Discoid Lupus Erythematosus (DLE).\textsuperscript{17, 26, 44} Chronic Cutaneous Lupus Erythematosus or DLE has been reported to occur in 15-30% of patients with SLE.\textsuperscript{16}

Other less common cutaneous manifestations included patchy erythema, livedo reticularis, urticarial lesions and paniculitis.\textsuperscript{8, 26, 44} Cutaneous vasculitis is damage of the vessels of the skin and typically appears as small red-purple spots.\textsuperscript{20} Vasculitis can also present as hive-like or wheal-like lesions, urticarial vasculitis or small red-purple hives or spots in the folds of the fingers or tips of the fingers.\textsuperscript{20} Raynaud’s phenomenon is another common cutaneous symptom in SLE.\textsuperscript{24, 52} Hay and Smith reported that Raynaud’s phenomenon occurs in approximately half of the individuals with SLE.\textsuperscript{52} Hauptman stated that 20-25% of individuals with SLE will exhibit signs of Raynaud’s over the course of their disease.\textsuperscript{24}
Alopecia or hair loss is common in SLE and can be diffuse or patchy. It is often associated with an exacerbation of the disease and hair can re-grow during periods of remission. Nass reported that approximately half of individuals with SLE have alopecia. In individuals with SLE, skin lesions and alopecia can be caused or exacerbated by exposure to the sun. Robinson reported that approximately one third to two thirds of patients with SLE have photosensitivity resulting in exacerbation of cutaneous and systemic symptoms.

During the screening process unusual fatigue, weight loss, alopecia, rashes and photosensitivity may be detected by PTs, which may assist them in their differential diagnosis.

**Pulmonary:**

The most common pulmonary manifestation in SLE is pleuritis. Symptoms of pleuritis include pleuritic chest pain, cough, dyspnea and fever. It has been reported to affect 40-60% of individuals with SLE. Immune complex deposition is thought to be related to the pathogenesis of pleuritis. Other less common pulmonary complications include pneumonitis, hemorrhage, interstitial fibrositis and pulmonary fibrosis. Pneumonitis is an infection of the lung tissue and can be caused by infection or active SLE disease process. A less common form of pneumonitis found in SLE is Chronic Diffuse Interstitial Lung Disease. Paran et al reported that Chronic Diffuse Interstitial Lung Disease affects 3-8% of SLE patients. Symptoms include chronic non-productive cough, dyspnea especially upon exertion, and recurrent pleuritic chest pain. This form can result in permanent lung scarring, impaired oxygen diffusion and difficulty breathing.
Pathogenesis involves "cytokines and mediators secreted by alveolar macrophages and inflammatory cells".  

Pulmonary Hypertension (PHT) is characterized by higher than normal pulmonary artery pressures, at rest and with exercise.  

PHT can be classified as primary or secondary.  

The cause of primary Pulmonary Hypertension is unknown but it is thought to be a result of an autoimmune process.  

Secondary PHT in SLE may be the result of valvular disease, pulmonary embolism, interstitial lung disease or a combination of these factors.  

Pulmonary hypertension can be life threatening, has a poor prognosis and at present there is no effective therapy for this condition.  

Simonson et al found the prevalence of pulmonary hypertension to be 14 % (n=36) whereas Mills found the prevalence to be 5 % in individuals with SLE.  

Ling-Te Pan et al performed a retrospective study of a cohort of 786 SLE patients in Tan Tock Seng Hospital in Singapore. In this study 59 patients were found to have PHT (11.6%).  

Of the 46 individuals with PHT who were able to complete the study, 22 subjects had primary PHT and 24 subjects had secondary PHT.  

Recurrent atelectasis results in lung damage and has been referred to as "shrinking lung syndrome".  

Shrinking Lung Syndrome refers to diaphragmatic dysfunction which is characterized by "dyspnea, pleuritic chest pain, reduced lung volume as demonstrated on chest radiographs and pulmonary function tests, accompanied by a restrictive pulmonary function pattern and diaphragmatic elevation".  

Weakness of the diaphragm and respiratory muscle fatigue have been linked to the pathogenesis of this syndrome.  

Karim et al performed a retrospective study of all SLE patients in St. Thomas Hospital and Royal London Hospitals between 1984 and 2002 (n=2650) and 7 patients (3.78%) were found to have recurrent atelectasis.
Pulmonary complications can result in decreased lung capacity, dyspnea and decreased physical function. Hellman and associates looked at 25 patients with SLE and found that dyspnea was reported by 60% (95% CI 39-79%) of their patients, with 20% (95% CI 7-40) having severe dyspnea and 12% (95% CI 3-31%) having moderate dyspnea. They also reported that those patients with dyspnea had lower total lung capacity (p=0.002) and reduced maximum oxygen consumption (p=0.01).

PTs may be working with individuals with SLE who demonstrate a variety of pulmonary complications that affect their function. It is important to be able to recognize signs of pulmonary involvement and understand the effect on physical function.

Cardiac:

Cardiac complications occur frequently in individuals with SLE. Kuper and Failla reported between 40-50% of patients with SLE experience cardiovascular and pulmonary manifestations. The major cardiovascular complication is accelerated atherosclerosis. It is a major cause of death in the later stages of SLE. Possible reasons cited for this acceleration of atherosclerosis include the effects of pharmacotherapy, increased blood pressure, elevated serum cholesterol and body weight. Patients with SLE are at a greater risk for developing hypertension. Factors associated with this increased risk include renal disease, accelerated arteriosclerosis, corticosteroid use and weight gain. In a study of 28 women with SLE over a 3 year period, Kippen et al found a significant increase in body mass and fat mass (p=0.03). Hypertension was also found to be associated with mortality. Seleznick and Fries, in their prospective study of patients with SLE at Stanford University Immunology Clinic between January 1, 1970 and October 15, 1982,
reported that each millimeter unit increase in systolic blood pressure corresponded to a 2% increase in mortality rate.\textsuperscript{63}

Other cardiac manifestations include pericarditis and myocardial infarction (MI). The incidence of pericarditis has been reported to be 20-50\%.\textsuperscript{18, 26, 59} Manzi et al.\textsuperscript{64} reported that myocardial infarction was fifty times more common in women with SLE compared to control groups. In Surfelt's epidemiological based study of 74 patients in a defined area of Sweden from 1981-1988, 7 out of 74 patients (9.45\%) were found to have had a MI.\textsuperscript{59} Esdaile and associates reported an 8.3\% increased risk of myocardial infarction in patients with SLE compared to control subjects who were matched according to traditional cardiac risk factors.\textsuperscript{65}

The presence of antiphospholipid antibodies and Antiphospholipid Syndrome (APS) has been linked with SLE. The incidence of APS in individuals with SLE has been reported to be approximately 33-50\%.\textsuperscript{20, 21, 44} The presence of these antibodies have been shown to increase the likelihood of developing arterial and venous thrombus, thrombocytopenia, and valvular heart disease.\textsuperscript{11, 16, 20, 21, 44} Thrombus formation may lead to myocardial infarct and aortic occlusion.\textsuperscript{21, 44} Surfelt reported that 27\% (n=74) of the individuals in his study were found to have valvular disease.\textsuperscript{59} A statistically significant association (p<0.01) between immunoglobulin G (IgG) anticardiolipin (aCL) and the presence of valvular abnormalities was reported.\textsuperscript{59} Mills reported pericardial effusions were common and often the first manifestation in individuals with SLE.\textsuperscript{18} SLE has been shown to affect the cardiac system.

PTs should be aware of the wide range of cardiac involvement, especially the possibility of accelerated atherosclerosis in the younger SLE population, and the effect of SLE on
overall health and function. In addition to care after known cardiac event, PTs can assist the individual with SLE in modification of cardiac risk factors.

Renal:

Most individuals with SLE demonstrate some renal involvement over the course of their disease.\textsuperscript{17,21,23,26,28} Glomerulonephritis has been reported to occur in approximately 50-75% of individuals with SLE.\textsuperscript{9,21,23} The severity of nephritis is variable and the World Health Organization (WHO) has classified lupus nephritis based on glomerular lesions (Table 3).\textsuperscript{23} Individuals with SLE have progressive disease which may lead to translation to another class.\textsuperscript{23} Symptoms of glomerulonephritis include proteinuria, hematuria and pyuria.\textsuperscript{1,17,19,20,26,28,66} Other renal involvement includes large vessel vasculitis, interstitial nephritis, and renal tubular acidosis.\textsuperscript{17,21,26,28}

Hematological:

Hematological manifestations of SLE include anemia, leuokopenia, lymphocytopenia, thrombocytopenia and thrombosis formation.\textsuperscript{11,16,17,20,21,26} Anemia is the most common hematological manifestation with incidence of 40-75% in individuals with SLE.\textsuperscript{16,21,25} Anemia is thought to be the result of iron deficiency, active disease and autoimmune reaction to RBC, gastrointestinal blood loss from intestinal disease and medications used to treat SLE.\textsuperscript{16,17,21,26} Neutropenia and lymphopenia are said to be less common and thought to be the result of active disease processes, pharmacotherapy and infection.\textsuperscript{20,26} The incidence of these manifestations varies with reports of 15-60% of individuals with SLE having these conditions.\textsuperscript{16,21} Thrombocytopenia is also relatively common in individuals with SLE with reported incidence of 15-35%.\textsuperscript{16,17,21,26} The presence of Antiphospholipid Syndrome (APS) is reported to be approximately 33-50%. It has been linked to thrombus formation, myocardial
infarction, anemia, fetal loss, neurological abnormalities, valvular disease and livedo reticularis.\textsuperscript{11, 16, 20, 44}

**Gastrointestinal:**

Gastrointestinal (GI) manifestations and their severity are variable. Frequent GI complaints are nausea, vomiting, anorexia and abdominal pain.\textsuperscript{1, 16, 17} Abdominal pain caused by vasculitis, thrombosis, tissue infection and ischemic bowel has been reported to occur in approximately one-third of individuals with SLE.\textsuperscript{2} Thrombosis, vasculitis and tissue infection can lead to peritonitis, nonspecific inflammatory liver disease, pancreatitis, mesenteric vasculitis and inflammatory bowel disease.\textsuperscript{1, 17, 18, 26} The incidence of pancreatitis in adults with SLE has been reported to be 3-8%.\textsuperscript{67}

**Neurological:**

Involvement of the Nervous system is a frequent manifestation of SLE, with the incidence of involvement to be 14-90%.\textsuperscript{2, 21, 68-70} Some of the variability may be explained by different diagnostic criteria or research methodology.\textsuperscript{71} The causes of neuropsychiatric manifestations in SLE include: direct disease activity (antibody production, microvasculopathy and pro-inflammatory cytokines) and secondary changes as a result of infection, metabolic complications and drug therapy.\textsuperscript{21, 69, 72-74} There is a spectrum of neuropsychiatric involvement such as stroke, seizure, peripheral neuropathy, chorea, dementia, psychosis, anxiety, depression, and attention, memory and visual-spatial abnormalities.\textsuperscript{2, 18, 71} Nineteen neuropsychiatric syndromes for SLE (NPSLE) have been classified by the American College of Rheumatology (ACR) ad HOC Committee on Neuropsychiatric Lupus Nomenclature and are shown in Table 4.\textsuperscript{19}
In a study by Brey and associates of 128 individuals with SLE, 80% of the subjects had one or more of the NPSLE syndromes defined by ACR.\textsuperscript{71} Of the subjects with NPSLE, 67 received neuropsychological testing. Forty-three percent had mild impairment, 30% had moderate impairment and 6% had severe neuropsychological involvement.\textsuperscript{71} In Ainiala’s et al\textsuperscript{70} study of 46 individuals with SLE in Finland 91% (n=42) demonstrated signs of neuropsychiatric involvement. The major syndromes seen were cognitive dysfunction (80%, n=37), headache (54%, n=25), mood disorders (44% n=20), polyneuropathy (28%, n=13), cerebrovascular disease (15%, n=17), and anxiety disorders (13%, n=6).\textsuperscript{70}

A common CNS abnormality seen in SLE is microfocal scarring as a result of changes in the arterioles.\textsuperscript{18} Arteritis of the larger vessels or thromboembolism may result in stroke or intracranial hemorrhage.\textsuperscript{18} Furlow reported that intracranial hemorrhages caused by brain aneurysm rupture occurs in more than 40% of SLE patients who had hypertension or thrombocytopenia.\textsuperscript{2} Infections may result in CNS involvement.\textsuperscript{2} Seizures, another CNS manifestation, are usually complex partial.\textsuperscript{18,21}

Psychological symptoms are variable; they commonly include depression, anxiety and labiality.\textsuperscript{74} Kozora et al\textsuperscript{74} examined the psychological processes (measures of life stress, coping styles, depression, social support and cognitive ability) in 52 patients with SLE, 29 patients with RA and 27 healthy control subjects. The participants completed The Beck Depression Inventory (BDI), The Profile of Mood States (POMS), The Psychiatric Epidemiological Research Instrument (PERI), Life Events and Difficulty Schedule (LEDS), The Coping Style Inventory (CSI), The Social Support Questionnaire (SSQ) and The Cognitive Impairment Index.\textsuperscript{74} Symptoms of depression were found in 25% of the individuals with SLE and in 7% of the patients with RA.\textsuperscript{74} The subjects in the control group
did not report depression. The individuals with SLE were also “more distressed” as evident on POMS (p<0.001).

Costa et al. assessed sleep quality in 100 women with SLE using the Pittsburgh Sleep Quality Index (PSQI). Fifty-six percent of the participants had moderate/severe sleep disturbance. The decrease in sleep quality was linked to depressed mood (p<0.0001), prednisone use (p<0.001) and decrease in physical activity (p=0.001).

A frequent but controversial neuropsychiatric syndrome is the presence of headache in individuals with SLE. Headaches have been reported to occur in 5.6-68% of individuals with SLE. The pathogenesis of SLE-related headaches is unknown. Some theories of the pathogenesis of primary headaches in individuals with SLE include vascular reactivity, neuronal excitation followed by neuronal inhibition, circulating cytokines, neuronal damage and muscle tension. Secondary headaches may be the result of infections, meningitis, hypertension, subdural hematoma, sinusitis, and venous sinus thrombosis.

Cuadrado and Sanna reviewed current literature relating to headaches in individuals with SLE. They suggested that the variability of prevalence was due to the differences in the classification systems for headache and the wide range of research designs. Mitsikostas and associates performed a meta-analysis of 8 studies that used the International Headache Society (IHS) criteria. Using these criteria 57.1% of patients with SLE reported headaches, 31.7% were migraine and 23.5% were tension headaches. There was no particular pathogenic mechanism identified for headaches in the individuals with SLE. The studies reviewed by Mitsikostas and associates reported varying incidence of headache, however there was no significant difference in the incidence from the normal population.
Glanz et al \textsuperscript{77} surveyed 414 patients who met the ACR criteria for SLE. One hundred and eighty-six patients completed the questionnaire that was based on the IHS guidelines for headaches. In addition there were questions relating to other manifestations of SLE. One hundred and fifteen respondents (62\%) reported headaches (39\% met the criteria for migraines and 23\% non-migraine headaches). They reported no significant association between migraine and other clinical features of SLE.\textsuperscript{77}

Physical therapists may encounter patients with a variety of neurological involvement, both in the central and peripheral nervous systems. Cognitive impairment is also variable and should be taken into consideration when developing interventions. Headaches may be confused with those arising from other sources such as tension headache and migraines. Seizure disorders can occur and PTs should be aware of this CNS manifestation.

**Pediatric:**

Because SLE can occur across the lifespan and affect multiple systems, PTs may encounter children with SLE in various practice settings. Furlow reported that SLE in children represents 20\% of all SLE patients.\textsuperscript{2} The most frequent initial manifestations of SLE in children include malar rash, photosensitivity, arthritis, fever, cardiac, neurological and renal involvement.\textsuperscript{2,67,79,80} In a study by Bader-Meuner et al\textsuperscript{67} of 155 patients who developed SLE prior to the age of 16, the most common initial manifestations were hematologic (72\%), cutaneous (70\%), musculoskeletal (64\%), renal (50\%) and fever (58\%). Abdominal pain is a “nonclassical” initial presentation, however it was found in 1/3 of the study participants.\textsuperscript{67} In a study by Lilleby and Forre of 71 individuals with childhood onset SLE, the most frequent clinical manifestations at diagnosis were: malar rash (55\%), photosensitivity (55\%), arthritis (48\%), fever (45\%) and renal involvement (25\%).\textsuperscript{79}
Pediatric or childhood onset SLE is usually more aggressive with a higher rate of organ involvement.\textsuperscript{2, 60, 79} Bader-Meuner et al\textsuperscript{67} reported that 40% of the subjects had severe organ involvement within the first month after the diagnosis. Lilleby and Forre used the SLE Disease Activity Index (SLEDAI) and The Systemic Lupus International Collaborating Clinics/American College of Rheumatology Damage Index (SDI) to measure disease activity and non-reversible organ damage respectively.\textsuperscript{79} In individuals who had onset of SLE in childhood, the most common organ systems to be affected were neuropsychiatric (28%), cerebrovascular (10%), cognitive (13%), renal (13%) and musculoskeletal (13%). Major causes of morbidity and mortality in this population are brain ischemia, atherosclerosis and cardiac disease.\textsuperscript{2} Risk factors for the accelerated atherosclerosis found in pediatric SLE include arterial vasculitis, APL antibodies, steroid induced obesity and hypertension.\textsuperscript{2} Pericarditis and Myocarditis have been reported to occur in approximately 30% and 25% of children with SLE, respectively.\textsuperscript{80}

Renal involvement is a major concern, occurring in 50-90% of children with SLE.\textsuperscript{80} It has been found to be a common initial manifestation and is usually more severe in children at the onset than adults.\textsuperscript{80} Renal disease is the major cause of mortality in children with SLE.\textsuperscript{80} The risk of atherosclerosis is increased by lupus nephritis and its effects on blood pressure and lipid levels.\textsuperscript{80}

The incidence of neurological involvement in children with SLE is approximately 20-35% and is also a leading cause of death.\textsuperscript{80} As with adults, neurological involvement is vast and variable. The most common neurologic manifestations in children are psychosis and seizures.\textsuperscript{80} Boon reported that 10% of children also report severe headaches.\textsuperscript{80} Children with SLE and antiphospholipid antibodies were found to be at a greater risk for strokes.\textsuperscript{2} The
neuropsychiatric manifestations may be difficult to diagnose because children may present with more vague complaints such as decreased ability to concentrate, mood changes or decreased performance in school.\textsuperscript{80}

Common hematologic manifestations of SLE in children include thrombocytopenia, leukopenia, vasculitis and thrombosis formation.\textsuperscript{80} Vasculitis in SLE affects the small blood vessels of the body and can affect various organ systems.\textsuperscript{80} Boon reported that 1/3 of children with SLE have APL antibodies which are associated with thrombosis, stroke, myocardial infarction, headaches and Raynaud’s Phenomenon.\textsuperscript{80}

GI symptoms may be more difficult to detect in children with vague complaints of abdominal pain reported. Inflammation of the GI vascular system can cause esophageal motility abnormalities, colitis, and peritonitis.\textsuperscript{80} The child may present with abdominal pain, anorexia and weight loss. Pancreatitis may occur and can be life threatening.\textsuperscript{80}

Integumentary involvement in children is similar to adults with SLE with the most common manifestations including malar rash, photosensitivity, cutaneous vasculitis, alopecia and oral/pharyngeal ulcerations.\textsuperscript{80}

Musculoskeletal involvement in children with SLE is similar to that of adults with SLE. Complaints of arthralgia and myalgias are common in children with reported incidence to be 85%.\textsuperscript{80} As with adults, the arthritis in children with SLE is non-erosive but can lead to contracture and subluxations. Tenosynovitis and AVN can also occur in children with SLE.\textsuperscript{80}

The most common pulmonary involvements in children with SLE are pleuritis and pleural effusions.\textsuperscript{80} Children with SLE have also been found to have abnormalities in diffusion and restrictive lung disease.\textsuperscript{80} A complete list of pulmonary manifestations, as described by Boon, are included in Table 5.
Late-Onset SLE:

Even though SLE is most common in the childbearing years, it can be seen in the geriatric population. With recent advances in the treatment of SLE more individuals are surviving into “old age”\(^8\). There is also a subset of individuals with SLE who are diagnosed after the age of 50 and are said to have Late-Onset SLE.\(^8\) Late-Onset SLE differs from early onset SLE in its clinical presentation. It is still more common in women, however the ratio of female to male is reduced from 9:1 to 6.9:1 in late-onset SLE.\(^8\) There is an increase in the predominance of late onset SLE in the White population.\(^8\)

The symptoms of late-onset SLE are variable and may not present in the typical fashion.\(^8\) Individuals with late-onset SLE can present with more vague symptoms such as stiffness, weakness, fever, malaise and weight loss.\(^8\) These symptoms are commonly seen in the elderly and may not be recognized as symptoms of Late-Onset SLE.\(^8\) Differences in the manifestations have been reported with alopecia, malar rash, nephropathy, and lymphadenopathy occurring less frequently in individuals with Late-Onset SLE.\(^8\) An increase in serositis and pulmonary involvement in Late-Onset SLE has been reported.\(^8\)

Even though there is a high incidence of arthritis (72.3%)\(^8\) in late-onset SLE some of the arthritis may be attributed to osteoarthritides and chondrocalcinosis which is found in normal elderly population.\(^8\) Glomerulonephritis is also less frequent (28.6%)\(^8\) however it still occurs in 1/5 to 1/4 of the individuals with late-onset SLE.\(^8\) CNS involvement is also less common but still effects 1/4 of those with Late-Onset SLE.\(^8\)

Diagnosis of late-onset SLE may be more difficult in the elderly due to the difference in manifestations, underlying problems found in the normal elderly population, and the fact that antinuclear antibody testing is not a reliable indicator of SLE in the elderly population.\(^8\)
atypical and vague symptoms may be mistakenly attributed to the aging process. Physical
therapists should be aware of the symptoms and the possibility of late-onset SLE during the
screening process. In the geriatric setting, PTs may deal with the long term effects of early
onset SLE as well as those of late-onset SLE. Therapists should be aware of the differences
in the types of adult SLE and the clinical manifestations when establishing a plan of care.

**Aerobic Capacity:**

Decrease in aerobic capacity and physical function is common in individuals with SLE. Tench et al.\(^{85}\) examined the aerobic fitness, muscle strength, fatigue and physical disability in 93 individuals with SLE and 41 control subjects. They reported that the SLE group had a significant reduction in aerobic fitness (p<0.001), exercise capacity (p<0.001) and muscle strength (p<0.001) compared to control subjects.\(^{85}\) Individuals with SLE also scored lower on multiple levels of the SF-36, specifically: physical function (p=0.001), general health (p<0.001), social function (p<0.001) and pain (p<0.001).\(^{85}\) Fatigue severity was found to be significantly higher in the SLE group (p<0.001).\(^{85}\) They looked at various measures of aerobic capacity and individuals with SLE. The subjects in the SLE group had a significantly lower VO2 peak (p<0.001), lower minute ventilation (p<0.001), lower respiratory exchange ratio (p=0.006) and achieved a lower maximum heart rate (p<0.001) on treadmill exercise testing compared to control subjects.\(^{85}\) They also demonstrated lower submaximal oxygen uptake (p=0.05) compared to the control group.\(^{85}\) Resting lung function, as measured by FEV1 < 75% and FVC < 75% of predicted, was reduced in 16-19% of the SLE subjects respectively.\(^{85}\)

Ramsey-Goldman et al.\(^{86}\) looked at the effect of exercise in individuals with SLE. They examined the effects of aerobic exercise and ROM/strengthening on measures of fatigue,
functional status, disease activity, cardiovascular fitness and strength in individuals with SLE. They reported that both aerobic exercise and ROM/strengthening programs were safe with no significant increase in disease activity index noted. Both exercise groups showed some improvement in fatigue, functional status, exercise tolerance and muscle strength.\textsuperscript{86}

Robb-Nicholson and Daltroy reported that individuals with SLE performed at only 45\% of expected aerobic capacity at baseline.\textsuperscript{87} After an 8 week aerobic program, individuals with SLE increased aerobic capacity by 19\%, and 70\% reported "more energy and improved psychological well being".\textsuperscript{87} In a study by Keyser and associates, 18 subjects with SLE and 16 sedentary control subjects underwent a treadmill test to determine aerobic capacity.\textsuperscript{88} Peak VO2 was significantly lower in the SLE group (p<0.0006) compared to the control group and lower than age/sex expected values (p<0.0001).\textsuperscript{88} Peak oxygen uptake was compared to what is required for performance of instrumental activities of daily living (IADL). The control subjects’ peak oxygen uptake was significantly higher (p<0.0005) than what is needed for IADL performance.\textsuperscript{88} The peak oxygen uptake for the subjects in the SLE group was similar to the upper level requirements for IADL.\textsuperscript{88} The authors proposed that this left little reserve for participation in occupational and recreational activities.\textsuperscript{88} Rosenilda et al\textsuperscript{89} looked at the effects of a supervised 12-week exercise program on the aerobic capacity of 60 women with SLE. The women were divided into 2 groups; training group (n=41) and non-exercise group (n=19). After a 12 week supervised exercise program the training group showed a significant improvement in aerobic capacity (p<0.001).\textsuperscript{89} The training group also demonstrated improvement in measures of depression p<0.001 and quality of life (p<0.01).\textsuperscript{89}
SLE and Physical Therapy

Because of its varied clinical presentation, widespread system involvement and ability to affect individuals across the life span, PTs may encounter SLE in many practice settings. SLE is difficult to diagnose because of its diverse clinical presentation and fluctuation of symptoms. The Lupus Foundation of America reported that individuals with SLE have symptoms for at least 4 years and visited on the average 3 doctors before receiving a diagnosis of SLE.90 The United States Department of Health and Human Services National Institutes of Health has published common signs and symptoms of SLE (Table 6).91

PTs should be aware of the symptoms of SLE during the patient screening process. PTs may be involved with patients with SLE as a primary diagnosis or as a co-morbid condition. It is important for the PT to understand the disease process and its effects on the various systems of the body. The increased risk of cardiac and neurological events in individuals with SLE needs to be considered when planning interventions. In addition, the PT needs to have an understanding of the cardiac, renal, hematological, GI and neurological issues that may interfere with physical therapy interventions. Physical therapy interventions can address the various impairments that may be present and assist in modifying risk factors for cardiac and neurological events. Exercise and aerobic conditioning have been shown to have positive effects on both physical and physiological measures. As experts in movement science, PTs can be instrumental in designing exercise programs.

Fernado and Isenberg suggested referral to physical therapy for management of fatigue, pain, mobility impairments and musculoskeletal problems.92 Sutton, a registered nurse, described the role of the physical therapist as a means to assist an individual with SLE to become or remain functionally independent and treat musculoskeletal problems through
evaluation, exercise, education, recreational exercise and aerobic conditioning. Crofts and D’Cruz, both nurses, outlined their facility’s program of education for individuals with SLE. The role of the physical therapist is to provide advice on appropriate exercise, modalities and education regarding relaxation, joint protection and healthy lifestyle behaviors. SLE was discussed in the general context of arthritis by Jette and Keysor. They recommended exercise and physical activity for individuals with arthritis in an effort to minimize impairments and functional limitations.

SLE can involve many systems of the body affecting both physical and psychological function. PTs may encounter individuals with SLE in various practice settings. There were references to SLE in medical, nursing and dental literature; however we did not find any specific reference to SLE in physical therapy literature. There were references to SLE as an “arthritic” condition, however no specifics as to the disease process and specific physical therapy interventions were found in physical therapy literature. The purpose of this survey is to examine the knowledge base of PTs in districts of New York as it pertains to SLE.

Methods:

The authors designed a survey related to SLE and PT Practice based on a review of current literature. It was divided into 3 main areas: demographic information including information regarding current and previous practice settings, questions pertaining to the methods/settings where information regarding SLE was obtained, and questions regarding the clinicians’ knowledge base as it relates to SLE. The survey was given to core faculty members of The Sage Colleges Physical Therapy Program for feedback regarding item design. This research project was approved by The Sage Colleges Institutional Review Board. A copy of the IRB approval, the survey and cover letter are included in Appendix 1.
The subjects’ names and addresses were obtained from the mailing list of the New York Physical Therapy Association. The survey and cover letter were mailed in July of 2005 to New York Physical Therapy Association members in the following districts: Catskill, Central, Eastern, Finger Lakes, Southern Tier and Western. The survey was anonymous. All identifying information was removed from the surveys and the surveys were coded by number for data analysis. SPSS version 11.5[^6] was used to analyze the data. Descriptive statistics and Mann-Whitney *U* Tests were used for analysis of the survey responses. The respondents were placed into 2 groups; novice PTs, those who graduated 5 yrs ago or less, and experienced PTs, those who graduated more than 5 yrs ago. Mann-Whitney *U* Tests were performed on selected questions comparing novice PTs and to experienced PTs. All data is reported in aggregate with no individual responses identified.

**Results:**

Eight-hundred and twenty-seven surveys were mailed to PTs who were members of the APTA in districts in New York. Two hundred and ninety eight surveys were returned for a response rate of 36%. Six of these surveys were not completed because the respondents were no longer working as PTs and thus were not used in this study (actual response rate 35.3%). The data from the remaining surveys (n=292) was analyzed using into SPSS version 11.5[^6] The data reported reflect the remaining surveys unless otherwise indicated. Frequency distributions were performed on the survey questions. Two hundred females (68.7%) and 91 males (31.3%) completed the demographic information, with a single respondent failing to complete these questions. The respondents’ ages were grouped for ease of analysis and are included in Table 7. The distribution of years of practice is provided in Table 8. A
bachelor’s degree was the most common (n=163, 55.8%) degree awarded to the respondents. The complete distribution is provided in Figure 1.

Because clinicians could have primary job responsibilities in more than one clinical setting, they were asked to indicate their primary site(s) of clinical practice. The majority of the respondents indicated that their primary site of clinical practice is “private practice” (n=103, 35.3%). Fifty-two respondents (17.8%) indicated that their current primary site of practice is an outpatient hospital based facility and 38 (13.0 %) indicated acute care as their primary site of practice. Figure 2 details information regarding clinical practice sites.

The participants were asked to indicate how they acquired information regarding SLE. Three respondents did not complete this question, thus n=289 for this response. One hundred and sixteen respondents (39.7%) indicated that they obtained information regarding SLE while involved in clinical practice. The participants were asked to indicate in which practice setting they obtained this information. The majority of the respondents were practicing in private practice (n=55, 19.1%), acute care (n=50, 17.1%) and a hospital based outpatient facility n=29, 9.9%). The complete distribution of the practice areas where SLE information was acquired can be found in Figure 3.

Twenty-five respondents (8.6%) acquired information regarding SLE through continuing education and 68 (23.3%) acquired information regarding SLE from personal experience with someone with SLE who was not a patient/client. Two hundred and four respondents (69.9%) indicated that they acquired knowledge of SLE through classroom/lab education and 28 (9.6%) indicated that information regarding SLE was obtained during student clinical education. Respondents who received information regarding SLE in their PT education were asked to rate how thoroughly the topic was covered. The majority of the respondents
(n=172, 58.9%) rated the coverage of SLE in their curriculum as minimal, 51 (17.5%) rated coverage as adequate, 3 (1%) rated coverage as thorough and 2 respondents indicated that the topic was not covered.

The remaining questions dealt with the individual respondent’s knowledge of SLE. Two hundred and sixty two subjects (93.9%) correctly identified the pathogenesis of SLE as an autoimmune disease and 12 (4.3%) indicated that they did not know the pathogenesis of SLE. The pathogenesis of SLE was incorrectly indicated to be congenital by 1 respondent (.4%) and genetic by 4 (1.4%). Thirteen of the 292 respondents failed to answer this question, thus the number of responses is 279 for this item.

The clinicians were asked to indicate which systems of the body could be involved in SLE. The systems that were correctly indicated most often were Musculoskeletal (n=264,91.7%), Integumentary (n=234,81.3%), Immunologic (n=214,74.3%), Cardiovascular (n=195,67.7%) and Neuromuscular (n=178,61.8%). Approximately half of the respondents correctly indicated possible involvement of the Pulmonary (n=155,53.8%), Genitourinary (n=70,24.3%), Renal (n=139,48.3%) and Hematologic (n=136,47.2%) systems. Approximately 8% (n=23) of the respondents did not know which systems could be involved. Even though 67.7% (n=195) of the respondents correctly identified Cardiovascular system and 53.8% (n=155) the Pulmonary system, only approximately ¼ of the respondents correctly identified chest pain and shortness of breath as clinical signs of SLE.

The participants were asked to identify the common clinical manifestations/symptoms of SLE. All the symptoms listed on the survey were common symptoms found in individuals with SLE. The 4 symptoms that were correctly recognized by the most clinicians were joint pain (89.6%), muscle pain (80.6%), fatigue (87.8%) and photosensitivity (52.8%).
Approximately half correctly recognized depression (42.4%) and fever (42.4%) as common clinical manifestations. Cardiovascular, pulmonary and neurological symptoms were not as frequently recognized, with chest pain correctly recognized by 22.2%, shortness of breath correctly recognized by 29.2% and seizures correctly recognized by 13.9% of the respondents. Figure 4 outlines the percentage of correct responses for each symptom.

The participants were asked in which physical therapy practice patterns (Cardiovascular/Pulmonary, Integumentary, Musculoskeletal, Neuromuscular) could SLE be classified. This question was completed by 286 of the respondents. One hundred and thirty-three (46.5%) correctly identified the Cardiovascular/Pulmonary, 178 (62.2%) correctly identified the Integumentary practice pattern, 223 (78%) correctly identified the musculoskeletal practice pattern and 135 (47.2) accurately identified the Neuromuscular pattern. Forty-four individuals (15.4%) indicated that they did not know in which physical therapy practice patterns SLE could be classified.

The final question asked the participants to rate their knowledge of SLE by indicating one of the following: thorough knowledge of SLE, adequate knowledge of SLE, minimal knowledge of SLE or no knowledge of SLE. Nine respondents (3.1%) failed to answer this question. Of the remaining 283 who completed this question, the majority (n=212, 74.9%) rated their knowledge base regarding SLE as minimal, 55 (19.4%) reported having an adequate knowledge base, 14 (4.9%) reported having no knowledge of SLE and 2 respondents (.7%) reported having thorough knowledge pertaining to SLE. Table 9 summarizes the respondents’ self assessments.

Mann-Whitney U Tests were used to compare the responses of novice PTs and experienced PTs on items relating to therapists’ knowledge base as it relates to SLE. A
significance difference between the 2 groups was found in selected responses to items relating to how SLE knowledge was acquired, the clinical presentation of SLE, and the association of SLE to physical therapy practice patterns. There was a significant difference between the groups in the area of acquisition of SLE knowledge. Novice PTs were more likely than experienced PTs to have obtained information regarding SLE through clinical practice (p=.001) and continuing education (p=.007). Experienced PTs were more likely to have obtained information through student classroom/lab education (p=.003). When looking at recognition of symptoms of SLE, the novice PTs recognized joint pain (p=.022) and muscle pain (p=.020) more often than experienced PTs. Question 14 looked at the classification of SLE in PT practice patterns. The only significant difference between the groups was that novice PTs more often placed SLE into the Musculoskeletal Practice Pattern (p=.008) than did the experienced PTs.

Discussion

We analyzed 292 surveys for a response rate of 35.3%. Portney and Watkins stated that a 60-80% response rate would be considered excellent and a more realistic response rate would be 30-60%. Our response rate is within the expected realistic rate as described by Portney and Watkins. When asked how information regarding SLE was obtained, 69.9% of the respondents indicated that information was obtained through classroom/lab education, however the majority of the respondents rated the coverage of SLE in their curriculum as minimal. With PT education moving to the DPT and an expansion of educational information being provided, we had assumed that the more recent graduates would have been more likely to obtain information regarding SLE in the PT curriculum than those who graduated more than 5 years ago, this was not the case. Novice PTs reported obtaining their SLE information
in clinical practice while experienced PTs obtained it in the classroom/lab components of their professional education. Although most respondents rated their knowledge of SLE as minimal, they were able to recognize some of the most common symptoms of SLE including joint pain, muscle pain, fatigue and photosensitivity. This also corresponds to most respondents being able to correctly identify the musculoskeletal practice pattern. The novice PTs were more likely to recognize the musculoskeletal manifestations of SLE and were more likely to place SLE in the Musculoskeletal Practice Pattern than experienced PTs. The questions pertaining to the manifestations of SLE were general and respondents may have used deduction to answer the question if they had a general idea that SLE is an arthritic condition.

There was a discrepancy between the respondents correctly categorizing SLE in the Cardiovascular and Pulmonary Practice Pattern (46.5%) with only 22% correctly recognizing chest pain and shortness of breath as clinical manifestations. When looking at Figure 4, symptoms correctly selected by clinicians, only 4 of the 13 clinical symptoms of SLE were correctly identified by 50% or more of the respondents. Joint and muscle pain were 2 of the 4 of the most frequently recognized areas and are typically treated in PT. The remaining 9 symptoms were recognized by fewer than 50% of the respondents with shortness of breath and chest pain being recognized by fewer than 20% of respondents. Approximately 8% of respondents reported that they did not know the symptoms of SLE.

SLE can be seen across the lifespan and PTs could encounter individuals with SLE in a variety of clinical settings. In our study the majority of the subjects reported their primary site of clinical practice as private practice, outpatient hospital based practice or acute care. In our study 39.7% of the respondents indicated that they obtained information regarding SLE
through clinical practice. When comparing the novice PTs to the experienced PTs, the
novice PTs were more likely to obtain information through clinical practice. We thought that
the opposite would be true since the experienced PTs had been working longer and the
possibility of having cared for patients with SLE in multiple clinical settings was presumed
to be higher.

In our literature review we did not find any specific reference to SLE in the physical
therapy literature. The primary sources of information were medical and nursing literature.
SLE has been a topic of continuing professional development in both nursing and medical
practice.\textsuperscript{2, 25, 98} Methods for continuing education related to SLE include online tutorials,
journal-based clinical updates, and manuscripts.\textsuperscript{2, 25, 98} Other methods of continuing SLE
education could include privately sponsored courses or "in house" presentations. A case
could be made for any of these venues. We feel that information regarding SLE should also
be included in the tDPT curriculum since there appears to be gaps in the knowledge base of
practicing clinicians as it relates to SLE. Further areas of study could examine various
methods of instruction and their effectiveness.

There are limitations to our study. We surveyed only districts in regions of New York
State thus limiting its ability to be generalized. We asked the participants if they acquired
information regarding SLE through continuing education however we did not ask specifically
the method or type of continuing education. A future study could look at information
pertaining to SLE in physical therapy curriculum. We attempted to address this issue in an
electronic survey that was sent to physical therapy programs in the United States; due to the
poor response rate no significant data could be reported. The poor response rate may have
been related to the method of distribution.
In this study, we had 3 respondents who reported coverage of SLE content as “thorough”. We looked at the graduation dates and the model of curricular delivery to determine if there were trends. Due to the small number no significant information could be reported.

Conclusion

SLE can affect people of all ages and can affect multiple organ systems. PTs may be involved in the care of these individuals in varied clinical settings. As independent practitioners, PTs need to be able to recognize symptoms of SLE in order to make clinical differential diagnoses and to be able to refer the patient/client to the appropriate medical practitioners. PTs will also need to provide and/or direct the patient/client to the appropriate educational resources. PTs will need to design a plan of care that is comprehensive and addresses the impairments and functional limitations that are presented, but in addition be aware of the possibility of underlying organ involvement and possible effects on PT intervention. In order to be able to achieve these goals the therapist must have an adequate knowledge of the disease and its effects. It is the researchers’ belief that there needs to be a mechanism for delivery of material regarding SLE in both PT curriculum and in continuing education for practicing clinicians. Further studies regarding the methods of education delivery are needed.
References


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96. SPSS. Version 11.5. Chicago, IL; 2002.


Tables:

Table 1. Criteria for Diagnosis of SLE *

<table>
<thead>
<tr>
<th>Eleven Criteria for Diagnosis of SLE</th>
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<tbody>
<tr>
<td>1. Malar Rash</td>
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<td>2. Discoid Rash</td>
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<td>3. Photosensitivity</td>
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<tr>
<td>4. Oral or Nasal</td>
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<tr>
<td>Pharyngeal Ulceration</td>
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<td>5. Non-erosive arthritis</td>
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<td>- 2 or more peripheral joints</td>
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<td>6. Pleuritis or Pericarditis</td>
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<td>7. Renal Disorder</td>
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<tr>
<td>- proteinuria</td>
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<tr>
<td>- cellular casts</td>
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<td>8. Neurologic disorders</td>
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<td>- headaches</td>
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<td>- seizures</td>
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<td>- psychosis</td>
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<td>9. Hematologic Disorders</td>
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<td>- thrombocytopenia</td>
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<tr>
<td>- positive findings of</td>
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<tr>
<td>antiphospholipid antibodies</td>
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<td>11. Positive Antinuclear Antibody</td>
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*Based on American College of Rheumatology AD HOC Committee on Systemic Lupus Erythematosus 19
Table 2. Traditional and SLE Related Risk Factors for Development of Osteoporosis*

<table>
<thead>
<tr>
<th>Traditional Risk Factors</th>
<th>SLE Related Risk Factors</th>
</tr>
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<tbody>
<tr>
<td>Female sex</td>
<td>Active disease or frequent SLE flares</td>
</tr>
<tr>
<td>Women age &gt;65 years</td>
<td>- Early onset may prevent reaching optimal peak bone mass</td>
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<tr>
<td>White or Asian</td>
<td>- Symptoms related to SLE may prevent physical activity</td>
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<tr>
<td>Low body weight</td>
<td>- Hypogonadism due to active SLE</td>
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<tr>
<td>Personal or maternal history of fragility fracture</td>
<td>Sun exposure and non-use of sunscreens</td>
</tr>
<tr>
<td>Early menopause (&lt;40 years of age)</td>
<td>Chronic glucocorticoid use (&gt;3 months)</td>
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<tr>
<td>Recurrent falls</td>
<td>Therapy related premature menopause</td>
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<td>Low physical activity</td>
<td>Therapy associated low bone mineral density</td>
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<td>Current smoking</td>
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<tr>
<td>Low dietary calcium or vitamin D</td>
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<tr>
<td>Excessive alcohol use (&gt;2 drinks/day)</td>
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</tbody>
</table>

*Based on Lee and Ramsey-Goldman

Table 3. Lupus Nephritis Classification Based on Glomerular Lesions by WHO *

<table>
<thead>
<tr>
<th>Disease Classification</th>
<th>Characteristics of the Class</th>
</tr>
</thead>
<tbody>
<tr>
<td>Class I</td>
<td>Normal</td>
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<tr>
<td>Class II</td>
<td>Minimal Change/Mesangial Changes</td>
</tr>
<tr>
<td></td>
<td>Mild Mesangial Change and/or Proliferation</td>
</tr>
<tr>
<td>Class III</td>
<td>Focal Glomerular Proliferation</td>
</tr>
<tr>
<td>Class IV</td>
<td>Diffuse Glomerular Proliferation</td>
</tr>
<tr>
<td>Class V</td>
<td>Membranous Glomerulonephritis</td>
</tr>
<tr>
<td>Class VI</td>
<td>Chronic Glomerular Sclerosis</td>
</tr>
</tbody>
</table>

*Based on World Health Organization Classification

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Table 4. Neuropsychiatric Syndromes in SLE

<table>
<thead>
<tr>
<th>CNS</th>
<th>PNS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aseptic Meningitis</td>
<td>Acute Inflammatory Demyelinating</td>
</tr>
<tr>
<td></td>
<td>Polyradiculoneuropathy</td>
</tr>
<tr>
<td>Cerebrovascular Syndrome</td>
<td>Autonomic Disorders</td>
</tr>
<tr>
<td>Demyelinating Syndrome</td>
<td>Mononeuropathy (single/multiplex)</td>
</tr>
<tr>
<td>Headache</td>
<td>Myasthenia Gravis</td>
</tr>
<tr>
<td>Movement Disorders</td>
<td>Neuropathy</td>
</tr>
<tr>
<td>Myelopathy</td>
<td>Polyneuropathy</td>
</tr>
<tr>
<td>Seizure Disorder</td>
<td></td>
</tr>
<tr>
<td>Acute Confusional State</td>
<td></td>
</tr>
<tr>
<td>Anxiety Disorder</td>
<td></td>
</tr>
<tr>
<td>Cognitive Dysfunction</td>
<td></td>
</tr>
<tr>
<td>Mood Disorder</td>
<td></td>
</tr>
<tr>
<td>Psychosis</td>
<td></td>
</tr>
</tbody>
</table>

ACR ad HOC Committee on Neuropsychiatric Lupus Nomenclature

Table 5. Pulmonary Manifestation in Children with SLE

<table>
<thead>
<tr>
<th>Pulmonary Manifestations in Children with SLE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pleuritis</td>
</tr>
<tr>
<td>Pulmonary Fibrosis</td>
</tr>
<tr>
<td>Pulmonary Hypertension</td>
</tr>
<tr>
<td>Acute Pneumonitis</td>
</tr>
<tr>
<td>Alveolar Hemorrhage</td>
</tr>
<tr>
<td>Respiratory Muscle Myopathy</td>
</tr>
</tbody>
</table>

*Based on Information from Boon S., McCurdy D.
Table 6. Common Symptoms of SLE as Described by NIH *

<table>
<thead>
<tr>
<th>Signs and Symptoms of SLE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Painful swollen joints</td>
</tr>
<tr>
<td>Muscle pain</td>
</tr>
<tr>
<td>Unexplained fever</td>
</tr>
<tr>
<td>Red rashes</td>
</tr>
<tr>
<td>most common on the face</td>
</tr>
<tr>
<td>Chest pain upon deep breathing</td>
</tr>
<tr>
<td>Sensitivity to sun</td>
</tr>
<tr>
<td>Swelling in legs or around eyes</td>
</tr>
<tr>
<td>Mouth ulcers</td>
</tr>
<tr>
<td>Swollen glands</td>
</tr>
<tr>
<td>Extreme fatigue</td>
</tr>
<tr>
<td>Pale or purple fingers from cold or stress</td>
</tr>
</tbody>
</table>

*Based on United States Department of Health and Human Services National Institutes of Health

Table 7. Respondent Demographic Information (n=291)

<table>
<thead>
<tr>
<th>AGE</th>
<th>Number of Respondents</th>
<th>AGE</th>
<th>Number of Respondents</th>
</tr>
</thead>
<tbody>
<tr>
<td>21-25 yrs</td>
<td>27</td>
<td>51-55 yrs</td>
<td>36</td>
</tr>
<tr>
<td>26-30 yrs</td>
<td>45</td>
<td>56-60 yrs</td>
<td>18</td>
</tr>
<tr>
<td>31-35 yrs</td>
<td>42</td>
<td>61-65 yrs</td>
<td>4</td>
</tr>
<tr>
<td>36-40 yrs</td>
<td>25</td>
<td>66-70 yrs</td>
<td>1</td>
</tr>
<tr>
<td>41-45 yrs</td>
<td>45</td>
<td>71-75 yrs</td>
<td>1</td>
</tr>
<tr>
<td>46-50 yrs</td>
<td>47</td>
<td>76-80 yrs</td>
<td>1</td>
</tr>
</tbody>
</table>
Table 8. Number of Years Practicing as a Physical Therapist (n=283)

<table>
<thead>
<tr>
<th>Number of years</th>
<th>Number of Respondents</th>
</tr>
</thead>
<tbody>
<tr>
<td>Less than 1 year</td>
<td>n = 18 (6.4%)</td>
</tr>
<tr>
<td>1-5 yrs</td>
<td>n = 53 (18.7%)</td>
</tr>
<tr>
<td>&gt;5-10 yrs</td>
<td>n = 47 (16.6%)</td>
</tr>
<tr>
<td>&gt;10-15 yrs</td>
<td>n = 30 (10.6%)</td>
</tr>
<tr>
<td>&gt;15-20 yrs</td>
<td>n = 33 (11.7%)</td>
</tr>
<tr>
<td>&gt;20-25 yrs</td>
<td>n = 38 (13.4%)</td>
</tr>
<tr>
<td>&gt;25-30 yrs</td>
<td>n = 35 (12.4%)</td>
</tr>
<tr>
<td>&gt;30-35 yrs</td>
<td>n = 17 (6.0%)</td>
</tr>
<tr>
<td>&gt;35-40 yrs</td>
<td>n = 9 (3.2%)</td>
</tr>
<tr>
<td>&gt;40-45 yrs</td>
<td>n = 0 (0%)</td>
</tr>
<tr>
<td>&gt;45-50 yrs</td>
<td>n = 2 (0.7%)</td>
</tr>
<tr>
<td>&gt;50 yrs</td>
<td>n = 0 (0%)</td>
</tr>
</tbody>
</table>

Table 9. Therapist Self-rating of SLE Knowledge (n = 292)

<table>
<thead>
<tr>
<th>Self-rating of SLE Knowledge</th>
<th>Percentage of Respondents</th>
</tr>
</thead>
<tbody>
<tr>
<td>Thorough</td>
<td>0.7%</td>
</tr>
<tr>
<td>Adequate</td>
<td>19.4%</td>
</tr>
<tr>
<td>Minimal</td>
<td>74.9%</td>
</tr>
<tr>
<td>No knowledge</td>
<td>4.9%</td>
</tr>
</tbody>
</table>

No response from 3.1% of the therapists
Figures:

Figure 1. Distribution of degrees earned by respondents (n = 291)

Figure 2. Clinical sites of practice
Figure 3. Clinical practice sites in which SLE information was acquired

Figure 4. Symptoms correctly selected by clinicians
June 20, 2005

Janet Hakey-Brusgul and April Friedman  
Department of Physical Therapy  
The Sage Colleges  
45 Ferry Street  
Troy, NY 12180

IRB PROPOSAL #049-05  
Reviewer: Samuel W. Hill, Chair

Dear Ms Hakey-Brusgul and Ms Friedman:

The Institutional Review Board has reviewed your application and has approved your project entitled “Systemic Lupus Erythematosus Information in Physical Therapy Practice in Districts of New York State”. Good luck with your research.

Please refer to your IRB Proposal number whenever corresponding with us whether by mail or in person.

The Sage IRB Committee has approved your project for one year from the date of this letter. Should your research extend beyond one year, you must reapply to the Institutional Review Board.

We look forward to a report of your results at the Sage Graduate School Research Symposium.

Sincerely,

Sam Hill /nm

Samuel W. Hill, PhD  
Chair, IRB  

SWH/nm
Appendix 1a

**Systemic Lupus Erythematosus in Physical Therapy Practice**

Please complete the following as it relates to Systemic Lupus Erythematosus (SLE)

**Demographics of individual completing the survey:**

1. Age: 

2. Sex
   ( ) Female ( ) Male

**Program:**

3. Year of graduation from an accredited Physical Therapy Program 

4. From what physical therapy program did you graduate?
   

5. What degree was awarded upon graduation from your physical therapy program?
   ( ) Certificate ( ) BS ( ) BS/MS ( ) MA/MS/MPT
   ( ) DPT ( ) Other 

6. How many years have you been a practicing physical therapist? 

7. In what clinical setting(s) do you practice?
   Please place the number (1) next to your current primary site(s) of practice and a (2) next to any other previous sites of practice.
   ( ) Acute care hospital
   ( ) Sub acute/rehab hospital inpatient
   ( ) Health system or hospital based outpatient facility or clinic
   ( ) Private outpatient office or groups practice
   ( ) Skilled nursing facility/extended care facility/intermediate care
   ( ) Patient’s home/home care
   ( ) School system (pre-school/primary/secondary)
   ( ) Academic institution (post secondary)
   ( ) Health and wellness facility
   ( ) Research center
   ( ) Industry
   ( ) Other, please specify 

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Systemic Lupus Erythematosus in Physical Therapy Practice

The following questions are about your knowledge of SLE

8. How did you acquire your knowledge of Systemic Lupus Erythematosus?
   ( ) Student classroom/lab education
   ( ) Student clinical education
   ( ) Clinical practice
   ( ) Continuing education
   ( ) Personal experience with someone with SLE who was not a patient/client
   ( ) Other

9. If you received information regarding SLE in your physical therapy education program please answer the following. If you did not receive information regarding SLE in your physical therapy program please skip to question #9.
   Please rate how thoroughly your physical therapy program covered the topic of SLE.
   ( ) Thorough coverage of the topic
   ( ) Adequate coverage of the topic
   ( ) Minimal coverage of the topic
   ( ) Topic not covered

10. If you have acquired information regarding SLE in your clinical practice, in which type of setting did you obtain information? If you did not acquire information regarding SLE in the clinical setting please skip to question #10.
   ( ) Acute care hospital
   ( ) Sub acute/rehab hospital inpatient
   ( ) Health system or hospital based outpatient facility or clinic
   ( ) Private outpatient office or groups practice
   ( ) Skilled nursing facility/extended care facility/intermediate care
   ( ) Patient’s home/home care
   ( ) School system (pre-school/primary/secondary)
   ( ) Academic institution (post secondary)
   ( ) Health and wellness facility
   ( ) Research center
   ( ) Industry
   ( ) Other, please specify

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Systemic Lupus Erythematous in Physical Therapy Practice

11. Which of the following is the pathogenesis of SLE?
   ( ) Autoimmune disease
   ( ) Congenital disease
   ( ) Community acquired disease
   ( ) Contagious disease
   ( ) Genetic disease
   ( ) Do not know

12. Which of the following systems could be involved in SLE?
    (Please check all that apply)
    ( ) Cardiovascular
    ( ) Gastrointestinal
    ( ) Genitourinary
    ( ) Hematologic
    ( ) Hepatic
    ( ) Immunologic
    ( ) Integumentary
    ( ) Lymphatic
    ( ) Musculoskeletal
    ( ) Neuromuscular
    ( ) Pulmonary
    ( ) Renal
    ( ) Do not know

13. Which of the following are common clinical presentations/symptoms of SLE?
    (Please check all that apply)
    ( ) Anemia
    ( ) Chest pain
    ( ) Depression
    ( ) Edema
    ( ) Fatigue
    ( ) Fever
    ( ) Joint pain
    ( ) Muscle pain
    ( ) Oral ulcers
    ( ) Photosensitivity
    ( ) Raynaud's
    ( ) Seizures
    ( ) Shortness of breath
    ( ) Do not know

14. In which of the following physical therapy practice patterns could SLE be classified?
    (Please check all that apply)
    ( ) Cardiovascular/Pulmonary
    ( ) Integumentary
    ( ) Musculoskeletal
    ( ) Neuromuscular
    ( ) Do not know

15. How would you rate your knowledge of SLE?
    ( ) Thorough knowledge of SLE
    ( ) Adequate knowledge of SLE
    ( ) Minimal knowledge of SLE
    ( ) No knowledge of SLE

Thank you for your participation.
Please return the survey in the stamped, addressed envelope provided.
Appendix 1b

Dear Physical Therapist,

We are contacting you to ask for your participation in a research study related to Systemic Lupus Erythematosus (SLE). The purpose of this study is to determine the knowledge level of practicing clinicians in the area of SLE. The intent of the researchers is to determine the need for additional information regarding SLE in the expanding field of physical therapy. The cover letter and survey were approved by the IRB of The Sage Colleges on 6-20-05, IRB # 049-05 for distribution.

Your name and address were obtained through the New York Physical Therapy Association. Information gathered that could identify you will be removed and the completed surveys will be retained in a file cabinet in the locked office of Janet Hakey-Brusgul at the Sage Colleges and used exclusively for this research project. Anonymity of your responses will be assured by assignment of case numbers to surveys for data analysis. All data gathered and presented will be reported in aggregate. No single individual will be identified in any project reports.

We ask that you complete the survey and return it by September 1, 2005, in the enclosed stamped, addressed envelope. The survey should take approximately 10 minutes to complete. Submission of the survey constitutes your consent to participate in this study.

There are no anticipated risks to participants. The benefits of participation include advancement of the scholarship of teaching within the physical therapy field. For questions or a synopsis of findings please contact either April Friedman at frieda@sage.edu or Janet Hakey-Brusgul at hakeyj@sage.edu.

Thank you for your participation in this project.

April Friedman, PT MS
Assistant Professor
The Sage Colleges

Janet Hakey-Brusgul, PT MS, CSCS
Assistant Professor
The Sage Colleges