2019

Relationships Between SLE Disease Activity and Damage, Depression and Work Productivity Impairment in the Georgians Organized Against Lupus Study

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Abstract

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by

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MPH, University of Illinois, Chicago, 2015

MS, DePaul University, 2003

BS, University of Maryland, College Park, 1997

Doctoral Study Submitted in Partial Fulfillment

of the Requirements for the Degree of

Doctor of Public Health

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Abstract

Systemic lupus erythematous (SLE) is an autoimmune and inflammatory disease that can affect all organs of the body. The purpose of this quantitative cross-sectional study was to examine SLE-related issues associated with depression and work-productivity impairment, and to assess if depression mediated the relationship between SLE disease activity and damage and work-productivity impairment. Participants were 257 residents of the state of Georgia in the United States with SLE and were recruited from the Georgians Organized Against Lupus study. Bandura's social cognitive theory was the guiding theoretical framework of the study. Findings showed that the majority of participants worked full time (78.2%), identified as Black (72.8%), female (94.2%), above poverty level (77.4%), and had private health insurance (70.0%). Mean and median score results indicated that participants missed, on average, slightly less than half a day of work every 7 days, and had mild-to-moderate levels of work productivity impairment. Mean and median scores showed that participants reported mild-to-moderate levels of SLE disease activity and damage and depression. Linear regression results revealed significant relationships between SLE activity and damage and work productivity impairment. Hierarchical linear regression for mediation findings indicated that depression partially mediated the relationship between SLE disease activity and damage and work productivity impairment. The findings from this study might help to increase stakeholder awareness of SLE disease activity and damage and SLE effects on depression and work functioning.

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Dedication

This capstone is dedicated to the males in my life of which there are three that live with me. My husband Lenin Cuevas, my constant companion who has encouraged me for more than 25 years to continue pursuing my education throughout my life and has always been the biggest supporter of my career development. To my two sons, Nicholas and Alexander: This is for you two because I know the both of you are very proud of your mom for completing her studies, allowing more time to be with you now particularly as you are growing into young men. The final person this capstone is dedicated to is my grandfather, Dr. Enrique Canessa. One of the great public health professionals in Latin America in the past century. Although he passed away in 2011, I know he would be very proud of completion of my doctoral studies as he was the only other person in my family with a DrPH degree.

Acknowledgments

I want to acknowledge several individuals during my Walden doctoral journey. First, my chair Dr. Aaron Mendelsohn for providing guidance for the DrPH capstone from the proposal to the dissertation defense stage. Second, to those who provided the data including Dr. Cristina Drenkard and Charmayne Dunlop-Thomas at Emory University, for facilitating the agreement to access the GOAL dataset. Dr. Drenkard also provided review and recommendations on the GOAL data design and analysis sections. Additionally, my mentor Dr. Rosalind Ramsey-Goldman at Northwestern University provided generous time for review of lupus content and was a secondary lupus expert for the capstone project of which her expertise was invaluable throughout this final process.

For my colleagues at Northwestern University, including my primary lupus research study team, thank you for your support for the past four years. For my colleagues within the public health field, I appreciate the encouragement to further research in an understudied chronic disease public health specialty area. For my fellow Latina social science investigators, I found your mentorship invaluable as few become academicians.

To my close inner circle of Latinx friends I reserve my final acknowledgements. This includes Dr. Beatriz Tapia my best friend for almost twenty years, who was always supportive of my decision to continue my studies. Also, to my very good friend and associate Dr. Louis Martos and his wife Dr. Sorelly Gil-Martos for their encouragement particularly during the last challenging dissertation phase of the capstone.

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Section 1: Foundation of the Study and Literature Review

Introduction

Systemic lupus erythematosus (SLE or lupus) is a heterogeneous, autoimmune, inflammatory disease that can involve practically any organ of the body (Lim & Drenkard, 2015). One notable feature of SLE is that the signs and symptoms of the disease can vary in terms of presentation and severity (Alarcón, 2008; Askanase, Shum, & Mitnick, 2013). The disease can place substantial behavioral burden on affected individuals, particularly with regards to their ability to work and perform activities while at work, resulting in higher levels of work productivity impairment (Al Dhanhani et al., 2014; Baker & Pope, 2009; Cosatti et al., 2017; Drenkard et al., 2014a; Garris, Oglesby, Sulcs, & Lee, 2013). Depression, common among patients with SLE, can further contribute to absenteeism and poor work productivity (Auberbach & Beckerman, 2012; Shen, Tang, & Feng, 2013). Although scholars have noted significant associations between SLE damage and activity and work productivity impairment (Al Dhanhani et al., 2014; Cosatti et al., 2017; Drenkard et al., 2014a; Garris et al., 2015) and depression (Jordan et al., 2018; Zakeri, Shakiba, & Narouie, 2012), no scholar has examined the potential mediating effects of depression on the relationship between SLE damage and activity and work productivity impairment.

In this study, I examined the direct relationship between SLE disease activity (i.e., range of disease activity from mild to severe) and damage (i.e., irreversible organ damage) and work productivity impairment. I also assessed if depression mediated the relationship between SLE disease activity and damage and work productivity

impairment. I analyzed data from 257 Georgians Organized against Lupus (GOAL) study participants who were in the workforce at the time of data collection. The GOAL study used gold standard measures: (a) the Systemic Lupus Activity Questionnaire (SLAQ; Karlson et al., 2003), which measured SLE disease activity, (b) the self-administered Brief Index of Lupus Damage (SA-BILD; Drenkard et al., 2014a), which assessed SLE disease damage; (c) the Patient Health Questionnaire (PHQ-9; Kroenke, Spitzer, & Williams, 2001), a measure of depression; and (d) the Work Productivity and Activity Impairment instrument (WPAI; Reilly, Zbozek, & Dukes, 1993), which assessed work productivity impairment. This study is expected to contribute to positive social change by providing clarity on how SLE disease activity and damage and depression, singly and collectively, impact work productivity among persons with SLE.

In this section, the problem statement, the study purpose, and the research questions and hypotheses will be examined. The theoretical foundation, Bandura's (1986) social cognitive theory (SCT), is described, followed by a discussion of the nature of the study and the analytical approach to be used. The literature search strategy will be discussed to illustrate the databases used to identify relevant articles from the scientific literature to inform the present study. Next, the assumptions, scope, and delimitations for executing this study and analyzing the data will be discussed. Finally, the section will be concluded with details on the significance of the research and a summary of the chapter.

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Problem Statement

SLE is a complex autoimmune disease that has significant clinical, behavioral, and societal implications (Kheirandesh, Tareh-Fagazi, & Paragomi, 2015; Lim et al., 2014). Work productivity impairment is an ongoing concern among individuals with SLE, as the disease creates challenges in maintaining a job and contributes to reduce work productivity (Drenkard et al., 2014a, 2014b; Gordon et al., 2013a; Utset et al., 2014; Yelin et al., 2007). Because the disease activity changes due to intensity and severity, the degree of unpredictability can cause SLE sufferers to either leave a job earlier than expected or work less than fulltime (Al Dhanhani et al., 2014; Baker & Pope, 2009; Cosatti et al., 2017; Drenkard et al., 2014a; Garris et al., 2015). Due to the complexity of the disease, such as disease activity and organ damage, further research is needed to understand the implications of work productivity impairment on individuals with SLE (Drenkard et al., 2014a).

Depression can exacerbate SLE symptoms and contribute to organ damage (Greco, Carr, & Sattar, 2009; Jorge et al., 2017), increase perceptions of pain and illness (Nowicka-Sauer et al., 2018), and result in poor disease management practices (Julian, Yelin, & Yazdany, 2009; Nowicka-Sauer et al., 2018) and can impede socialization and the capability to cope with the disease (Auberbach & Beckerman, 2012; Mazzoni & Cicognani, 2011). Depression can be caused or influenced by work loss or difficulties in working, which creates further obstacles for ongoing treatment and care, and may impact health insurance coverage (Jordan et al., 2018; Shen et al., 2013). Individuals with SLE often seek support for depressive symptoms from primary care providers to reduce

associated risks of suicide ideation and increased medical management of their treatment regimen (Zakeri et al., 2012). Between 30% and 47% of SLE patients have clinical depression (Bachen, Chesney, & Criswell, 2009).

Purpose of the Study

This quantitative study, using a cross-sectional research design, was conducted with a sample of 257 GOAL participants who were employed at the time of data collection. The study had a three-fold purpose. First, I examined aspects of work productivity impairment among the GOAL participants. Second, I determined if significant relationships existed between SLE disease activity and damage and work productivity impairment. Third, I examined if depression mediated the relationships between SLE disease activity and damage and work productivity impairment. The study included an examination as to whether key demographic (e.g., ethnicity, gender, marital status, years of education), healthcare (e.g., type of health insurance), and health (i.e., SLE disease duration) were significantly associated with work productivity impairment.

Research Questions and Hypotheses

This study had three research questions. The first research question was descriptive, and as such, did not have associated null and alternative hypotheses (Salkind, 2010). The second and third research questions were analytical and, therefore, had associated null and alternative hypotheses. These are presented below.

Research Question 1 (RQ1): What are the SLE-related issues (activity and damage) that impact work productivity impairment among GOAL cohort participants?

Research Question 2 (RQ2): Is SLE disease activity associated with work productivity impairment among GOAL participants (2.1), and does depression mediate the relationship (2.2)?

$H_0$2a: There is no statistically significant association between SLE disease activity and work productivity impairment among GOAL participants.

H_a2a*:* There is a statistically significant association between SLE disease activity and work productivity impairment among GOAL participants.

$H_0$2b: There is no statistically significant mediation effect of depression on the relationship between SLE disease activity and work productivity impairment among GOAL participants.

H_a2b: There is a statistically significant mediation effect of depression on the relationship between SLE disease activity and work productivity impairment among GOAL participants.

Research Question 3 (RQ3): Is SLE disease damage associated with work productivity impairment among GOAL participants (3.1), and does depression mediate the relationship (3.2)?

$H_0$3a: There is no statistically significant relationship between SLE disease damage and work productivity impairment among GOAL participants.

H_a3a: There is no statistically significant mediation effect of depression on the relationship between SLE disease damage and work productivity impairment among GOAL participants.

$H_0$3b: There is a statistically significant relationship between SLE disease damage and work productivity impairment among GOAL participants.

H_a3b: There is a statistically significant mediation effect of depression on the relationship between SLE disease damage and work productivity impairment among GOAL participants.

Theoretical Foundation for the Study

The theoretical framework that informed this study and the study research questions and hypotheses was Bandura's (1986) SCT. According to SCT, learning occurs in a social context, with a dynamic and reciprocal interaction of the person, environment, and behavior. Central to SCT is the concept of reciprocal determinism (Figure 1) where behavior (B) is the function of the person (P) interacting with his or her environment (E). As depicted in Figure 1, each component of the reciprocal determinism model is further delineated into attributes. The environmental stimuli and reinforcement contingencies influence and interact with personal attributes of personality characteristics, cognitive factors, and skills to influence the nature, frequency, and intensity of behavior (Bandura, 1986). In this study, it was posited that lupus disease activity and damage are products of the person interacting with her/his environment and influence work-related behavior, specifically, work productivity impairment.

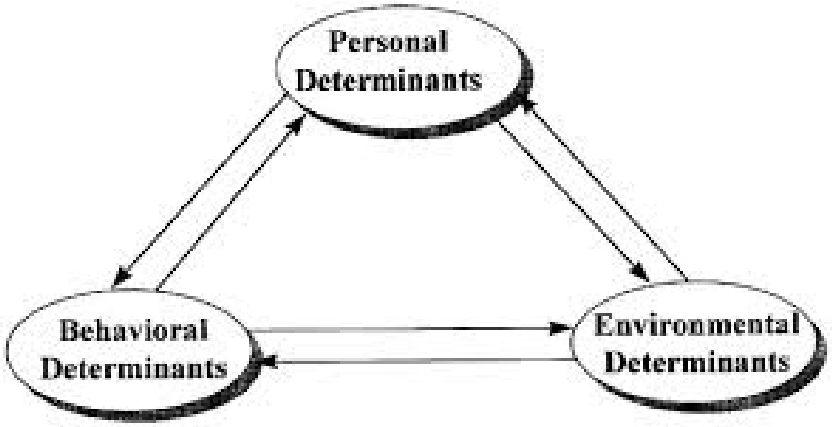

Figure 1. Bandura's social cognitive theory: Reciprocal determinism.

SCT has three overarching principles (Bandura, 1986; 2011). The first principle is observational learning, where the person observes others performing a behavior and rehearses it. Reinforcement, the second principle of SCT, pertains to the response elicited by external stimuli to the degree that the health behavior is strengthened over time. The third principle of SCT is that people are more likely to adopt behavior that is reinforced if an influential person is performing the behavior (Bandura, 1986, 2011). The unique feature of SCT is the emphasis on social influence and external and internal social reinforcement (Bandura, 1986). These past experiences influence reinforcements, expectations, and expectancies, all of which shape whether a person will engage in a behavior and reinforce the reasons a person engages in that behavior. The goal of SCT is to explain how people regulate their behavior through control and reinforcement to achieve goal-directed behavior that can be maintained over time (Bandura, 1986).

People's perceptions of their efficacy influence how individuals construct and reiterate anticipatory scenarios (Bandura, 2004, 2011). Those with high efficacy

visualize the potential for success while those who view themselves as inefficacious are more inclined to visualize failure scenarios (Bandura, 2004, 2011). High efficacy adaptation is the goal for reduction of depression among individuals with SLE (Marks, 2014). Depression impairs the ability to initiate and sustain adaptive activities and diminishes perceptions of self-efficacy (Kavanaugh & Bower, 1985; Marks, 2014; Nowicka-Sauer et al., 2018). Depression may negatively influence work self-efficacy (Nery et al., 2007; Panopalis et al., 2008; Mok, Cheung, Ho, Yu, & To, 2008). Due to the relevance of SCT to disease perceptions and mental health constructs, it is one of the most used theories to inform and guide health and medical interventions on a range of topics from chronic disease to ongoing prevention efforts (Bandura, 2004, 2005; Glanz & Bishop, 2010).

Nature of the Study

The selection of a research design for a quantitative epidemiological study entailed a "top-down" approach. The study intent and purpose, the nature of the research questions, the manipulation of the independent variable, data collection time points, and sample attributes all informed the research design selection process (Manja & Lakshminrusimha, 2014). In epidemiology, a descriptive research design is employed if the intent of the study is to describe disease occurrences and processes (Manja & Lakshminrusimha, 2014). In contrast, an analytical research design is used when the intent of the study is to examine associations between and among variables (Manja & Lakshminrusimha, 2014). The nature of the research questions–one descriptive and two analytical–required the use of both a descriptive and an analytical research design. As

the data used to address all three research questions were collected at one point in time, the study was also cross-sectional in design. Moreover, as the two analytical research questions pertained to the examination of relationships or association between naturally occurring variables, the study was nonexperimental (observational) (Creswell, 2009).

I addressed the complexities surrounding the diagnosis of SLE in a sample of individuals with SLE who resided in Georgia and participated in the GOAL study. The GOAL study is a longitudinal cohort of individuals with a validated diagnosis of SLE, primarily derived from the population-based Georgia Lupus Registry (Drenkard et al., 2013, 2014a). Funded by the Center for Disease Control and Prevention (CDC), the overarching goal of the GOAL study is to gain a better understanding of the burden of lupus and health disparities in high-risk lupus populations, including the impact of SLE disease activity and damage on patients' work and mental health outcomes. GOAL study investigators recruited study participants having a validated diagnosis of SLE from the GLR, Emory and Grady SLE clinics, and participating community rheumatologists. The purpose of the GOAL study is to better understand how SLE impacts patients' lives and to assess how covariates (e.g., income, work status, and health insurance) reflect the realities of dealing with depression (Lim & Drenkard, 2015).

This study was driven by the idea that understanding how depression impacts work productivity impairment will help provide increased awareness of SLE and lead to the development of meaningful interventions aimed at reducing depression and enhancing work productivity among SLE patients. The study was unique in that it was the first to examine the potential mediating effects of depression with regard to SLE disease activity

and damage and work productivity impairment. The study also included the role of numerous demographic (e.g., ethnicity, marital status), health-related (e.g., type of medical insurance, yearly out-of-pocket medical expenses) and health (i.e., SLE disease duration) on work productivity impairment. There have been significant associations between the study covariates and work productivity outcomes found in prior research (Cosatti et al., 2017; Drenkard et al., 2014a; Utset et al., 2015; Yelin et al., 2009).

Literature Search Strategy

The primary focus of the search strategy was to identify literature related to work productivity impairment and the prevalence of SLE, impact of the disease on morbidity and mortality outcomes, depression, and GOAL. Several key terms were used in the literature search efforts with the main key terms being lupus and minority populations, lupus and the State of Georgia, SLE and communities of color, CDC lupus registries, lupus morbidity and mortality, epidemiology of lupus, lupus and public health, work productivity impairment and lupus patients, lupus work productivity impairment, depression and lupus patients, lupus and SA-BILD measures, lupus and SLAQ measures, Georgia Lupus Registry, and the GOAL study.

The primary databases that I used were Cumulative Index of Nursing and Allied Health Literature (CINAHL), MEDLINE, PubMed, and ProQuest. The Google scholar search tool was also used to search for open access journals, and articles found via Google scholar were verified to ensure that they were published in peer-reviewed journals using CINAHL Plus. Internet searches were also conducted, and information pertinent to the study was retrieved from organization websites, namely the Lupus

Foundation of America (LFA), American College of Rheumatology, the Lupus Research Alliance, CDC, and the GOAL website, as well as textbooks. Literature from 1985 to present that contained information pertinent to my study was selected for analysis.

Literature Review Related to Key Variables and/or Concepts

Work Productivity Impairment

Because work productivity impairment and unemployment are highly prevalent among individuals with SLE, indirect costs related to loss of income can increase the burden on individuals and the society (Barber & Clarke, 2017). Between 15% and 40% of SLE patients are unemployed within 5 years of diagnosis (Bertoli, Fernandez, Alarcon, Vila, & Reveille, 2007). Even if a person with SLE continues working, flares, organ damage, or poor health can diminish productivity, contributing to the risk of permanent disability (Bertoli et al., 2007). A mean annual productivity cost of $8,659 was reported for individuals with SLE, and higher costs were associated with older age, greater disease activity, and worse health (Panopalis et al., 2008).

Only a few studies in the United States have compared work outcomes between SLE and the general population (Panopalis et al., 2008). Although SLE patients from minority groups are at high risk of poor disease outcomes, and potentially more likely to lose their jobs, they have not been adequately represented in large U.S. studies (Drenkard et al., 2014b). Additionally, SLE symptoms may worsen because of overworking and not resting enough (Askanase, Shum, & Mitnick, 2013), particularly in individuals with lower household income and education, who tend to do worse than the general population (Blanco, 2013).

Patients with SLE function at levels below the general population with regards to self-reported physical and mental functioning (Alarcon et al., 2004). According to Zhang et al. (2010), rheumatologic conditions such as rheumatoid arthritis impact paid work in employed people by making them miss time from work (absenteeism), reducing performance while at work (presenteeism), and reducing routine working hours through changing or losing jobs (employment status change). Increased work absenteeism and work disability rates range from 20% to 50% in a variety of SLE population studies (Campbell, Cooper, & Gilkeson, 2009). Demographic factors associated with work disability include increased age, low educational attainment, and low socioeconomic status (Yelin et al., 2009). Even fewer studies have quantified work absenteeism (Yelin et al., 2009).

Assessing the impact of work disability on productivity in individuals with SLE (Utset et al., 2015) demonstrates the prevalence of, and correlates with, presenteeism and absenteeism. Increased work disability in patients with SLE and depressive symptoms were positively correlated with higher levels of presenteeism (Utset et al., 2015). Highly physical and cognitive jobs are challenging to lupus patients, therefore creating higher absenteeism. Work disability was found to be higher in Black patients who were older and had less formal education (Utset et al., 2015). Employability of persons with lupus can be enhanced by addressing and treating pain, fatigue, and depressive symptoms that worsen cognitive function (Utset et al., 2015).

Garris, Oglesby, Suchs, and Lee (2013) showed that lost income per week increased among hourly employees as the degree of SLE disease activity increased (from

$49 in mild SLE symptoms to $522 in severe SLE symptoms). Work disability can have repercussions for the individual and family, including loss of self-esteem, diminished opportunity to socialize with a person's peer group, reductions in current earnings, loss of health insurance and work-related benefits, and inability to accumulate retirement assets (Garris et al., 2013). Patients with SLE may require work schedule or environment modifications (e.g., jobs featuring flexible work hours, ability to telecommute) to accommodate SLE manifestations and maximize their productivity (Garris et al., 2013). Greater than one-third of employed subjects with SLE in the longitudinal study reported changing jobs because of their SLE diagnosis, seeking reduced stress and flexible work schedules (Garris et al., 2013).

Depression and SLE

Depression is common in patients with chronic diseases (Skare, DaSilva, & Siquiera, 2014). Contributing factors include physical disability, concerns about disease prognosis, emotional effects of skin lesions, and medication use (Beckerman, Auerbach, & Blanco, 2011). Depression in patients with SLE is frequently underdiagnosed (Skare et al., 2014). Depression can lead to changes in quality of life, and it may increase the mortality risk in SLE patients (Greco et al., 2009). A diagnosis of depression in the SLE population ranges from 20% to 47% (Palagini, Mosca, & Tani, 2013). There is a higher prevalence of depression (four times higher) in persons with SLE compared to a matched, non-SLE population (Shen et al., 2013).

Depression has been linked to poor clinical outcomes, including increased work disability (Nery et al., 2007). Shen et al. (2013) found that low socioeconomic status

(SES), low education levels, and unemployment increased the risk of depression among individuals with SLE. Depressive symptoms can also pose obstacles to medication adherence (Julian et al., 2009), further stressing the importance of accurate diagnosis and the treatment of depression in SLE patients. Because of the unpredictability of the disease, SLE patients experience may psychological symptoms due to anxiety and depression, which decreases health-related quality of life outcomes (Auberbach & Beckerman, 2012).

Depression in SLE can also impact medical adherence, which was demonstrated by Julian et al. (2009), where 46% of patients forgot to take medications, and depressive symptoms severity was a predictor for adherence difficulties. Additionally, SLE symptoms such as alopecia can exacerbate self-image problems particularly in women and cause depression (Jolly, Pikard, & Mikoliatitis, 2012). For all forms of depression, depressed mood is the distinctive characteristic, regardless of additional features and of their intensity and variation (Maj & Santorius, 2002). Depressed mood is a sustained emotional state characterized by sadness, low morale, misery, discouragement, hopelessness, emptiness, unhappiness, distress, and overall pessimism (Maj & Santorius, 2002). The sadness and associated feelings pervade all domains of personal and social life of affected individuals (Zakeri et al., 2012).

Symptoms of depression are also commonly observed in SLE patients and are associated with SLE-induced physical disability and the stress of living with a chronic disease (Bachen et al., 2009). Zakeri et al. (2012) indicated that depressive symptoms are frequent in patients with SLE and that depression depth is linked with the severity of the

disease. Additionally, the distribution and frequency of symptoms depend on age, disease duration, and SLE disease severity (Zakeri et al., 2012). Psychiatric symptoms, including depression in SLE patients, are associated with suicide and cardiovascular risks (Jorge et al., 2017; Karassa, Magliano, & Isenberg, 2003).

Illness perception, according to Nowicka-Sauer et al. (2018), was associated with depression in patients with SLE. Glattacker, Heyduck, and Meffert (2013) emphasized that illness and treatment beliefs are related to patient mood, which can be influenced by depression and a cognitive model of illness that can cause interference. Unemployment leads to depression, which may be exacerbated by a lack of access to medical care and support organizations that may mitigate depressive symptoms (Karol, Criscione-Schreiber, & Min, 2013). Pain has also been demonstrated to be influential in the depression diagnosis in individuals with SLE (Gupta, 2015). Treatment of depression in patients with other forms of arthritis has been shown to not only reduce depressive symptoms, but also decrease pain and increase functional status and quality of life (Lin, Caton, & Von Corff, 2003). Kheirandesh et al. (2015) found that patients recently diagnosed with SLE were more prone to depression. Kheirandesh et al. recommended that clinical providers be aware of the need to further mental health supportive services during this initial diagnosis period for the patient with SLE.

Gaps in the Literature

SLE epidemiology studies. In the United States, there were 1.5 million individuals diagnosed with SLE in 2017 and that the disease was more common in people of color (Gupta, 2015). Estimates of the incidence and prevalence of SLE in America

have consistently shown that the disease is approximately three times higher among Black compared to White women, with prevalence rates ranging between 180 and 430 per 100,000 Black women in some communities (Lim & Drenkard, 2015). SLE is relatively uncommon in the general population and is not a reportable disease; thus, it is expensive to capture all diagnosed cases reliably for epidemiologic studies at the national level (Bartels & Ramsey-Goldman, 2014). The concept of reliability in SLE public health research is dependent on the acquisition of precise data, determining the accuracy of clinical variables based on source documents, such as clinical progress notes, lab variables, or even biopsies (Blanco, 2013; Brandt et al., 2017).

National estimates of SLE prevalence and incidence are not accurate due to limitations with acquisition of SLE-related epidemiologic data (Brandt et al., 2017). In 2009, the CDC launched a program to obtain more accurate estimates of the incidence and prevalence of lupus in U.S. minority populations (Hendricks, 2012). The CDC funded four states (Georgia, Michigan, California, and New York) to conduct SLE surveillance among populations with high proportions of Black, Asian Americans, and Hispanic Americans, in addition to Whites (Hendricks, 2012; Lim & Drenkard, 2015). Moreover, data collected through the Indian Health Service system were used to estimate the burden of lupus among Native Americans (Hendricks, 2012; Lim & Drenkard, 2015).

To develop these SLE registries, a partnership was developed between the state health department and an academic counterpart (Hendricks, 2012; Lim & Drenkard, 2015). The state health department has the power to conduct public health surveillance and provides surveillance expertise (Lim et al., 2009; Lim & Drenkard, 2015). Because

of its legal authority, the state health department is a public health authority under the Health Insurance Portability and Accountability Act (HIPAA), and health care providers are allowed under the HIPAA Privacy Rule to provide protected health information, without written patient consent, to state health departments and their designated agents (Lim et al., 2009). Therefore, the state health departments contracted with the academic partners (including Emory University) to implement the CDC grant by managing the project and collecting the data. In addition, all pertinent local, university, state, and CDC institutional review board reviews and approvals had been obtained (Lim et al., 2009).

The CDC-funded Georgia Lupus Registry provides an accurate epidemiologic estimate of lupus prevalence in the DeKalb and Fulton counties in Atlanta, Georgia (Lim & Drenkard, 2015). The estimates are generalizations in the larger population, and the CDC-funded projects have a precise measurement of the prevalence of SLE in the targeted geographic areas (Lim & Drenkard, 2015). The findings from the analysis of this registry reveal that Black populations have three to four times the risk of being diagnosed with SLE than their White counterparts, and that the disease onset occurs at a younger age among Black individuals (Lim & Drenkard, 2015). Furthermore, the Georgia Lupus Registry incidence and prevalence rates for Black women were among the highest ever reported in the United States (Lim & Drenkard, 2015). In addition to having a higher prevalence of SLE, Black women are more likely to have organ damage caused by SLE (Uribe & Alarcon, 2003).

SLE incidence estimates have already been described in the CDC-funded lupus registries and include data from the Michigan Lupus Registry (Somers, Marder, &

Cagnoli, 2014). This registry documented an estimated annual incidence of SLE that was much higher for Black than White individuals in Washtenaw and Wayne counties in Michigan (111.6 versus 47.5 per 100,000 people) (Somers et al., 2014). Moreover, the Georgia Lupus Registry reported an estimated annual incidence that was much higher for Black as compared to White individuals in DeKalb and Fulton counties in Georgia (128.0 versus 39.9 per 100,000 people) from 2002 to 2004 (Somers et al., 2014). Additionally, the annual incidence for different racial/ethnic groups from 2002–2004 was much higher for Blacks than Whites in Michigan (7.9 vs. 3.7 100,000 people) (Somers et al., 2014) and in Georgia (9.4 vs. 3.2 per 100,000 people; Lim et al., 2014).

Although GOAL studies have established an SLE estimate for counties in Georgia (Drenkard et al., 2014a, 2014b; Lim & Drenkard, 2015), it is still unknown how SLE impacts communities of color in the state of Georgia (Lim et al., 2014). The GOAL study is the only derived study from a CDC-funded lupus registry to delve further into impactful SLE disparities measures such as work productivity impairment, depression, and disease activity and damage measures (Drenkard et al., 2014a, 2014b; Lim & Drenkard, 2015).

GOAL studies. The gaps in the literature are significant with regards to findings in epidemiologic surveillance of SLE, as the GOAL cohort was designed to address gaps in epidemiologic and outcome knowledge about SLE (Drenkard et al., 2014a, 2014b; Lim & Drenkard, 2015). Six articles have been published on GOAL, including the article by Chae et al. (2015), which examined associations between unfair treatment and attributing unfair treatment to racial discrimination with cumulative disease damage (using SA-

BILD) among Black women with lupus. The second GOAL article was the first study to examine the rates of self-reported preventive care in an ethnically and economically diverse cohort of patients with SLE (Drenkard et al., 2013). The third article examined the burden of SLE on work loss, unemployment, and work productivity impairment in the GOAL cohort (Drenkard et al., 2014a). The fourth GOAL article, by Brandt et al. (2017), described the use of the Lupus Impact Tracker (LIT), which is a validated, SLE-specific health-related quality of life (HRQoL) tool that inquires about concentration, medication side effects, fulfilling family responsibilities, feelings of being worn out upon waking, bodily pain and aching, limitation of activities due to pain/fatigue, anxiety, depression, self-consciousness related to physical appearance, and ability to plan activities. The fifth GOAL article, by Drenkard et al. (2014b), assessed the reliability and validity of SA-BILD, a patient-reported measure of organ damage in SLE patients. The last GOAL article examined whether older age was associated with lower HRQOL among patients with SLE and whether disease activity and disease damage explained this association (Plantinga et al., 2016). No prior GOAL study has examined if depression mediates the relationships between SLE disease activity and damage and work productivity impairment, which reflects a gap in the literature and the need for further analysis of the GOAL dataset (Drenkard et al., 2013, 2014a; Plantinga et al., 2016).

SLE studies. Lupus in Minorities: Nature versus Nurture (LUMINA) cohort study researchers have found that high SLE disease activity has been consistently and independently associated with several socioeconomic, demographic, psychological, and behavioral features, such as lack of health insurance, abnormal illness-related behaviors,

and poor social support (Alarcón, 2008; Fernandez et al., 2007). Race, poverty, and factors closely associated with reduced access to quality healthcare, reduced comprehension of disease and the medical system, increased competing home and work demands, and reduced self-confidence and social support have also been correlated and are predictive of SLE disease activity, organ damage, and functional ability of self-care (Alarcón, 2008; Fernandez et al., 2007). Furthermore, racial or ethnic minority group status and poverty status are significant predictors of increased risk of SLE and poor SLE outcomes (Demas & Costenbader, 2009).

A research study conducted using the Composite International Diagnostic Interview (CIDI) and the SLAQ analyzed women with SLE (Bachen et al., 2009). Based on assessing stress and depression in relation to functional health behaviors in the study, it was determined that 47% reported major depressive disorder (Bachen et al., 2009). In a study by Williams et al. (2014), depressive symptoms affected functionality and created moderate effects on social/role limitations. Assessing psychosocial impact has also yielded similar findings with regards to depression and individuals with SLE (Beckerman et al., 2011). In an exploratory study by Beckerman et al. (2011), the highest general causes of self-reported depressive and anxious feelings were changes in appearance due to SLE and limitations in physical abilities due to muscle and joint pain related to SLE. This was in addition to the secondary effects of medication use such as weight gain, and complications derived from autoimmune changes causing hair loss in Black and Hispanic patients. Medicaid recipients were also more likely to need psychosocial assistance with

depression. On the other hand, the higher the self-perceived control of SLE, the less likely respondents reported feeling depressed (Beckerman et al., 2011).

Additionally, while all SLE patients should be assessed comprehensively, Black and Hispanic women may require more psychosocial resources and support (Jordan et al., 2018). Every effort should be made to provide culturally competent assessments and interventions (Jordan et al., 2018). Beckerman et al. (2011) stated that SLE health care treatment teams need to be aware of the potential psychosocial and mental health impacts of SLE. Danoff-Burg and Friedburg (2009) conducted a study to assess the unmet needs of SLE patients and found that 71% reported needing assistance for depression. An Australian study determined that SLE patients should be assessed early for the likelihood of depressive sequelae (Moses, Wiggers, & Nicholas, 2005). Lastly, the relationship between SLE and depression is still inconclusive. Some studies suggest that depression is due to disease activity while others suggest that depression can trigger stress hormones to increase disease activity (Duvdevany, Cohen, & Minkser-Valtzer, 2011). Ongoing research using the GOAL dataset will contribute to the body of SLE-related depression literature where depression is multi-factorial and directly or indirectly related to the disease (i.e. stress, finances, and medication side effects). Treatment for both the SLE diagnosis and depression therefore needs to be approached simultaneously (Greco et al., 2009; Gupta, 2015; Julian et al., 2009).

It is important to understand that SLE can also reduce a patient's ability to work, and results in high rates of work disability, reductions in working hours, and/or changes in the nature of a patient's work (Baker & Pope, 2009). Prior studies on work disability

in patients with SLE have demonstrated greater job loss impact in Black patients affected by organ damage and lower SES (Bertoli et al., 2007; Bultink et al., 2008; Mok et al., 2008). In the study by Bertoli et al. (2007) it was determined that SLE patients with more severe disease and lower socioeconomic status were at higher risk of being disabled. In the study by Mok et al. (2008), more than 50% of patients reported memory loss, and 85% of patients reported fatigue as the primary reasons they had to quit their jobs. A Dutch study by Bultink, Franktein, and Dijkmans (2008) also supported this finding with SLE patients and found work dropout in 75% of cases, largely due to disease-related factors. In addition, there was a significant association between unemployment, disease-related characteristics, and reduced quality of life (Bultink et al., 2008). The study by Gordon, Isenberg, and Lerstrom (2013) revealed that the SLE burden affected respondent's employment, career choices, and productivity, which included impact on health-related quality of life indices. It is the intent of the current study to assess the degree of correlation between work impairment and depression and assess whether this correlation is found in the work impairment and depression scale data collection responses of GOAL participants.

Study Rationale

The literature review provided a sufficient rationale for the proposed study. As documented in the literature review, the strengths in the existing SLE literature include comprehensive analyses of varied SLE cohorts detailing how work productivity impairment, depression, disease activity (severity), and damage (disease and medications) impact SLE patients (Alarcón, 2008; Bachen et al., 2009; Baker & Pope, 2009; Barber &

Clark, 2017; Carter et al., 2016; Drenkard et al., 2014a; Garris et al., 2013; Gordin et al., 2013; Karol et al., 2013; Kheirandesh et al., 2015; Palagini et al., 2013; Utset et al., 2014). However, the existing literature is limited, as no study to date has comprehensively examine the relationships between and among SLE disease activity and damage, depression and work productivity impairment utilizing a SLE cohort (Drenkard et al., 2014a; Jordan et al., 2018). Further study needed to determine if significant relationships exist among SLE disease activity and damage and work productivity impairment and if depression mediates this relationship, in an SLE cohort

Definition of Terms

Depression: a disorder of the brain. There are a variety of causes, including genetic, biological, environmental, and psychological factors. Depression can happen at any age, but it often begins in teens and young adults. It is much more common in women (Gupta, 2015).

Health Insurance: insurance against loss generated by illness, especially insurance providing compensation for medical expenses (Sommers, Gawande, & Baicker, 2017).

Income: a gain or recurrent benefit usually measured in money that derives from capital or labor; also, the amount of such gain received in a period of time (Sommers et al., 2017).

Race: a category of humankind that shares certain distinctive physical traits (Smedley, 2007).

SA-BILD: Self-Administered Brief Index of Lupus Damage is a patient-reported measure of lupus damage (Drenkard et al., 2014a).

SLAQ: Systemic Lupus Activity Questionnaire is a validated self-questionnaire to evaluate disease activity. It has the advantages of being inexpensive, being easy to use in large cohort studies, and having been used in several studies (Karlson et al., 2003).

Systemic Lupus Erythematosus (SLE, Lupus): Lupus is a chronic autoimmune disease that can damage any part of the body (skin, joints, and/or organs; Lim et al., 2009).

Work Productivity Impairment: the degree that impairment impacts paid work and activities in lupus patients (Panopolis et al., 2008).

Assumptions, Limitations, and Delimitations

Assumptions

All studies have assumptions, or statements of truth relevant to the type of study conducted (Creswell, 2009). One assumption of the study was that it was important that SLE patients have the capability to work despite facing potential barriers including depression. it is assumed that the GOAL participants answered the survey questions truthfully. Prior research and the gaps in the existing literature provide support for this assumption (Cosatti et al., 2017; Drenkard et al., 2014a; Utset et al., 2015; Yelin et al., 2009). Another assumption of this study was that Bandura's (1986) SCT provided a meaningful theoretical framework to understand SLE disease activity and damage, depression, and work productivity. This assumption was met, as SCT has been used extensively in the disease prevention and health promotion empirical literature (Bandura,

2011). Finally, based on prior literature (Drenkard et al., 2013, 2014a, 2014b; Jordan et al., 2018), there were assumptions that the GOAL data set was comprised of a research-relevant cohort and that the gold standard instruments accurately captured the constructs of SLE disease activity and damage, depression, and work productivity impairment.

Limitations and Delimitations

This study has a certain scope with regard to a specific focus of the study, and delimitations/limitations that result from the study scope (Creswell, 2009). This study focused on SLE disease activity and damage, depression, and work productivity impairment in a specific SLE cohort, 2014 GOAL participants. The study was not inclusive enough to overcome the threat of population validity, or inability to generalize findings beyond the population represented by the study sample (Delgado-Rodriguez & Llorca, 2004; Kumar & Acharya, 2014). Due to this focus, the degree of inference of study findings was limited to individuals with SLE who resided in Georgia and who were employed; results cannot be generalized to the broader SLE population, especially SLE patients who are not in the workforce.

The use of cross-sectional correlational research design not only precluded the ability to determine causality, it introduced certain threats to internal validity into the study. One internal validity threat was the inability to ensure temporal precedence (i.e., that the predictor and mediating variables preceded the criterion variable) (Drost, 2011; Kumar & Acharya, 2014). Another internal validity threat was confounding, in which an unmeasured variable significantly covaries with study variables (Zyphur & Pierides, 2017). While this study included and tested many potential confound variables, it was

beyond the scope of this study to include all potential confounds. Medication adherence, social support, self-efficacy, fatigue and various work variables (i.e., type of job/position, number of years employed) could be potential confounding variables in the study. Moreover, this study did not examine work impairment rates over time; impairment rates likely would have increased as the disease progressed among participants.

As noted by Drenkard et al. (2014a, p. 885), GOAL study participants were "not a true incident cohort," and the use of a convenience sampling may have increased the likelihood of certain methodological biases. These include *selection bias*, in which certain individuals (i.e., those who have less severe SLE symptoms and damage) may have been more likely to participant in the GOAL study), which is associated with the *healthy worker effect* (HWE) (Shah, 2009). The HWE refers to the fact that employed study participants are more likely to be healthy, and they tend to have lower morbidity and mortality rates than non-employed participants (Shah, 2009). Other methodological biases that could have affected this study were *recall bias*, or differences in response to study instruments due to changing perceptions regarding disease severity and functioning, and s*ocial desirability bias*, or the tendency to provide more socially-acceptable responses on a survey (Creswell, 2009; Drost, 2011).

Significance of the Study

This study can add to the existing epidemiologic SLE research by furthering empirical understanding as to SLE disease activity and damage directly affect poor work productivity as well as how depression mediates the relationship between SLE disease activity and damage and work productivity impairment among individuals with SLE.

Empirical research has noted significant associations between SLE damage and activity and depression (Jordan et al., 2018; Zakeri et al., 2012) and SLE damage, SLE activity, and work impairment (Cooper et al., 2009; Garris et al., 2013). However, the relationships between SLE disease activity and damage, depression, and work activity impairment are not well understood (Drenkard et al., 2014a; Jordan et al., 2018). This study was particularly critical as there has been no prior empirical work examining the mediating effect of depression with regard to SLE disease activity and damage and work productivity impairment.

Implications for Social Change

Prior studies have shown that employability and work outcomes can be enhanced by improving treatment of depressive symptoms in SLE patients (Boehmer et al., 2016; Náfrádi, Nakamoto, & Schulz, 2017; Nery et al., 2007). Findings from this study can be used to inform the development of SLE-based programs and treatments aimed at reducing depression and increasing work productivity. These findings can be applied outside of the state of Georgia and facilitate efforts to support patients with SLE who are dealing with issues related to depression and work productivity impairment. .

Transition and Summary

This concludes the first section of the dissertation. This section provided the research questions and presented an overview of the study methodology and design. It included a summary of Bandura's (1986) SCT, the guiding theoretical framework, and provided a comprehensive literature review on pertinent topics, including aspects of SLE disease activity, symptomatology, and damage, the role of depression in SLE disease

perceptions, and SLE and work productivity. In this section, pertinent constructs were defined, and the study scope and delimitations were stated. The following section is devoted to a more detailed review of the study methodology, including the study design, data collection procedures, the instruments used to measure study constructs, and the data analysis plan.

Section 2: Research Design and Data Collection

Introduction

Scholars have suggested significant links between SLE severity and damage and work productivity impairments and increased depression (Drenkard et al., 2014a; Karol et al., 2013; McCormick et al., 2018; Zhang et al., 2017). However, there is little understanding as to whether depression mediates the associations between SLE severity and damage and work productivity impairments (Zhang et al., 2017). The purpose of this quantitative study was to determine whether SLE disease activity and damage were significantly associated with work productivity impairment and whether depression mediates between SLE disease activity and damage work productivity impairment in a GOAL study cohort of adult SLE patients from Georgia who were in the workforce at the time of data collection.

The purpose of this chapter is to provide a comprehensive overview of the study methodology. The first section of the chapter pertains to the research design; it includes the study research questions and associated hypotheses, identification of the study variables, and a rationale for selecting the design used in the study. Subsequent sections address (a) the study population, sample, and sampling; (b) GOAL study participant recruitment and data collection procedures; (c) the study instruments and operationalization of study variables; (d) the data analysis plan; and (e) ethical procedures.

Research Design and Rationale

In this quantitative study, I examined the associations between SLE damage and activity, depression, and work productivity impairment in a sample of GOAL study participants who were in the workforce at the time of data collection. This study had three research questions. The first research question was descriptive and examined aspects of work productivity impairment. The second and third research questions, inferential in design, assessed if there were direct associations between SLE disease activity and damage and work productivity impairment and whether depression acted as a mediator between SLE disease activity and damage and work productivity impairment.

This study had two independent (predictor) variables: lupus activity, measured using the SLAQ (Karlson et al., 2003), and lupus damage, assessed using the SA-BILD (Drenkard et al., 2014a). The mediating variable was depression, measured using the PHQ-9 (Kroenke et al., 2001). The dependent (criterion) variable was work productivity impairment, measured using the WPAI (Reilly et al., 1993). This study included as potential covariates (a) participants' demographics (i.e., ethnicity, gender, age, educational level, poverty status, marital status, work status), participant work status (i.e., employed full- or part-time), (b) healthcare factors (i.e., type o health insurance, yearly out-of-pocket expenses), and (c) health factors (i.e., SLE disease duration).

Type of Research Design

The implementation of the study required the use of cross-sectional and observational (nonexperimental) research designs. I employed a cross-sectional observational research design that had descriptive and analytical components. The study

was cross-sectional, as data used to address all three research questions were collected at one point in time (Alexander, Lopes, Ricchetti-Masterson, & Yeatts, 2013; Levin, 2006). Cross-sectional studies are common in epidemiology and public health and provide empirical information that can inform future public health policy planning efforts (Alexander et al., 2013). A large sample size of study participants is more easily obtained in cross-sectional studies, and the use of this design eliminates the attrition bias (Levin, 2006).

The cross-sectional observational design did have certain methodological weaknesses (Alexander et al., 2013). The use of this design did not allow for the examination of data over time or across time-points, and it precluded the ability to confirm temporal precedence (i.e., that the predictor preceded the mediator and both preceded the criterion variable; Alexander et al., 2013). However, the cross-sectional design was fitting for this study, as there were time and resource constraints due to the nature of the doctoral program. A cross-sectional design is both time and cost-effective for a study, in contrast to a study that employs a longitudinal design that requires more capital, infrastructure, and support (Alexander et al., 2013).

I used an observational (nonexperimental) design, as Research Questions 2 and 3 pertained to the examination of relationships among the naturally occurring variables of SLE damage and severity, depression, and work productivity impairment. The observational approach is analogous to the nonexperimental, correlational design in social sciences research: both are used to examine associations among nonmanipulated variables (as opposed to the examination of group/cohort differences; Salazar, Crosby, &

DiClemente, 2015). In accordance with the observational design, the independent variable is identified as the predictor variable and the dependent as the criterion variable (Salazar et al., 2015), nomenclature used in this study.

There are some methodological weaknesses of the observational design (Salazar et al., 2015). The use of an observational design as opposed to an experimental design increases the likelihood of confounding bias (Salazar et al., 2015), discussed in detail near the end of this chapter. The testing of potential covariates and the incorporation of covariates in hierarchical linear regression (HLR) models will reduce confounding bias but will not completely eliminate it (Salazar et al., 2015).

Theoretical Framework

The theoretical framework that guided this study and informed the study research questions and hypotheses was Bandura's (1986) SCT. Central to SCT is the concept of reciprocal determinism where B is the function of the P interacting with his or her E (Bandura, 1986, 2011). According to reciprocal determinism, environmental stimuli and reinforcement contingencies influence and interact with personal attributes of personality characteristics, cognitive factors, and skills to influence the nature, frequency, and intensity of behavior (Bandura, 1986, 2011). In this study, it was posited that SLE disease activity and damage and depression, products of the person interacting with her/his environment, significantly influenced work-related behavior, specifically, work productivity impairment, in a sample of SLE patients from the GOAL study.

Methodology

Target Population and Sampling

The study's targeted population was individuals in Georgia with SLE. The prevalence rate of SLE in Georgia has been considerably higher than the national rate, 145.8 as compared to 17.5 per 100,000 people (Chae et al., 2015; Lim & Drenkard, 2015). The study sample was the 2014 GOAL study cohort of adult SLE patients from metropolitan Atlanta, Georgia. The GOAL participant inclusion criteria were (a) a validated diagnosis of SLE (documentation of ≥ 4 ACR criteria per the patient's physician) or cutaneous lupus erythematosus (documentation of a consistent skin biopsy or clinical diagnosis by a dermatologist or rheumatologist of discoid lupus, lupus panniculitis, chilblain lupus, or other types of skin lupus); (b) adult status (i.e., ≥18 years of age); (c) Georgia residence; (d) capacity to provide informed consent; and (e) willingness to provide access to medical records by signing a medical record release form to confirm diagnosis (Drenkard et al., 2014b). Participants who were excluded from the GOAL were those unable or unwilling to provide informed consent to give study researchers access to their medical records and/or to complete periodic questionnaires (Drenkard et al., 2014b). This study had an additional inclusion criterion that participants had to work full-time or part-time for pay at the time of data collection.

Power Analysis

I conducted an *a priori* power analysis for multiple linear regression (MLR) using G*Power (Faul, Erdfelder, Lang, & Buchner, 2007) to determine the sample size needed to achieve adequate power for the study. I set certain parameters for the power analysis:

(a) the significance value was set to $p < .05$; (b) power was set to .80; (c) the number of predictors (i.e., predictor variables and the dummy-coded potential covariates) was set to 14; and (d) in accordance with findings from relevant SLE meta-analyses (Leslie & Crowe, 2018; Zhang, Fu, Yin, Zhang, & Shen, 2017), the effect size was set to small-to-medium, $f^2 = .15$. The sample size needed to achieve adequate power was determined to be $N = 135$ (see Figure 2 for G*Power output). The study sample of 257 GOAL study participants well exceeded this sample size.

F tests - Linear multiple regression: Fixed model, R^2 increase		
Analysis:	A priori: Compute required sample size	
Input:	Effect size f^2	= 0.15
	α err prob	= 0.05
	Power (1-β err prob)	= 0.80
	Number of tested predictors	= 14
	Total number of predictors	= 14
Output:	Noncentrality parameter λ	= 20.25
	Critical F	= 1.78
	Numerator df	= 14
	Denominator df	= 120
	Total sample size	= 135
	Actual power	= 0.80

Figure 2. G*power (Faul et al., 2007) power analysis findings.

GOAL study recruitment procedures. GOAL study researchers used convenience sampling when selecting study participants. Convenience sampling is a type of nonprobability sampling in which researchers select study participants based on participants' accessibility and willingness to participate in the study (Etikan, Musa, & Alkassim, 2015). The sample of participants was limited to th**e** 527 of GOAL study participants who worked full- or part-time and who completed baseline surveys.

It was important to GOAL researchers that study participants represented the full spectrum of socioeconomic and racial groups with SLE (Chae et al., 2015; Drenkard et al., 2013; Lim & Drenkard, 2015). This was achieved, in large part, by recruiting individuals who were (a) on the Georgia Lupus Registry, (b) patients at the Emory and the Grady SLE clinics, or (c) patients of participating community rheumatologists. Recruitment patient lists were prepared from the GLR, Emory/Grady Clinics, community rheumatologists, and the LFA. These lists included information useful in locating the patients, such as name, date of birth, and most current available address and telephone number. The GOAL study researchers used three types of recruitment strategies (Chae et al., 2015; Drenkard et al., 2013, 2014a), discussed in the following sections.

GOAL study mail-based recruitment. GOAL researchers mailed a study recruitment package to qualifying patients. The study packet included (a) a personalized cover letter that explained the aims of the GOAL study, the participant's role in the study, informed consent procedures, and contact information of the GOAL study research coordinator; (b) a GOAL study brochure; (c) the GOAL study informed consent form; and (d) a postage-paid return envelope for interested patients to return their signed informed consent form (Chae et al., 2015; Drenkard et al., 2013, 2014a). The GOAL study research coordinator was available to answer patients' questions about the study and their role as participants, and/or to explain the informed consent form. The research coordinator documented contacts as required by the institutional review board (IRB) from subsequent conversations with patients; she referred questions to the principal investigators (PIs) as needed. All patients returning a signed informed consent form were

considered enrolled in the GOAL Cohort Study (Chae et al., 2015; Drenkard et al., 2013, 2014a, 2014b).

An adaptation of Dillman's (2000) tailored design method (TDM) ensured a high response rate (Chae et al., 2015; Drenkard et al., 2013, 2014a, 2014b). TDM involves a series of subsequent mail and telephone attempts to contact patients starting a week after the mailing of the study packet (Dillman, 2000). Per the TDM procedure, the GOAL study research coordinator mailed a follow-up postcard to all recipients approximately 1 week after the mailing of the study packet. The postcard served as a "thank you" for those who returned a signed consent form and acted as a reminder to others to return their signed consent form should they be interested in participating in the study. The GOAL study research coordinator sent a reminder letter to patients who had yet to return a signed informed consent approximately 3 weeks after the mailing of the study packet (2 weeks after the mailing of the postcard). The last attempt to contact patients yet to return their informed consent form occurred approximately 1 month after the mailing of the study packet and was by telephone (Chae et al., 2015; Drenkard et al., 2013; 2014a).

GOAL study face-to-face recruitment. Using a study script, the research coordinator introduced the GOAL study, its intent, and informed consent information during face-to-face visits with patients. The face-to-face visits occurred at Emory and Grady Lupus clinics or community rheumatologists, with the research coordinator recruiting patients who were previously screened for eligibility (Chae et al., 2015; Drenkard et al., 2013, 2014a). The research coordinator answered any questions posed by the patients and obtained written informed consent from patients who showed interest

in participating in the study (Chae et al., 2015; Drenkard et al., 2013, 2014a). She placed the signed informed consent forms in separate folders, which she immediately returned to the GOAL study office to place in a locked file cabinet accessible only to her the and the GOAL study researchers.

GOAL study telephone-based recruitment. GOAL study researchers received an IRB waiver of the requirement of written informed consent for telephone-based recruitment (Chae et al., 2015; Drenkard et al., 2013, 2014a, 2014b). During the telephone call, the research coordinator, following a script, reminded patients of the GOAL study, its intent, and the role of the patient in the study and gave participants the option to provide oral consent. The researcher telephoned patients who were previously screened for eligibility by physicians at the Emory and Grady clinics or community rheumatologists but who had yet to return informed consent and/or were not available during the coordinator's clinic visits (Drenkard et al., 2013, 2014a, 2014b). During the telephone call, the researcher, following a study script, discussed with the patients the study, its intent, the role of the patient in the study, and the informed consent process. Interested patients provided oral informed consent, which was documented by the research coordinator (Drenkard et al., 2013, 2014a, 2014b).

GOAL study data collection procedures. GOAL study researchers used various data collection procedures. Participants could complete the GOAL survey online (Drenkard et al., 2013, 2014a, 2014b). The online consent document was built in a HIPPA-protected system, which also included the study surveys. Participants had a unique log-in and personal password to access their account in the system (Drenkard et

al., 2013, 2014a, 2014b). Upon consent, patients could complete a hard copy of the study survey provided by the coordinator during their face-to-face clinic meeting. Once participants completed the study survey, the coordinator assigned an associated ID for each survey and placed the survey in a folder (separate from the informed consent form). She returned the survey folders to the GOAL study office, where she locked them in a file cabinet separate from the consent forms in the GOAL study office. Only GOAL study researchers had access to the study surveys.

Accessing GOAL study archival data. Upon Walden IRB approval and confirmation of Emory University IRB approval and after completion of the material transfer agreement (MTA), I accessed the GOAL study data through Emory University. The data access protocol implemented by Emory University required that I, as the student scholar, (a) identified the study variables necessary to conduct the study, (b) submitted an online data access request form to Emory GOAL researchers, and (c) retrieved data from the GOAL study data sets.

Instrumentation and Operationalization of Constructs

The following sections provide information on the study predictor variables of SLE damage and SLE severity, the mediator of depression, the criterion variable of work productivity impairment, and the potential demographic covariates. Variable information presented in the following sections include the variable operational definition, type of response coding (i.e., nominal/categorical, ordinal, interval, ratio), and the computation and interpretation of the variable score. The instrument used to assess the predictor,

criterion, or mediating variables is presented, as is evidence of the instrument's psychometric (i.e., validity and reliability) information.

Predictor Variable: SLE Disease Activity

SLE disease activity was measured using the 24-item SLAQ (Karlson et al., 2003). The SLAQ is a patient self-report instrument adapted from the physician-reported Systemic Lupus Activity Measure (SLAM; Bae et al., 2001) and is the only self-report SLE activity instrument used in epidemiological studies with large cohort samples (Castrejón, Tani, Jolly, Huang, & Mosca, 2014). The respondent completes the SLAQ by recalling whether, in the past 3 months, he or she experienced fatigue, weight loss, and fever as well as disease activity across nine systems, including the integumentary (e.g., mouth ulcers, rashes, alopecia), cardiovascular (e.g., hypertension), lymphatic (e.g., lymphadenopathy), respiratory (e.g., dyspnea), and nervous (e.g., seizures, headaches) systems (Karlson et al., 2003). The 24 items are coded where 0 = *no problem*, 1 = *mild problem*, 2 = *moderate problem*, and 3 = *severe problem* (Karlson et al., 2003). Item scores are weighted by organ system to compute the ratio-coded SLAQ total scale score, which can range from 0.00 to 44.00, with a higher score denoting higher SLE activity (Karlson et al., 2003).

Psychometric findings indicated that the SLAQ is a sound self-report instrument to gauge SLE activity (Castrejón et al., 2014; Karlson et al., 2003; Romero-Diaz, Isenberg, & Ramsey-Goldman, 2011; Yazdany et al., 2009). Sensitivity and specificity indices ranged from the low-.80s to the mid-.90s (Castrejón et al., 2014). Exploratory factor analysis findings have showed that items cluster according to system, supporting

the construct validity of the SLAQ (Romero-Diaz et al., 2011). SLAQ strongly correlates with physician-rated disease activity, physician scores on the SLAM, and patient daily functioning scales, providing evidence of its criterion-related concurrent validity (Castrejón et al., 2014; Karlson et al., 2003; Romero-Diaz et al., 2011; Yazdany et al. 2009). The SLAQ has sound internal consistency, with an average Cronbach's alpha of .87 (Karlson et al., 2003; Romero-Diaz et al., 2011; Yazdany et al . 2009).

Predictor variable: SLE disease damage. SLE disease damage was assessed using the self-administered Brief Index of Lupus Damage (SA-BILD; Drenkard et al., 2014a). The SA-BILD is a patient self-report measure of perceived SLE damage and is an adaptation of the physician-reported SLE International Collaborating Clinics/American College of Rheumatology Damage Index (SDI). The 28 items on the SA-BILD inquire as to whether the patient has received a diagnosis of 37 SLE-associated medical problems as they pertain to the eye (e.g., cataracts), brain (e.g., seizures, stroke), kidneys (e.g., kidney transplant), lungs (e.g., pulmonary hypertension), heart (e.g., angina, pericarditis), blood vessels (e.g., deep vein thrombosis), stomach/bowel (e.g., small intestine, spleen) surgery, muscles and bones (e.g., osteoporosis), skin (e.g., skin ulcer), Type II diabetes, and cancer. A score of 1 is given for each diagnosed medical problem (a score of 0 indicates absence of a diagnosed medical issue). Scores for each of the 37 medical conditions are summed to create the SA-BILD total scale score. The ratio-coded SA-BILD total sale scores can range from 0 to 37 points, with a higher score denoting a higher degree of perceived SLE damage (Drenkard et al., 2014a).

The SA-BILD is psychometrically sound (Castrejón et al., 2014)) and has been validated in a sample of African American GOAL participants (Drenkard et al., 2014a). Castrejón et al. (2014) provided evidence of the construct and criterion-related concurrent validity of the SA-BILD, identifying it as a gold standard assessment of SLE damage. Criterion-related concurrent validity of the SA-BILD has been confirmed in studies documenting 81% to 99% correspondence between SA-BILD and SDI items (Drenkard et al., 2014a). Study findings have further shown significant associations between the SA-BILD and the SID and the Lupus Damage Index Questionnaire (LDIQ) (ps < .001) and the SA-BILD and length of time since SLE diagnosis, patient- and physician-reported health quality, and number of hospitalizations due to SLE (ps < .01 to <.001) (Castrejón et al., 2014; Drenkard et al., 2014a). The SA-BILD has excellent inter-item reliability, with Cronbach's alphas in the low to mid-.90s and sound 30-day test-retest reliability (r=0.92, p < .001) (Drenkard, et al., 2014a).

Mediating variable: Depression. The Patient Health Questionnaire (PHQ-9; Kroenke et al., 2001) was used as a measure of depression in this study. When answering the PHQ-9, respondents respond to the statement, "Over the last two weeks, how often have you been bothered by any of the following problems?" by providing answers to nine items that align with DSM-IV depression symptom indicators. Sample items are "little interest or pleasure in doing things" and "feeling tired or having little energy". The response coding for each PHQ-9 item is 0 = not a at all, 1 = several days, 2 = more than half the days, and 3 = nearly every day. The nine items are summed to provide the PHQ-9 full scale score, which can range from 0 to 27 points. Scores between 0 and 4 indicate

no to minimal depression, scores between 5 and 9 indicate mild depression, scores between 10 and 14 indicate moderate depression, scores between 15 and 19 indicate moderately severe depression, and scores between 20 and 27 indicate severe depression (Kroenke et al., 2001). Scholars consider a PHQ-9 score of 10 or higher to be indicated of clinical depression (Kroenke et al., 2001; Manea, Gilbody, & McMillan, 2015; Mitchell, Yadegarfar, Gill, & Stubbs, 2016; Smarr & Keefer, 2011).

Medical, public health, and nursing research scholars have extensively utilized the PHQ-9, as noted in several meta-analysis studies examining the diagnostic and psychometric soundness of the PHQ-9 (Manea et al., 2015; Mitchell et al., 2016; Moriarty, Gilbody, McMillan, & Manea, 2015). High diagnostic sensitivity indices (ranging from .80 to .95) and specificity indices (ranging from .72 to .91) coupled with strong psychometric findings in studies with diverse patient samples provided evidence that the PHQ-9 is one gold standard measure of depression ((Moriarty et al., 2015; Smarr & Keefer, 2011). The Veteran's Administration (VA) and the National Institute for Health and Clinical Excellence (NIHCE) endorse the use of the PHQ-9 in primary care settings (Smarr & Keefer, 2011).

Exploratory and confirmatory factor analysis findings have indicated that the PHQ-9 items comprise a single factor (Mitchell et al., 2016; Smarr & Keefer, 2011). Significant correlations between the PHQ-9 and the Hospital Anxiety and Depression Scale (HADS) ($r = .66$, $p < .001$) and the Hamilton Rating Scale for Depression (HRSD) ($r = .78$, $p < .001$) provide evidence of its criterion-related concurrent validity (Mitchell et al., 2016; Smarr & Keefer, 2011). The inter-item reliability of the PHQ-9 is sound,

with an average Cronbach's alpha of .92, and seven-day test-retest correlations ranging from rs = .81 to .96, $p < .001$) (Mitchell et al., 2016; Smarr & Keefer, 2011)

Criterion variable: Work productivity impairment. Work productivity impairment was measured using the Work Productivity and Activity Impairment instrument (WPAI; Reilly, Zbrozek, & Dukes,1993). The WPAI total score is derived following a specific process outlined by Reilly et al. (1993). The first question of the WPAI is, "During the past seven days (not including today), how many hours did you miss work because of problems with your health?" The second question on the WPAI is, "During the past seven days (not including today), how many hours did you actually work?" The participant's response (in hours) to question 1 is divided by the participant's response (in hours) to questions 1 and 2 (i.e., Q/ [Q1 + Q2]), then multiplied by 100. To provide an example: if a participant answered '8 hours' to question 1, 8 hours would be added to the participant's response to question 2, for example, 32 hours, so that 8/8 + 32 = 8/40 = .20 X 100 = 20. The third WPAI question is "During the past seven days, how much did your health affect your productivity while you were working?" The response coding for the third question is 0 to 10, with a higher score denoting that the participant's health affected his/her work productivity to a great deal/degree. The participant's response on the third WPAI question is divided by 10, the highest score on this question (i.e., Q3/10), then multiplied by 100 (Reilly et al., 1993). To provide an example: if a participant answered 4 on the third WPAI question, the score would be computed as 4/10 or .40 X 100 = 40. The total WPAI scale score is computed by using this formula: (Q1/ [Q1 + Q2] + [1 – Q1]/ {Q1 + Q2}]) X 100) X ([Q3 score/10] X 100) (Reilly et al., 1993).

To provide an example using the aforementioned values: (8/ [8 + 32] + [1- 8]/ {8 + 32} X 100) = (8/40 + [-7/40]) = .20 + (-.175) = .025 X 100 = 2.5; plus 4/10 = .40 X 100 = 40; 2.5 X 40 =100. WPAI scale scores can range from 0.00 to 200.00, with a higher score denoting higher levels of work productivity impairment (Reilly et al., 1993).

There is considerable psychometric support for the WPAI in studies providing evidence of its content, construct, criterion-relatedness, and discriminant validity and test-retest reliability (Beaton et al., 2009; Holloway et al., 2014; Mattke, Balakrishnan, Bergamo, & Newberry, 2007; Prasad, Wahlqvist, Shikiar, & Shih, 2004; Reilly et al., 1993; Tang, Beaton, Boonen, Gignac, & Bombardier, 2011; Zhang et al., 2010). Reilly et al. (1993) reported that the WPAI was best structured as a one-factor scale, providing evidence of its construct validity. Significant correlations between the WPAI and similar health-related work productivity instruments (e.g., Work Limitations Questionnaire, Worker Productivity Index, Valuation of Lost Productivity questionnaire) and measures of work absenteeism, work functioning, and work-related stress and fatigue provide support for the criterion-related concurrent validity of the WPAI (Reilly et al., 1993; Beaton et al., 2010; Holloway et al., 2014; Zhang et al., 2010). Zhang et al. (2010) provided support for the discriminant validity of the WPAI in findings that showed lower mean scores in patients with less severe rheumatoid arthritis symptoms as compared to patients with more severe symptoms. The unusual coding of the WPAI precludes the use of Cronbach's alpha, due to low reliability, to determine its inter-item reliability (Reilly et al., 1993). Studies have shown that the test-retest reliability of the WPAI ranges from rs = .70 to .75, ps < .001 (Holloway et al., 2014; Prasad et al., 2004; Tang et al., 2011).

Covariates. I selected covariates based on empirical evidence documenting associations between these variables and SLE disease activity and damage, depression, as well as work productivity impairment (Abu Baker, Shaharir, Mohamed Said, & Mohd, 2018; Baker & Pope, 2009; Bertoli et al., 2015; Cosatti et al., 2018; Drenkard et al., 2013, 2014a; Holloway et al., 2014; Jordan et al., 2018; Mazzoni & Cicognani, 2011; Mazzoni, Cicognani, & Prati2017; Utset et al., 2015; Zhang et al., 2017). The operational definition and coding of the potential covariates are presented in the following sections.

Covariate: Work status. Work states was a dichotomous variable coded where 0 = work full-time and 1 = work part-time.

Covariate: Gender. Gender was a dichotomous variable coded where 0 = female and 1 = male.

Covariate: Race. Race was categorically coded where 1 = Black, 2 = White, and 3 = Other Racial Group.

Covariate: Poverty status. Poverty status was a dichotomous variable coded where 0 = below poverty level and 1 = above poverty level.

Covariate: Marital status. Marital status was a categorically-coded variable, where 1 = single/never married, 2 = married, 3 = no longer married (i.e., widowed, divorced, or separated), and 4 = living with partner.

Covariate: Age. Age was an interval-coded variable, and ages ranged from 22.4 to 73.7 years.

Covariate: Years of education. The years of education variable was interval-coded, and years of education ranged from 8 to 23 years.

Covariate: Health insurance. In accordance with Drenkard et al. (2014a), the potential covariate of health insurance was coded and reported as a categorical variable where 1 = private, 2 = no insurance, 3 = Medicaid, 4 = Medicare, 5 = military benefits, and 6 = Medicaid and Medicare.

Covariate: Out-of-pocket medical costs. The potential covariate of out-of-pocket medical care expenses was measured using the GOAL study survey question, "How much have you spent out-of-pocket on medical expenses over the past 12 months?" This is a categorically-coded variable, where 1 = none, 2 = $1 to $199, 3 = $200 to $499, 4 = $500 to $999, and 5 = $1,000 or more.

Covariate: Time since SLE diagnosis. Time since SLE diagnosis was coded as an interval variable in years, and time since SLE diagnosis ranged from 0.1 to 49.4 years.

Research Questions and Hypotheses

This study had three research questions. The first research question is descriptive and queried about SLE-related work productivity impairment. The second and third research questions were inferential and examine whether depression mediated the relationship between SLE disease activity and damage and work productivity impairment.

Research Question 1 (RQ1): What are the SLE-related issues (activity and damage) that impact work productivity impairment among GOAL cohort participants?

Research Question 2 (RQ2): Is SLE disease activity associated with work productivity impairment among GOAL participants (2.1), and does depression mediate the relationship (2.2)?

$H_0$2a: There is no statistically significant association between SLE disease activity and work productivity impairment among GOAL participants.

H_a2a*:* There is a statistically significant association between SLE disease activity and work productivity impairment among GOAL participants.

$H_0$2b: There is no statistically significant mediation effect of depression on the relationship between SLE disease activity and work productivity impairment among GOAL participants.

H_a2b: There is a statistically significant mediation effect of depression on the relationship between SLE disease activity and work productivity impairment among GOAL participants.

Research Question 3 (RQ3): Is SLE disease damage associated with work productivity impairment among GOAL participants (3.1), and does depression mediate the relationship (3.2)?

$H_0$3a: There is no statistically significant relationship between SLE disease damage and work productivity impairment among GOAL participants.

H_a3a: There is no statistically significant mediation effect of depression on the relationship between SLE disease damage and work productivity impairment among GOAL participants.

$H_0$3b: There is a statistically significant relationship between SLE disease damage and work productivity impairment among GOAL participants.

H_a3b: There is a statistically significant mediation effect of depression on the relationship between SLE disease damage and work productivity impairment among GOAL participants.

Data Analysis Plan

I used the GOAL (2014) data set that had been reviewed, cleaned, and organized by Emory University GOAL analysts and then sent to me as an Excel file via email. The data analysis plan commenced once I transferred the GOAL study data from an Excel file to an SPSS 25.0 data file. I utilized SPSS 25.0 to conduct all statistical analyses. I structured the data analysis plan to address both the descriptive and analytical research questions of the study. I conducted the data analyses following a sequential process, with analyses performed in steps.

Cleaning and Organization of Data Set

The first step of the data analyses pertained to data cleaning and organization. I first reviewed the data set to ensure that it contained data only from GOAL participants who reported working full- or part-time at the time of data collection. I found that the data set included data from GOAL study participants who were not employed at the time of data collection. I removed the data from the participants who were not employed at the time of data collection. I then identified multivariate outlier cases by computing Mahalanobis distances values for each case by conducting a multiple linear regression (MLR), with the SLAQ, SA-BILD, PHQ-9, and WPAI variables predicting a randomly-selected variable (age). I removed multivariate outlier cases. Finally, I examined the data set for missing values. The SLAQ and SA-BILD variables, which measured SLE

disease activity and damage, respectively, had no missing values. The PHQ-9 variable, which measured depression, had two missing values. The two missing values were replaced using linear interpolation.

Computation of Study Scales and Descriptive Statistics

The second step of the data analysis plan entailed the computation of study scales and descriptive statistics for study variables and covariates. I computed the SLAQ, SA-BILD, PHQ-9, and WPAI scales per instrument authors' guidelines. I then then conducted the descriptive statistical analyses on the SLAQ, SA-BILD, PHQ-9, and WPAI scales, computing the mean, median, standard deviation, and minimum and maximum scores. I also computed the descriptive statistics for the covariates. I calculated and reported the frequencies and percentages for the dichotomous/categorical variables (e.g., ethnicity, gender, poverty status) and the mean, median, standard deviation, minimum and maximum scores for the interval-coded variables (e.g., age, years of education).

Testing of Assumptions

The third step of the data analysis plan involved the testing of data assumptions. Linear regression (LR) and hierarchical linear regression (HLR) have assumptions regarding the data that must be met to ensure the accuracy of statistical findings (Nimon, 2012; Osborne, 2013). There are five key assumptions for LR/HLR: (a) univariate/ multivariate normality; (b) homoscedasticity, (c) linearity between the predictor/mediating and criterion variables, (d) lack of multicollinearity between the predictor and mediating variables; and (e) independence of errors (Nimon, 2012;

Osborne, 2013). The following sections include information on the testing of these assumptions.

Assumption of univariate/multivariate normality. To assess univariate normality, I computed $z_{skewness}$ values of the SLAQ, SA-BILD, PHQ-9, and WPAI by dividing the skewness value by the skewness standard error (Nimon, 2012). A $z_{skewness}$ value less than 3.29 ($p < .001$) indicates that the variable displays relative normality (Nimon, 2012). I recomputed skewed variables via logarithmic transformation, used when the variable is positively skewed and is ratio-coded (i.e., can have a value of 0) (Tabachnick & Fidell, 2013).

Assumption of homoscedasticity. The assumption of homoscedasticity refers to the equal distribution of criterion variable residual (error) scores across all predictor variable data points (Nimon, 2012; Osborne, 2013). I tested if data met the assumption of homoscedasticity by computing three scatterplots of standardized predicted versus actual residuals, one for the SLAQ-WPAI relationship, one for the SA-BILD-WPAI relationship, and one for the PHQ-9-WPAI relationship. The equal dispersal of residuals above and below a horizontal zero value on the scatterplot indicates that the assumption of homoscedasticity is met (Tabachnick & Fidell, 2013). LR/HLR models are robust against a violation of the homoscedasticity assumption (Nimon, 2012; Osborne, 2013).

Assumption of linearity. Another assumption for regression models is linearity between the predictor and criterion variables (Ernst & Albers, 2017). I tested for this assumption by computing three P-P (probability) plots of standardized predicted versus actual residuals, one for the SLAQ-WPAI relationship, one for the SA-BILD-WPAI

relationship, and one for the PHQ-9-WPAI relationship. A uniform distribution of residual data points along a diagonal indicates that the linearity assumption is met (Ernst & Albers, 2017; Tabachnick & Fidell, 2013). A violation of the linearity assumption is serious, as statistical results can increase the chance of committing a Type I error or rejecting the null hypothesis when in fact it should be retained (Ernst & Albers, 2017). One means to address the violation of the linearity assumption is to logarithmic transform the variable (Ernst & Albers, 2017; Tabachnick & Fidell, 2013).

Assumption of lack of multicollinearity. Multicollinearity, in which the predictor variables are so highly correlated that they essentially measure the same construct (O'Brien, 2007), was a possibility in this study. I tested if data met the assumption of lack of multicollinearity among predictor and mediating variables by computing variance inflation factors (VIF) for the SLAQ, SA-BILD, and PHQ-9. VIFs less than 4.00 indicate that the assumption of lack of multicollinearity is met (O'Brien, 2007).

Assumption of independence of errors. The fifth and last assumption tested was the independence of errors, or lack of autocorrelation of residuals (Jupiter, 2017; Osborne, 2013). I tested this assumption by computing Durbin Watson values. Errors are independent if the Durbin Watson values are between 1.00 and 3.00 (Tabachnick & Fidell, 2013). The assumption of independence of errors is a concern for longitudinal data (Jupiter, 2017; Osborne, 2013). This study was cross-sectional, reducing the likelihood of a violation of the assumption of independence of errors (Osborne, 2013).

Dummy Coding of Nominal/Categorical Variables

The numerical values for nominal/categorical variables are simply used to distinguish each category, level, or condition and cannot be interpreted as measuring strength or intensity (Nimon, 2012; Tabachnick & Fidell, 2013). For example, if a categorical variable of race/ethnicity is coded 1=*White,* 2=*Black,* and 3=*Hispanic*, the value of '3' is not 'higher' or 'better' than '1.' A value of '3' simply means that the participant identifies as Hispanic. In other words, categorical variables cannot be utilized as interval or ratio variables (Tabachnick & Fidell, 2013). Per the requirements for LR/HLR (Tabachnick & Fidell, 2013) I recoded all categorical variables into dummy-coded dichotomous variables, where each category was compared to the other categories (see Table 1).

Table 1

Dummy Coding: Proposed Covariates

Variable	*Dummy Coding*
Work status	Full-time = 0, Part-time = 1
Gender	Male = 0, Female = 1
Below Poverty Status	No = 0, Yes = 1
Ethnicity	
Black	No = 0, Yes = 1
White	No = 0, Yes, 1
Other Racial Group	No = 0, Yes =1
Marital Status	
Never Married	No = 0, Yes = 1
Married	No = 0, Yes = 1
No Longer Married	No = 0, Yes = 1
Living with Partner	**No = 0, Yes = 1**
Type of Health Insurance	
None	No = 0, Yes = 1
Private	No = 0, Yes = 1
Medicaid	No = 0, Yes = 1
Medicare	No = 0, Yes = 1
Military	No = 0, Yes = 1
Medicaid & Medicare	No = 0, Yes = 1
Out-of-pocket medical expenses	
$0	No = 0, Yes = 1
$1-$199	No = 0, Yes = 1
$200-$499	No = 0, Yes = 1
$500-$999	No = 0, Yes = 1
$1000 or more	No = 0, Yes = 1

Testing of Covariates

Covariate testing entailed the calculation of a series of point biserial and Pearson bivariate correlation analyses between pertinent demographic, healthcare, and health variables and the WPAI work productivity impairment variable. I conducted a series of point biserial correlation analyses between the dummy-coded variables and the WPAI work productivity impairment variable. A point biserial correlation, denoted as $r_{pb,}$ is used to examine the relationship between a "true dichotomous variable," which in this study were the dummy-coded potential covariates, and "a continuous variable," which was the WPAI measure of work productivity impairment (Dănăcică, 2017, p. 154). Pearson bivariate correlations are conducted to examine relationships between interval - or ratio-coded variables (Tabachnick & Fidell, 2013). I conducted Pearson correlations between the confound variables of age and SLE disease duration and work productivity impairment, measured using the WPAI.

Analysis for Research Question 1

I addressed the first research question of the study by computing and providing descriptive statistics (i.e., mean, median, standard deviation, minimum and maximum scores) for the WPAI variables, including (a) the WPAI total impairment scale, (b) the WPAI impairment scale, and (c) the WPAI active impairment scale.

Analysis for Research Questions 2 and 3.

The analyses that I conducted to address the second and third research questions were Pearson bivariate correlations and LRs and HLRs for mediation, conducted in accordance with recommendations by Baron and Kenny (1986). The predictor, mediator,

and criterion variables must all show significance with each other (at $p < .05$) in order to meet the basic requirement of mediation (Baron & Kenny, 1986). Therefore, the first step of the mediational process involved the computation of Pearson bivariate correlations (Barron & Kenny, 1986) between (a) the SLAQ and SA-BILD predictor and the WPAI criterion variables, (b) the SLAQ and SA-BILD predictor and the mediating PHQ-9 variables, and (c) the PHQ-9 mediating and the WPAI criterion variables. If all three relationships were significant (at $p < .05$), a series of LR/HLRs for mediation were conducted in accordance with Barron and Kenny (1986). In accordance with recommendations, to confirm if mediation was present, a Sobel test was conducted using an online publicly-available calculator (https://www.danielsoper.com/statcalc/calculator.aspx?id=31) (Baron & Kenny, 1986).

Threats to Validity

The merits of a quantitative study and its statistical findings depend on its internal, external, and statistical conclusion validity (Creswell, 2009; Kumar & Acharya, 2014). Each type of validity has associated threats, some of which are more common in survey and cross-sectional studies (Delgado-Rodriguez & Llorca, 2004;Levin, 2006). The following sections provide reviews of internal, external, and statistical conclusion validity and associated threats.

Internal Validity

Internal validity is “the extent to which systematic error is minimized” in a study to allow for a more precise assessment of associations among study variables (Pannucci & Wilkins, 2010, p. 623). Threats to internal validity are sources of bias or systematic

error (Delgado-Rodriguez & Llorca, 2004; Kumar & Acharya, 2014). The selection bias is a concern for epidemiological studies using non-probability sampling strategies (Tyrer & Heyman, 2016). Convenience sampling lessens the likelihood of obtaining a sample that is representative of the population (Acharya, Prakash, Saxena, & Nigam, 2013; Kumar & Acharya, 2014).

As individuals self-selected to participate in the GOAL study, they may have differed from those who chose not to participate on certain demographic, work, and health factors. GOAL study researchers implemented certain methodological procedures empirically known to minimize selection bias effects (Drenkard et al., 2014a). The GOAL study had clear and precise participant inclusion criteria. GOAL researchers utilized different sources of the population (i.e., the GLR; Emory and the Grady SLE clinics; and community rheumatologists) from which to recruit study participants, and they implemented three types of recruitment procedures (Drenkard et al., 2014a).

One internal validity threat, the *healthy worker effect (HWE)*, is associated with the selection bias (Chowdhury, Shah, & Payal, 2017), and it was a concern in this study. The HWE refers to the fact that healthier study participants are more likely to be employed, and they tend to have lower morbidity and mortality rates when compared to non-employed participants (Chowdury et al., 2017). Moreover, employment influences other factors, such as income level and access to healthcare and healthcare coverage. Resultantly, the HWE increases the likelihood that the study sample is not representative of the population (Chowdury et al., 2017; Shah, 2009). The HWE is less of a concern when the participants have similar work statuses (Shah, 2009). Due to the focus on work

productivity impairment, this study only included data from GOAL study participants who worked full- or part-time at the time of data collection. As full- versus part-time work may have qualitative differences, work status was controlled for statistically, as were demographic factors associated with the HWE (e.g., gender, annual household income, type of health insurance) (Chowdury et al., 2017; Shah, 2009).

Another threat to internal validity is the *information bias*, in which inaccurate measurement of study constructs introduces systematic error into the study (Kumar & Acharya, 2014; Pannucci & Wilkins, 2010). A specific type of information bias pertinent in studies that use self-report instruments is the social desirability bias (Althubaiti, 2016). The social desirability bias refers to the tendency of study participants to answer self-report survey questions in a more socially-acceptable and less truthful way (Althubaiti, 2016). GOAL study researchers implemented certain methodological procedures known to minimize this bias (Drenkard et al., 2014a; Platinga et al., 2016). One, they utilized extensively-validated gold-standard self-report instruments commonly used in SLE studies (Drenkard et al., 2014a; Platinga et al., 2016). Two, they ensured that medical and health information reported by the participants aligned with respective patient medical chart data when possible (Drenkard et al., 2014a, 2014b; Platinga et al., 2016).

Confounding, in which the presence of an extraneous variable distorts the true relationships among examined variables, is a concern for nonexperimental studies (Kumar & Acharya, 2014; Pannucci & Wilkins, 2010). Confounding can exaggerate, minimize, or hide the true associations among study variables (Kumar & Acharya, 2014;

Pannucci & Wilkins, 2010). The testing and use of covariates helped to minimize this bias (Tabachnick & Fidell, 2013).

The final internal validity threat of concern in this study is *causal ambiguity* (Pannucci & Wilkins, 2010). This is a threat specific to cross-sectional studies and refers to the inability to determine temporal precedence (i.e., that the predictor and mediating variables preceded the criterion variable) (Levin, 2006). Little can be done to minimize this threat when using a cross-sectional research design (Levin, 2006).

External Validity

External validity pertains to the generalizability of study findings (Drost, 2011; Zyphur & Pierides, 2017). The three key threats to the external validity of a quantitative study concern the study sample/population, study ecology, and specificity of variables (Zyphur & Pierides, 2017). The *threat of population validity* refers to the inability to generalize findings beyond the population represented by the study sample. The likelihood of a threat to population validity increases in proportion to the specificity of the study sample (Levin, 2006; Zyphur & Pierides, 2017). The sample for this study was GOAL study participants, all of whom were adults who have SLE and resided in the state of Georgia. This study was further limited to GOAL study participants who are employed full- or part-time. Findings from this study cannot be generalized to other populations, including (a) children with SLE; (b) SLE patients from specific ethnic groups not well-represented in the GOAL data; and (c) individuals with SLE who are not employed or who are retired, homemakers, or students.

The threat of *ecological validity* refers to the inability to generalize study findings to populations in other settings (Drost, 2011; Zyphur & Pierides, 2017). Settings can pertain to the geographical location and the type of setting in which the study was conducted (Drost, 2011; Zyphur & Pierides, 2017). It is uncertain if the findings from this study can be generalized to SLE populations that reside outside the state of Georgia. This is because the GOAL study participants are a subset of the Georgia Lupus Registry (Drenkard et al., 2014a). Therefore, the selected sample may be distinct from other SLE population groups who have yet to be studied epidemiologically.

The majority of GOAL participants completed the study survey online, and as such, it cannot be assumed that similar results would emerge in studies utilizing different data collection procedures. The threat of *specificity of variables* refers to the inability to generalize findings to other research settings that utilize different assessments and/or operational definitions of study constructs (Drost, 2011; Zyphur & Pierides, 2017). A strength of this study was the utilization of gold standard questionnaires that have been extensively validated and frequently utilized in studies on SLE (e.g., Abu Bakar et al., 2018; Cosatti et al., 2017; Dhanhani et al., 2014; Utset et al., 2015) and with GOAL cohorts (Drenkard et al., 2014a, 2014b; Jordan et al., 2018). It cannot, however, be stated that study findings using different measures of lupus activity and damage, depression, and work productivity would be the same as found in this study.

Statistical Conclusion Validity

Statistical conclusion validity concerns the degree to which correct statistical conclusions can be drawn from the analysis of data (De Vaus & De Vaus, 2013; Garcia-

Pérez, 2012). The most pertinent statistical conclusion threats are factors that increase the likelihood of making a Type I error, by either rejecting or failing to reject the null hypothesis (De Vaus & De Vaus, 2013; Garcia- Pérez, 2012). The Type II error rate is inversely related to study power: the error rate decreases as power increases equaling 1-power (Tabachnick & Fidell, 2013). In contrast, power increases as the sample and effect sizes (i.e., the magnitude of association between variables) increase (Tabachnick & Fidell, 2013). Results from an *a priori* power analysis indicated that a minimum sample size of $N = 135$ was needed to achieve adequate power of 80%. As violations of statistical assumptions and poor reliability of study instruments can increase the likelihood of making a Type I error (Drost, 2011; Tabachnick & Fidell, 2013), the data analyses included the testing and addressing of these two threats to statistical conclusion validity.

Ethical Procedures

GOAL study researchers followed ethical guidelines for research conducted with human subjects, including obtaining informed consent from study participants. I followed the ethical guidelines for the treatment of human subjects as they pertained to (a) IRB approval, (b) the use of a secondary data set, and (c) the treatment of data and related study materials. I first sought and was granted IRB approval from Walden University prior to obtaining the data and conducting the statistical analyses for the study. I also sought and received IRB approval from Emory University to conduct this study. I followed a specific data access and use protocol in accordance with Emory University, namely (a) identifying the study variables necessary to conduct the study; (b) submitting

an online data access request form to Emory GOAL researchers; and (c) retrieving data from the GOAL study data sets. To address ethical concerns, I received permission to use a de-identified and anonymous data set. Email transmission of the data set is encrypted and password-protected. I saved the data set on a password-protected jump-drive (and not on a computer hard drive), which she kept in a locked file cabinet in her home office, along with study-related materials (e.g., GOAL study survey template, SPSS 25.0 data, syntax, and output paper documents). The jump-drive will be destroyed and study-related materials shredded once five years have passed.

Transition and Summary

The purpose of section 2 was to provide a comprehensive overview of the proposed study methodology. The key topics discussed in this section were the (a) research design and rationale; (b) theoretical framework; (c) target population and sample, including results from the power analysis; (d) sampling strategy, recruitment, and data collection procedures; (e) study instruments and operationalization of study variables; (f) research questions and associated hypotheses; (g) data analysis; (h) validity and threats to validity; and (i) ethical procedures. The sections to come include a presentation of study findings and a discussion of these findings.

Section 3: Presentation of the Results and Findings

Introduction

SLE, an autoimmune inflammatory disease, can place burdens on affected individuals, particularly with regards to their ability to work (Gallop et al., 2012). The multisystemic health problems and organ damage, often progressive in nature, and cognitive and mental dysfunctions associated with SLE often impede work productivity outcomes, including higher rates of absenteeism and reduced performance while at work (Al Dhanhani et al., 2014; Baker & Pope, 2009; Cosatti et al., 2017; Drenkard et al., 2014a; Garris et al., 2015). Depression, common among patients with SLE, can further contribute to absenteeism and poor work productivity (Auberbach & Beckerman, 2012; Shen et al., 2013).

Although scholars have noted significant associations between SLE disease activity and damage and depression (Jordan et al., 2018; Zakeri et al., 2012) and work impairment (Cooper et al., 2009; Garris et al., 2013), few researchers have examined the potential mediating effects of depression on the relationship between SLE disease activity and damage and work productivity impairment. The relationships between SLE disease activity and damage, depression, and work activity impairment are not well understood (Drenkard et al., 2014a; Jordan et al., 2018).

This study, which used 2014 GOAL study data, contributed to and elaborated upon prior empirical work that has suggested that depression may mediate the relationship between SLE disease activity and damage and work productivity impairment (Abu Bakar et al., 2018; Al Dhanhani et al., 2014; Cosatti et al., 2017; Utset et al., 2015).

The purpose of the study was to identify, define, and describe SLE-related issues in association with depression and work productivity impairment among 261 GOAL participants. The central focus of this study was to test the proposed mediational model of SLE disease activity and damage, depression, and work productivity impairment. The mediational model is presented in Figure 3.

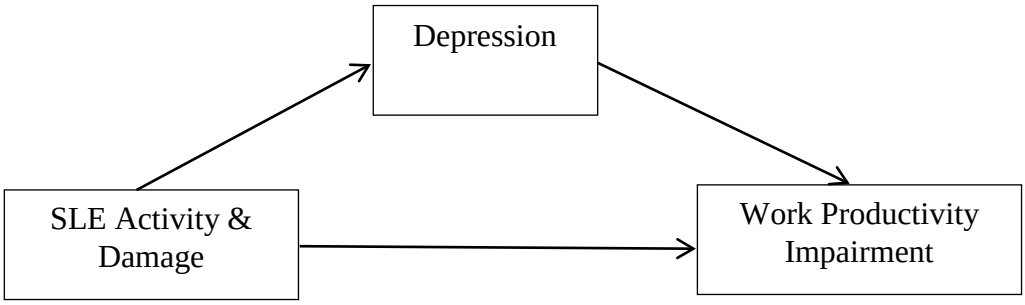

Figure 3. Hypothesized mediational model.

Research Questions and Associated Hypotheses

This study had three research questions. The first research question was descriptive, and as such, did not have associated null and alternative hypotheses (Salkind, 2010). The second and third research questions were analytical and had associated null and alternative hypotheses.

Research Question 1 (RQ1): What are the SLE-related issues (activity and damage) that impact work productivity impairment among GOAL cohort participants?

Research Question 2 (RQ2): Is SLE disease activity associated with work productivity impairment among GOAL participants (2.1), and does depression mediate the relationship (2.2)?

$H_0$2a: There is no statistically significant association between SLE disease activity and work productivity impairment among GOAL participants.

H_a2a*:* There is a statistically significant association between SLE disease activity and work productivity impairment among GOAL participants.

$H_0$2b: There is no statistically significant mediation effect of depression on the relationship between SLE disease activity and work productivity impairment among GOAL participants.

H_a2b: There is a statistically significant mediation effect of depression on the relationship between SLE disease activity and work productivity impairment among GOAL participants.

Research Question 3 (RQ3): Is SLE disease damage associated with work productivity impairment among GOAL participants (3.1), and does depression mediate the relationship (3.2)?

$H_0$3a: There is no statistically significant relationship between SLE disease damage and work productivity impairment among GOAL participants.

H_a3a: There is no statistically significant mediation effect of depression on the relationship between SLE disease damage and work productivity impairment among GOAL participants.

$H_0$3b: There is a statistically significant relationship between SLE disease damage and work productivity impairment among GOAL participants.

H_a3b: There is a statistically significant mediation effect of depression on the relationship between SLE disease damage and work productivity impairment among GOAL participants.

Section 3 presents the statistical findings of the study. Section 3 opens with a restatement of the GOAL study recruitment and data collection procedures, followed by a description of the study participants. Substantial attention is then given to the testing of data assumptions and study covariates, with statistical results reported and summarized. The study results, which include findings from Pearson bivariate correlations and LR and HLR analyses for mediation conducted to test study hypotheses, are then presented. A summary concludes the section.

Data Collection of the Secondary Data Set

I used 2014 GOAL study data. The GOAL study is a longitudinal cohort of individuals with a validated diagnosis of SLE, primarily derived from the CDC-funded, population-based GLR (Drenkard et al., 2013, 2014a). Funded by the CDC, the overarching goal of the GOAL study is to gain a better understanding of the burden of lupus and health disparities in high-risk lupus populations, including the impact of SLE disease activity and damage on patients' work and mental health outcomes. GOAL study investigators recruited study participants having a validated diagnosis of SLE from the GLR, Emory and Grady SLE clinics, and participating community rheumatologists. Patients who provided informed consent were enrolled into the GOAL study. GOAL study data collection primarily entailed the completion of validated patient-reported instruments at least on an annual basis. Participants could complete the GOAL survey

online, in person with assistance from a GOAL research coordinator during a scheduled clinic meeting, or via a phone call with a GOAL research coordinator (Drenkard et al., 2013, 2014a).

The procedures to obtain and use the 2014 archival GOAL data set were conducted as proposed in Section 2 of this document. Receipt of the 2014 archival GOAL data set was dependent upon the signing of MTA between Walden University and Emory University, the school responsible for all GOAL data. In September 2018, the MTA was signed by a Walden University administrator and submitted via e-mail to Emory University GOAL administrators. Upon approval of the MTA at the end of September 2018, a GOAL research administrator e-mailed an Excel data file to me. This data file included only the variables identified in the study for all GOAL study participants who completed the survey in 2014. The data were transferred from Excel to SPSS 25.0.

Removal of Cases and Final Sample Size

The original 2014 GOAL study data set contained data from 715 SLE patients (*n* = 565 [79.0%] Black, *n* = 136 [19.0%] White, *n* = 8 [1.1%] Asian American, *n* = 4 [0.6%) Native Hawaiian, and *n* = 2 [0.3%] Native American). The study focuses on work productivity impairment necessitated the removal of 454 (63.5%) participant cases who were not employed at the time of data collection. The resultant sample included 261 participant cases, 36.5% of all 2014 GOAL study participants. Per the approved methodology, I identified multivariate outlier cases by computing Mahalanobis distances values for each of the 261 cases by conducting a MLR, with the SLAQ, SA-BILD, PHQ-

9, and WPAI active impairment variables predicting a randomly-selected variable (age). As the MLR had four predictors, the Mahalanobis distance critical value was 18.38 (Tabachnick & Fidell, 2013). Four cases were identified as having Mahalanobis distance values greater than 18.38 (i.e., values of 83.23, 63.18, 21.59, and 19.88). Three of these multivariate outliers were White while one – which had the highest distance value of 83.23 – was an Asian American participant. These four cases were removed from the data set, reducing the number to 257, the final sample size. A post hoc power analysis, conducted using G*Power (Faul et al., 2009) with significance set at $p < .05$, medium effect size, $f^2 = .15$, and power set at .95, with 3 predictors, indicated 95% power.

Descriptive Statistics: Study Participants

Frequency and percentage distributions were calculated on participant work status, demographic, and health insurance data, with results presented in Table 2 to Table 5. Table 2 presents the descriptive data for the nominal work and demographic variables. Of the 257 participants, the majority worked full-time (n = 201, 78.2%), were of Black ethnicity (n = 187, 72.8%), female (n = 242, 94.2%), and above poverty level (n = 199, 77.4%). An almost equal number of participants were single/never married (n = 94, 36.0%) or married (n = 92, 35.8%). Fewer numbers of participants were divorced (n = 41, 16.0%), separated (n = 10, 3.9%), or widowed (n = 3, 1.2%).

Table 2

Frequencies & Percentages: Demographic Information (N = 257)

Variable	*Frequency* *N*	*Percentage* *%*
Work Status		
Full-time	201	78.2
Part-time	56	21.8
Ethnicity/Race		
Black	187	72.8
White	64	24.9
Asian	4	1.6
Native Hawaiian	1	0.4
American Indian	1	0.4
Gender		
Female	242	94.2
Male	15	5.8
Below Poverty Level		
No	199	77.4
Yes	37	14.4
Missing	21	8.2
Marital Status		
Single/Never Married	94	36.6
Married	92	35.8
Divorced	41	16.0
Living with Partner (Not Married)	16	6.2
Separated	10	3.8
Widowed	3	1.2
Missing	1	0.4

Table 3 presents the descriptive statistics for the two continuously-coded demographic variables of age at survey and years of education. All 257 participants

provided their age. The mean age of participants was 44.8 years (*Md* = 44.5 years, *SD* = 11.1 years), and participants ranged between 22.4 and 73.7 years of age. Of the 254 participants who provided their total years of education, the mean was 15.7 (*Md* = 16 years, *SD* = 3.1 years), equivalent to an associate's degree/some college education. Years of education ranged from 8 (equivalent to seventh grade) to 23 (equivalent to a doctorate degree).

Table 3

Descriptive Statistics: Age (Years) at Survey and Years of Education

	N	*M*	*Md*	*SD*	*Minimum*	*Maximum*
Age (Years) at Survey	257	44.8	44.5	11.1	22.4	73.7
Years of Education	254	15.7	16.0	3.1	8.0	23.0

Participants provided information on their health insurance coverage and yearly out-of-pocket health expenses. The frequencies and percentages for health insurance categories are presented in Table 4. The majority of participants (*n* = 180, 70.0%) had private health insurance, while 46 (17.9%) participants reported having no health insurance coverage. Smaller numbers of participants received Medicaid (*n* = 9, 3.5%), Medicare (n = 11, 4.3%) or both Medicaid and Medicare (*n* = 5, 1.9%). Six (2.3%) participants received military health benefits.

Table 4 also presents information on participants' yearly out-of-pocket health expenses. The smallest group of participants (*n* = 10, 3.9%) reported having no yearly out-of-pocket health expenses. Over one-fifth of participants reported spending $1-$199

(*n* = 54, 21.0%) or $200-$499 (*n* = 68, 26.5%), respectively, on out-of-pocket health expenses. Almost half (*n* = 125, 48.6%) of the participants reported spending from $500 to $1000 or more on out-of-pocket health expenses on a yearly basis.

Table 4

Frequencies & Percentages: Health Insurance and Out-of-Pocket Health Expenses (N = 257)

Variable	*Frequency* *N*	*Percentage* *%*
Type of Health Insurance		
Private	180	70.0
None	46	17.9
Medicaid	9	3.5
Medicare	11	4.3
Military	6	2.3
Medicare & Medicaid	5	1.9
Out-of-Pocket Health Expenses (Yearly)		
$0	10	3.9
$1-$199	54	21.0
$200-$499	68	26.5
$500-$999	46	17.9
$1000 or more	79	30.7

The final covariate examined was participants' reported disease duration, or number of years since their initial SLE diagnosis. As seen in Table 5, the mean disease duration was 14.1 years (*Md* = 13 years, *SD* = 8.9 years). Disease duration ranged from 0.1 to 49.4 years.

Table 5

Descriptive Statistics: SLE Disease Duration (Years) (N = 257)

	M	*Md*	*SD*	*Minimum*	*Maximum*
SLE Disease Duration (Years)	14.1	13.0	8.9	0.1	49.4

Descriptive Statistics: Study Measures

The study had four measures: (a) the SLAQ (Karlson et al., 2003), which measured SLE disease activity; (b) the SA-BILD (Drenkard et al., 2014a), which assessed SLE disease damage; (c) the PHQ-9 (Kroenke et al., 2001), a measure of depression; and (d) the WPAI (Reilly et al., 1993), which assessed work productivity impairment. The dataset was reviewed to determine if any of the study measures had missing data values. The SLAQ and SA-BILD instruments, which measured SLE disease activity and damage, respectively, had no missing individual values. The PHQ-9 instrument, which measured depression, had two missing individual values. The two missing data points were imputed using linear interpolation, a widely used and accepted imputation method (Zhang, 2016).

The data set included three WPAI scales: (a) the WPAI total impairment scale, (b) the WPAI impairment scale, and (c) the WPAI active impairment scale. The WPAI total impairment percent scale had 45 missing values, the WPAI impairment percent scale had 13 missing values, and the WPAI active impairment percent scale had six missing values. To determine which of the three WPAI variables should be used in analyses for hypothesis testing, a series of Pearson bivariate correlations were conducted, and

variance inflation factors (VIFs) were calculated between the three WPAI variables to assess if these variables displayed multicollinearity. A correlation of $r > .80$, $p < .001$ is indicative of the presence of multicollinearity, whereas a correlation of $r < .80$, $p > .001$ is indicative of the absence of multicollinearity (Tabachnick & Fidell, 2013). The second statistical test was the computation of VIFs. A VIF measures the degree to which "the estimated variance of the nth regression coefficient is increased above what it would be if R^2 equaled zero" due to high correlations among variables (O'Brien, 2007, p. 42). A VIF > 4.0 indicates high variance among predictors and resultant multicollinearity while a VIF < 4.0 indicates acceptable variance among predictors and lack of multicollinearity (O'Brien, 2007; Tabachnick & Fidell, 2013). I stopped reviewing here due to time constraints. Please go through the rest of your section and look for the patterns I pointed out to you. I will now look at your Section 4.

Results from the Pearson bivariate correlations and VIFs are presented in Table 6. The three WPAI variables were highly correlated with one another, all exceeding $r > .80$, $p < .001$. These high correlations indicated multicollinearity between the three WPAI scales. The WPAI total impairment percent scale had a VIF of 26.4, and the WPAI impairment percent scale had a VIF of 25.1, indicative of multicollinearity. The WPAI active impairment percent scale had an acceptable VIF value of 3.0. Moreover, the WPAI active impairment scale had the fewest missing values ($n = 6$). Data were imputed using linear interpolation, and the imputed WPAI active impairment percent variable, henceforth called the WPAI variable, was used in all analyses.

Table 6

Pearson Bivariate Correlations and VIFs: WPAI Total Impairment Percent, WPAI Impairment Percent, & WPAI Active Impairment Percent Variables (N = 257)

	WPAI % Total Impairment	*WPAI % Impairment*	*VIF*
WPAI Percent Total Impairment	--		26.4
WPAI Percent Impairment	.98***	--	26.1
WPAI Percent Active Impairment	.82***	.81 ***	3.0

Note. ***$p < .001$

Descriptive Statistics: SLAQ, SA-BILD, WPAI, and PHQ-9 Variables

Table 7 presents the descriptive statistics for the four study variables. The SLAQ measure of SLE disease activity had a mean of 12.8 (*Md* = 12, *SD* = 7.9), and SLAQ scores ranged from 0 to 34 points. The SA-BILD measure of SLE disease damage had a mean of 1.9 (*Md* = 1, *SD* = 1.9), and SA-BILD scores ranged from 0 to 23 points. The PHQ-9 measure of depression had a mean of 6.2 (*Md* = 5, *SD* = 5.1), and PHQ-9 scores ranged from 0 to 24 points. Of the 257 participants, 40 (15.6%) had a PHQ-9 score between 10 or 14, indicative of moderate depression, 19 (7.0%) had a PHQ-9 score between 15 and 19, denoting moderately severe depression, and 4 (1.6%) had a PHQ-9 score of 20 or higher, indicative of severe depression. The WPAI percent active impairment measure of work productivity had a mean of *M* = 38.9 (*Md* = 40, *SD* = 31.8), and WPAI scores ranged from 0 to 100 points.

Table 7

Descriptive Statistics: SLAQ, SA-BILD, PHQ-9, and WPAI (N = 257)

	M	*Md*	*SD*	*Minimum*	*Maximum*
SLAQ	12.8	12.0	7.9	0	34
SA-BILD	1.9	1.0	1.9	0	9
PHQ-9	6.2	5.0	5.1	0	24
WPAI	38.9	40.0	31.8	0	100

Note. The potential range of SLAQ scores is 0 to 44, with a higher score denoting higher SLE disease activity (Karlson et al., 2003). The SA-BILD can range from 0 to 37 points, with a higher score denoting a higher degree of perceived SLE damage (Drenkard et al., 2014a). For the PHQ-9, scores between 0 and 4 indicates no to minimal depression, scores between 5 and 9 indicate mild depression, scores between 10 and 14 indicate moderate depression, scores between 15 and 19 indicate moderately severe depression, and scores between 20 and 27 indicate severe depression (Kroenke et al., 2001). WPAI percent active impairment scale scores can range from 0 to 200, with a higher score denoting higher levels of work productivity impairment (Reilly et al., 1993).

Testing of Covariates

I conducted a series of statistical analyses for covariate testing. The potential covariates in this study were demographic factors (i.e., gender, race/ethnicity, highest level of education, poverty status, marital status), employment status (i.e., part- or full-time), length of time since SLE diagnosis, and healthcare variables (i.e., type of health insurance and out-of-pocket medical expenses), which have been found to be significantly associated with work productivity impairment in prior studies (Holloway et al., 2014; Jordan et al., 2018; Karol et al., 2013; Utset et al., 2015; Zhang et al., 2017). Most of the potential covariates were categorical; these nominal variables were recoded into new dichotomized dummy variables, with each category coded as ‘0’ = *no* or ‘1’ = *yes*.

For covariate testing, point biserial correlations were calculated to examine if there were any significant associations between the dummy-coded demographic, health, and healthcare variables and the WPAI work productivity impairment scale. A point biserial correlation, denoted as $r_{pb,}$ is used to examine the relationship between a "true dichotomous variable," which in this study were the dummy-coded potential covariates, and "a continuous variable," which was the WPAI measure of work productivity impairment (Dănăcică, 2017, p. 154). As denoted in Table 8, there were no significant point biserial correlations.

Table 8

Point Biserial Correlations: Work Status, Ethnicity, Gender, Poverty Status, and Marital Statuses and WPAI Work Productivity Impairment (N = 257)

Variable	*WPAI Work Productivity Impairment*	
	r_{pb}	*P*
Work status: Full-time = 0, Part-time = 1	.08	.216
Ethnicity: Black = 0, Not Black = 1	.12	.051
Ethnicity: White = 0, Not White = 1	.11	.094
Ethnicity: Other = 0; Not Other = 1	.09	.141
Gender: Male = 0, Female = 1	.04	.485
Below Poverty Status: No = 0, Yes = 1	.09	.162
Never Married: No = 0, Yes = 1	.03	.619
Married: No = 0, Yes = 1	-.04	.488
No Longer Married: No = 0, Yes = 1	.01	.852
Living with Partner: No = 0, Yes = 1	-.02	.792

Three potential covariates, age, years of education and SLE disease duration, were continuously-coded variables, as was the WPAI work productivity impairment variable,

which required the use of Pearson bivariate correlation analyses. As noted in Table 9, the Pearson bivariate correlation results indicated no significant associations between ages, years of education, and SLE disease duration and work productivity impairment.

Table 9

Pearson Bivariate Correlations: Age, Years of Education, SLE Disease Duration, and WPAWork Productivity Impairment (N = 257)

Variable	*WPAI Work Productivity Impairment*	
	r	*P*
Age	.09	.135
Years of Education	-.12	.061
SLE Disease Duration	-.02	.800

A final set of point biserial correlations were conducted between the dummy-coded health insurance and out-of-pocket medical expenses variables and the WPAI work productivity impairment variable, presented in Table 10. Neither set of variables was significantly associated with work productivity impairment.

Table 10

Point Biserial Correlations: Type of Health Insurance and Out-of-pocket Medical Expenses and WPAI Work Productivity Impairment Variable (N = 257)

Variable	*WPAI Work Productivity Impairment*	
	r_{pb}	*P*
Type of Health Insurance		
No Insurance: No = 0, Yes = 1	.03	.646
Private Insurance: No = 0, Yes = 1	-.09	.141
Medicaid: No = 0, Yes = 1	.02	.751
Medicare: No = 0, Yes = 1	.12	.063
Military: No = 0, Yes = 1	.06	.321
Medicaid & Medicare: No = 0, Yes = 1	-.04	.528
Out-of-pocket Medical Expenses		
$0: No = 0, Yes = 1	.04	.538
$1-$199: No = 0, Yes = 1	-.05	.468
$200-$499: No = 0, Yes = 1	.02	.743
$500-$999: No = 0, Yes = 1	.02	.721
$1000 or more: No = 0, Yes = 1	-.01	.819

Summary

Covariate testing entailed the calculation of a series of point biserial and Pearson bivariate correlation analyses between pertinent work, demographic, health, and healthcare variables and the WPAI work productivity impairment variable. Results indicated that none of the variables were significantly associated with work productivity

impairment. As such, no covariates were included in the LR and HLRs for mediation, conducted to address the second and third research questions.

There are three main topics specifically addressed in the following sections of the chapter. First, there is a presentation of findings from statistical analyses conducted to test the five key assumptions, discussed below, for LR and HLR. The descriptive statistics conducted to address the first research question are then presented. Results from the LRs and HLRs for mediation for the second and third research question complete this section.

Testing of Assumptions for LR/HLR

Linear regression models have assumptions regarding the data that must be met to ensure the accuracy of statistical findings (Tabachnick & Fidell, 2013). These are: (a) univariate/ multivariate normality; (b) homoscedasticity, (c) linearity between the predictor/mediating and criterion variables, (d) lack of multicollinearity between the predictor and mediating variables; and (e) independence of errors (Tabachnick & Fidell, 2013). Results from analyses testing thee five assumptions are presented in the following sections.

Assumption of Univariate Normality

To assess univariate normality, $z_{skewness}$ values were computed for the SLAQ, SA-BILD, PHQ-9, and WPAI variables. $Z_{skewness}$ values are computed by dividing the skewness value by the skewness standard error (Tabachnick & Fidell, 2013). A $z_{skewness}$ value less than 3.3 ($p < .001$) indicates that the variable displays relative normality (a $z_{skewness}$ value of 3.3 is acceptable, and the variable is considered normally distributed)

(Tabachnick & Fidell, 2013). The $z_{skewness}$ values for the study variables are presented in Table 11. The SLAQ variable had an acceptable $z_{skewness}$ value of 3.3, as did the WPAI variable, $z_{skewness}$ = 2.3. The SLAQ and WPAI variables were considered to have normally-distributed scores around the mean score. Two of the variables were not normally distributed, based on their $z_{skewness}$ values: the SA-BILD, which had a $z_{skewness}$ value of 8.8, and the PHQ-9, which had a $z_{skewness}$ value of 5.8.

Table 11

$Z_{skewness}$ Values: SLAQ, SA-BILD, PHQ-9, and WPAI Variables (N = 257)

Variable	$Z_{skewness}$
SLAQ	3.3
SA-BILD	8.8
PHQ-9	5.8
WPAI	2.3

As the SA-BILD and PHQ-9 variables were substantially positively skewed, they were logarithmically transformed. A logarithmic transformation is used when the variable is positively skewed and is ratio-coded (i.e., can have a value of 0) (Tabachnick & Fidell, 2013). Logarithmic transformation requires adding a constant value (e.g., 2) to the values and then computing the log of the variable (Tabachnick & Fidell, 2013). Once transformed, the two variables showed acceptable levels of normality: the transformed SA-BILD had a $z_{skewness}$ value of 2.32 and the transformed PHQ-9 variable had a $z_{skewness}$ value of -2.13.

Table 12 provides the descriptive statistics for the PHQ-9 and SA-BILD transformed variables. The SA-BILD (transformed) variable had a mean of M = 0.5 (*Md*

= 0.5, *SD* = 0.2), and SA-BILD (transformed) scores ranged from 0.3 to 1.0. The PHQ-9 (transformed) variable had a mean of *M* = 0.8 (*Md* = 0.9, *SD* = 0.3), and PHQ-9 (transformed) scores ranged from 0.3 to 1.4. The transformed SA-BILD and PHQ-9 variables were used in the LR/HLR for mediation analyses conducted for hypothesis testing.

Table 12

Descriptive Statistics: SA-BILD (transformed) and PHQ-9 (transformed) Variables (N = 257)

	M	*Md*	*SD*	*Minimum*	*Maximum*
SA-BILD (transformed)	0.5	0.5	0.2	0.3	1.0
PHQ-9 (transformed)	0.8	0.9	0.3	0.3	1.4

Assumption of Homoscedasticity

The LR/HLR assumption of homoscedasticity refers to the equal distribution of criterion variable residual (error) scores across all predictor variable data points (Tabachnick & Fidell, 2013). I tested whether data met the homoscedasticity assumption by computing three scatterplots of standardized predicted versus actual residuals, one for the SLAQ-WPAI relationship, one for the SA-BILD-WPAI relationship, and one for the PHQ-9-WPAI relationship. The equal dispersal of residuals above and below a horizontal zero value on the scatterplot indicates that the assumption of homoscedasticity is met (Tabachnick & Fidell, 2013). The scatterplots of standardized predicted versus actual residuals for the SLAQ-WPAI, SA-BILD-WPAI, and PHQ-9-WPAI relationships are presented in Figures 4 through 6. As denoted in the scatterplots, the data points were

equally distributed above and below the horizontal zero value, indicating that the assumption of homoscedasticity was met.

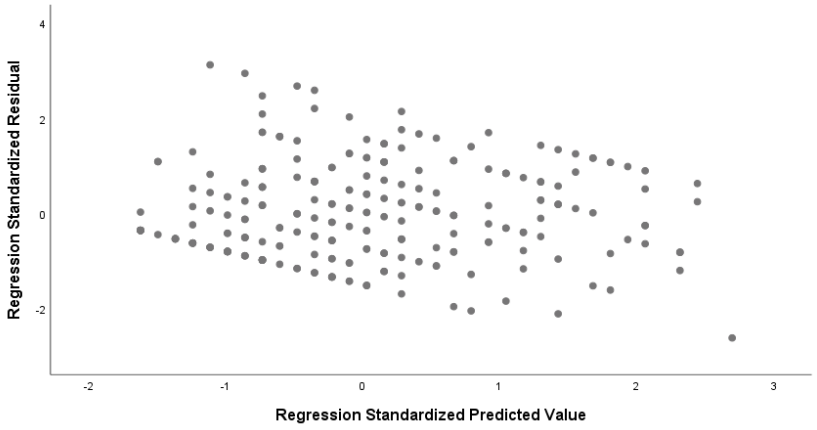

Figure 4. Scatterplot of predicted versus actual residuals: SLAQ and WPAI.

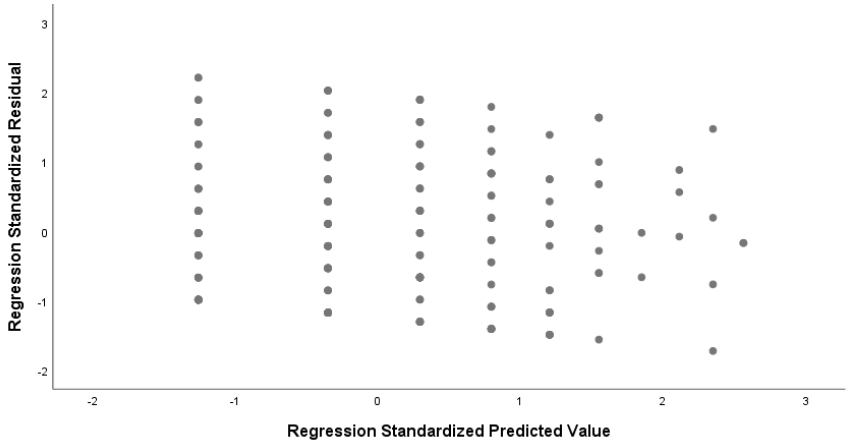

Figure 5. Scatterplot of predicted versus actual residuals: SA-BILD (transformed) and WPAI.

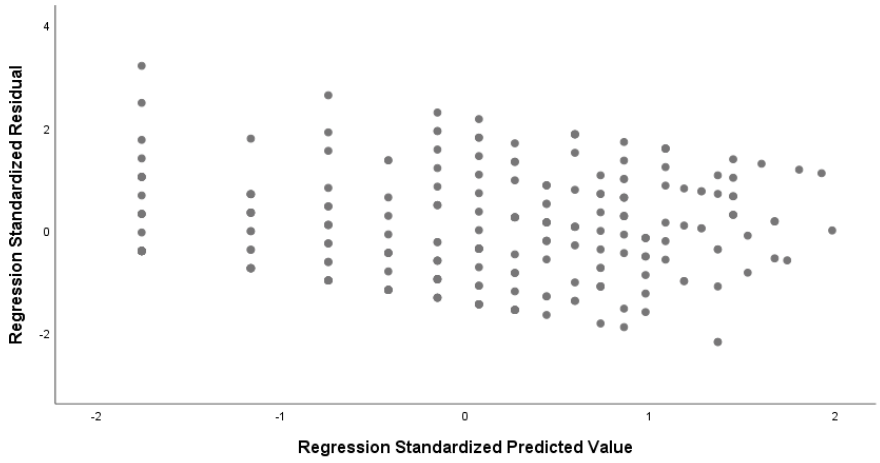

Figure 6. Scatterplot of predicted versus actual residuals: PHQ-9 (transformed) and WPAI.

Assumption of Linearity

The third assumption tested was linearity between the predictor and criterion variables (Tabachnick & Fidell, 2013). To test if the data met the assumption of linearity, I computed three P-P (probability) plots of standardized predicted versus actual residuals, one for the SLAQ-WPAI relationship, one for the SA-BILD-WPAI relationship, and one for the PHQ-9-WPAI relationship. A uniform distribution of residual data points along a diagonal indicates that the linearity assumption is met (Tabachnick & Fidell, 2013). The P-P plots are presented in Figures 7 through 9. As seen in the P-P plots, the residual data points were uniformly distributed along the diagonal. The assumption of linearity was met.

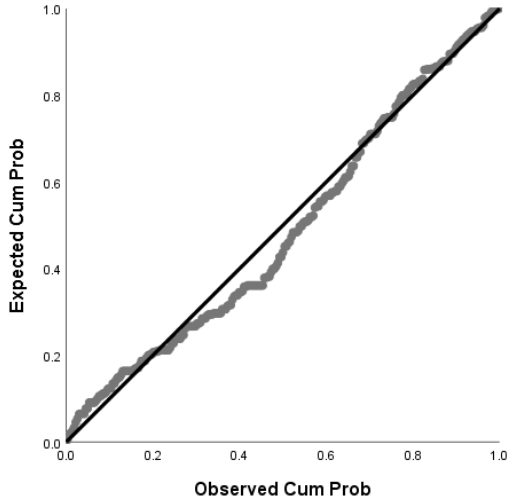

Figure 7. P-P plot of predicted versus actual residuals: SLAQ and WPAI.

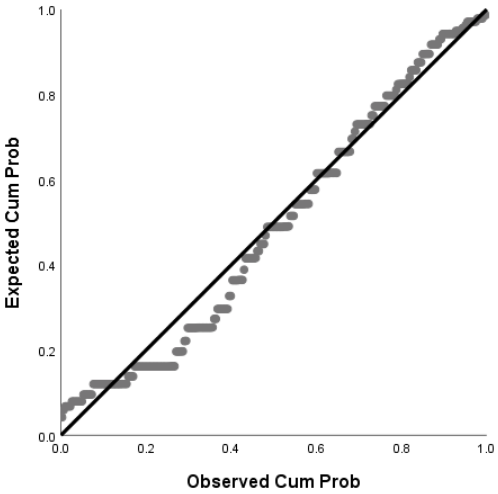

Figure 8. P-P plot of predicted versus actual residuals: SA-BILD (transformed) and WPAI.

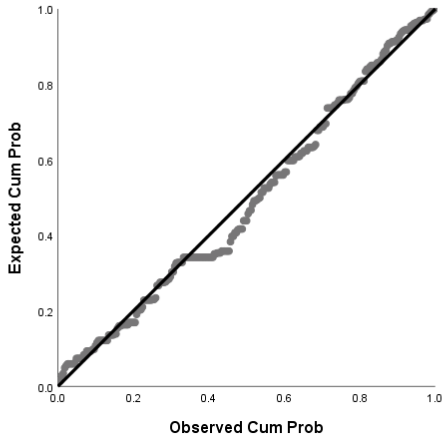

Figure 9. P-P plot of predicted versus actual residuals: PHQ-9 (transformed) and WPAI.

Assumption of Lack of Multicollinearity

The fourth assumption tested was lack of multicollinearity, that is, the SLAQ and SA-BILD (transformed) predictor variables and the PHQ-9 mediating variable are *not* so highly correlated with one another that they essentially measure the same construct. Two statistical tests were conducted to test for lack of multicollinearity. First, a series of Pearson bivariate correlations were conducted among the three variables. A correlation of $r >= .80$, $p < .001$, is indicative of the presence of multicollinearity, whereas a correlation of $r < .80$, $p > .001$, is indicative of the absence of multicollinearity (Tabachnick & Fidell, 2013). The second statistical test was the computation of variance inflation factors (VIFs). A VIF > 4 indicates high variance among predictors and resultant multicollinearity while a VIF < 4 indicates acceptable variance among predictors and lack of multicollinearity (O'Brien, 2007; Tabachnick & Fidell, 2013).

Table 13 presents the Pearson bivariate correlations between the SLAQ, SA-BILD (transformed), and PHQ-9 (transformed) and their VIFs. While the variables were

significantly correlated with one another, the correlations did not exceed $r > = .80$, $p < .001$. All VIFs were below the critical value of 4: the SLAQ had a VIF of 2, the SA-BILD (transformed) had a VIF of 1.1, and the PHQ-9 (transformed) had a VIF of 1.9. These findings indicated that the assumption of lack of multicollinearity was met.

Table 13

Pearson Bivariate Correlations and VIFs: SLAQ, SA-BILD, and PHQ-9 (N = 257)

	SLAQ	*SA-BILD (transformed)*	*PHQ-9 (transformed)*	*VIF*
SLAQ	--			2.0
SA-BILD (transformed)	.29***	--		1.1
PHQ-9 (transformed)	.68***	.18**	--	1.9

Note. **$p < .01$, ***$p < .001$

Assumption of Independence of Errors

Independence of errors, or lack of autocorrelation of residuals, was the fifth and last assumption, and it was tested by computing Durbin Watson values. The assumption of independence of errors is met if the Durbin Watson value is between 1 and 3 (Tabachnick & Fidell, 2013). I computed the Durbin Watson values for the SLAQ-WPAI, SA-BILD (transformed)-WPAI, and PHQ-9 (transformed)-WPAI associations. The Durbin Watson value for the SLAQ-WPAI association was 2.1. The Durbin Watson value for the SA-BILD (transformed)-WPAI association was 1.9, as was the Durbin Watson value for the PHQ-9 (transformed)-WPAI association. As all Durbin Watson values were between 1 and 3, the assumption of independence of errors was met.

Research Question 1 Results

The first research question was **"**What are the SLE-related factors that impact work productivity impairment among GOAL cohort participants?" To address the first research question, descriptive statistics (i.e., mean, median, standard deviation, minimum and maximum scores) were computed for the WPAI question: "What percentage of work hours did you miss in the past 7 days?" Each participant's percentage of hours missed was computed by dividing the numbers of hours missed in the past seven days due to poor health by the total number of hours worked in the past seven days. Descriptive statistics (i.e., mean, median, standard deviation, minimum and maximum scores) were also computed for the WPAI impairment percent and the WPAI active impairment percent variables.

Table 14 presents the descriptive statistics for the three additional WPAI variables. The 214 (83.2%) participants who provided a response regarding hours of work missed had a mean percent of 6.6% (*SD* = 15%) missed work time, equivalent to 3 hours or slightly less than half a day, missed in the past seven days. The median percent of hours of work missed in the past seven days was 0, and the percent of hours of work missed in the past seven days ranged from 0% to 100%. A majority of participants (*n* = 158, 61.5%) reported no (0) hours of work missed in the past seven days. However, seven (3.3%) of participants had percent scores between 50% and 100% hours of work missed in the past seven days.

As seen in Table 14, the mean score for the 211 (82.1%) participants with a WPAI total impairment percent score was 34.1 (*Md* = 30, *SD* = 30.4). The WPAI total

impairment percent scores ranged from 0% to 100%. Fifty-six (21.8%) participants reported no impairment, as indicated by a value of zero (0). However, four (1.6%) participants had a WPAI impairment percent score of 60%, seven (2.7%) had a percent score of 70%, and three (1.2%) were completely work impaired, as indicated by a score of 100%. The 244 (94.9%) participants who provided a WPAI impairment percent score had a mean of 33.6 (*Md* = 30, *SD* = 29.5). The WPAI impairment percent scores ranged from 0% to 100%. Sixty-two (24.1%) participants reported no impairment, as indicated by a value of zero (0), as seen in Table 13. However, 52 (21.3%) had WPAI impairment percent scores between 50% and 70%, 24 (9.4%) participants had WPAI impairment percent scores between 80% and 90%, and seven (2.7%) participants were completely work impaired, as indicated by a WPAI impairment percent score of 100%.

Table 14

Descriptive Statistics: WPAI Variables

	N	*M*	*Md*	*SD*	*Minimum*	*Maximum*
Hours of work missed in past 7 days percent	214	6.6	0	15.1	0	100
WPAI total impairment percent	211	34.1	30	30.4	0	100
WPAI impairment percent	244	33.6	30	29.5	0	100

Research Question 2 Results

The second research question was: “Is SLE disease activity associated with work productivity impairment among GOAL participants (2.1) and if so, does depression

mediate the relationship (2.2)?" A series of Pearson bivariate correlations and linear regression mediational model analyses in accordance were conducted to address the second research question.

Pearson Bivariate Correlations

A series of Pearson bivariate correlations were first conducted to determine if the SLAQ (predictor variable), PHQ-9 (transformed) (mediating variable), and WPAI (criterion variable) were significantly associated with one another (at $p < .05$). Table 15 presents the Pearson bivariate correlations. SLE disease activity, as measured by the SLAQ, was significantly associated with the mediator of depression, as measured by the PHQ-9 (transformed), r (257) = .69, $p < .001$. As SLE disease activity severity increased, so did the level of depression. SLE disease activity, as measured by the SLAQ, was also significantly associated with work productivity impairment, as measured by the WPAI, r (257) = .58, $p < .001$. As SLE disease activity severity increased, so did the degree of work productivity impairment. The mediating variable of depression, as measured by the PHQ-9 (transformed), was significantly associated with work productivity impairment, as measured by the WPAI, r (257) = .50, $p < .001$. As the level of depression increased, so did work productivity impairment severity. These significant correlations indicated that the first requirement for mediation was met.

Table 15

Pearson Bivariate Correlations: SLAQ SLE Disease Activity, PHQ-9 (transformed) Depression, and WPAI Work Productivity Impairment (N = 257)

	SLAQ	*PHQ-9 (transformed)*
SLAQ	--	
PHQ-9 (transformed)	.69***	--
WPAI	.58***	.50***

Note. *** $p < .001$

Linear Regression Mediational Models

In accordance with Baron and Kenny (1986), two linear regression models were built to test for mediation. The first was a simple linear regression (LR), with the SLAQ variable, the measure of SLE disease activity, entered on the first model (step) as a predictor of work productivity impairment, as measured by the WPAI. Table 16 presents the finding from the LR, which was significant, $F(1, 255) = 126.0$, $p < .001$, $R^2 = .33$. SLE disease activity, as measured by the SLAQ, was significantly associated with work productivity impairment, as measured by the WPAI, $\beta(257) = .58$, $p < .001$.

Table 16

Linear Regression Model: SLAQ SLE Disease Activity Predicting WPAI Work Productivity Impairment (N = 257)

Model		*B*	*SE B*	*β*	*p*
1	SLAQ	2.3	0.2	.58	< .001

Note. Model 1: $F(1, 255) = 126.0$, $p < .001$, $R^2 = .33$

The first null hypothesis for the second research question was, "$H_{2.1}$**:** There is no statistically significant relationship between SLE disease activity and work productivity impairment among GOAL participants." LR results indicated that SLE disease activity was significantly associated with work productivity impairment. As such, the first null hypothesis for the second research question was rejected (failed to be retained).

Table 17 presents the finding from the second linear regression model, a HLR, which provides information about mediation effects. Per the requirements for mediation, the PHQ-9 (transformed) variable, was entered on the first HLR model (step), followed by the SLAQ variable on the second HLR model (step). The β coefficient for the SLAQ-WPAI relationship in the LR (Table 16) was compared to the β coefficient for the SLAQ-WPAI relationship in the HLR (after the entry of the PHQ-9 [transformed] variable) (Table 17). If the β coefficient value was reduced in size from the LR to the HLR (after the entry of the PHQ-9 [transformed] variable), partial mediation occurred. If the β coefficient was reduced in size from the LR to the HLR (after the entry of the PHQ-9 [transformed] variable) to the degree that it was no longer significant, full mediation occurred. A Sobel test should be conducted to confirm if mediation is evident, and if so, if the findings indicate partial or full mediation (Baron & Kenny, 1986).

Results from the HLR are presented in Table 17. The first HLR model (step) was significant, $F(1, 255) = 82.9$, $p < .001$, $R^2 = .25$. Depression, as measured by the PHQ-9 (transformed), significantly predicted work productivity impairment, as measured by the WPAI, $\beta(257) = .50$, $p < .001$. The second HLR model (step) was significant, $F(1, 254) = 41.0$, $p < .001$, $R^2 = .11$. In the second HLR model, SLE disease activity, as measured

by the SLAQ, significantly predicted work productivity impairment, as measured by the WPAI, $\beta(257) = .44$, $p < .001$. However, the SLAQ β coefficient was reduced from $\beta = .58$, $p < .001$ (as seen in Table 15) to $\beta = .44$, $p < .001$ (as seen in Table 16). A Sobel test was conducted using an online publicly-available calculator (https://www.danielsoper.com/statcalc/calculator.aspx?id=31). The Sobel test was significant, $t(257) = 7.5$, $p < .001$, confirming that depression acted as a partial mediator between SLE disease activity and work productivity impairment. That is, a higher degree of SLE disease activity contributed to depression severity, which in turn, led to increased work productivity impairment.

Table 17

Hierarchical Linear Regression Models: PHQ-9 (transformed) Depression Predicting Work Productivity Impairment (Model 1), andPHQ-9 (transformed) Depression and SLAQ Disease Activity Predicting WPAI Work Productivity Impairment (Model 2) (N = 257)

Model		*B*	*SE B*	*β*	*p*
1	PHQ-9 (transformed)	52.9	5.8	.50	<.001
2	PHQ-9 (transformed)	20.4	7.4	.19	.006
	SLAQ	1.8	0.3	.44	<.001

Note. Model 1: $F(1, 255) = 82.9$, $p < .001$, $R^2 = .25$
Model 2: $F_{change}(1, 254) = 41.0$, $p < .001$, $R^2_{change} = .11$

The second null hypothesis corresponding to the second research question was, "$H_{2.2}$: There is no statistically significant mediational effect of depression on the relationship between SLE disease activity and work productivity impairment among GOAL participants." LR and HLR results showed that depression significantly mediated

the relationship between SLE disease activity and work productivity impairment. Due to these findings, the second null hypothesis for the second research question was rejected (failed to be retained).

Research Question 3 Results

The third research question was: "Is SLE disease damage associated with work productivity impairment among GOAL participants (2.1) and if so, does depression mediate the relationship (2.2)?" A series of Pearson bivariate correlations and linear regression mediational model analyses were conducted to address the third research question.

Pearson Bivariate Correlations

A series of Pearson bivariate correlations were first conducted to determine if the SA-BILD (transformed) (predictor variable), PHQ-9 (transformed) (mediating variable), and WPAI (criterion variable) were significantly associated with one another (at $p < .05$). Table 18 presents the Pearson bivariate correlation findings. SLE disease damage, as measured by the SA-BILD (transformed), was significantly associated with depression, as measured by the PHQ-9 (transformed), $r(257) = .18$, $p = .004$. As the degree of SLE disease damage increased, so did the level of depression. SLE disease damage, as measured by the SA-BILD (transformed), was also significantly associated with work productivity impairment, as measured by the WPAI, $r(257) = .20$, $p < .001$. As the degree of SLE damage increased, so did the level of work productivity impairment. The mediating variable of depression, as measured by the PHQ-9 (transformed), was significantly associated with work productivity impairment, as measured by the WPAI,

$r(257) = .50$, $p < .001$. As the rate of depression increased, so did the level of work productivity impairment. These significant correlations indicated that the first requirement for mediation was met.

Table 18

Pearson Bivariate Correlations: SA-BILD (transformed) SLE Disease Damage, PHQ-9 (transformed) Depression, and WPAI Work Productivity Impairment (N = 257)

	SA-BILD	*PHQ-9 (transformed)*
SA-BILD	--	
PHQ-9 (transformed)	.18**	--
WPAI	.20***	.50***

Note. **$p < .01$; *** $p < .001$

Linear Regression Mediational Models

In accordance with Baron and Kenny (1986), two linear regression models were built to test for mediation. The first was a LR, where the SA-BILD (transformed) variable, the measure of SLE disease damage, was entered in the first and only model (step) of the LR as a predictor of work productivity impairment, as measured by the WPAI.

Table 19 presents the finding from the LR. The LR model was significant, $F(1, 255) = 10.8$, $p = .001$, $R^2 = .04$. SLE disease damage, as measured by the SA-BILD (transformed) significantly influenced work productivity impairment, as measured by the WPAI, $\beta(257) = .20$, $p = .001$. As the degree of SLE disease damage increased, so did the level of work productivity impairment.

Table 19

Linear Regression Model: SA-BILD (transformed) Disease Damage Predicting WPAI Work Productivity Impairment (N = 257)

Model		*B*	*SE B*	*β*	*P*
1	SA-BILD (transformed)	33.0	10.1	.20	.001

Note. Model 1: $F(1, 255) = 10.8$, $p = .001$, $R^2 = .04$

The first null hypothesis for the third research question was, "$H_{3.1}$: There is no statistically significant relationship between SLE disease damage and work productivity impairment among GOAL participants." LR results were significant, indicating that SLE disease damage was significantly associated with work productivity impairment. As such, the first null hypothesis for the third research question was rejected (failed to be retained).

Table 20 presents the finding from the HLR. Per the requirements for mediation, the mediator of depression, as measured by the PHQ-9 (transformed), was entered in the first HLR model (step), followed by the predictor of SLE disease damage, as measured by the SA-BILD (transformed), in the second HLR model (step). The β coefficient for the SA-BILD (transformed)-WPAI relationship in the LR (Table 19) was compared to the β coefficient for the SA-BILD (transformed)-WPAI relationship in the HLR (after the entry of the PHQ-9 [transformed] variable) (Table 20). If the β coefficient value was reduced in size from the LR to the HLR (after the entry of the PHQ-9 [transformed] variable), partial mediation occurred. If the β coefficient was reduced in size from the

LR to the HLR (after the entry of the PHQ-9 [transformed] variable) to the degree that it was no longer significant, full mediation occurred.

Results from the HLR for mediation are presented in Table 20. The first HLR model (step) was significant, $F(1, 255) = 82.9$, $p < .001$, $R^2 = .25$. Depression, as measured by the PHQ-9 (transformed), significantly predicted work productivity impairment, as measured by the WPAI, $\beta(257) = .50$, $p < .001$. The second HLR model (step) was significant, $F_{change}(1, 254) = 4.5$, $p = .04$, $R^2_{change} = .01$. Depression, as measured by the PHQ-9 (transformed), remained a significant predictor of work productivity impairment, as measured by the WPAI, $\beta(257) = .48$, $p < .001$. SLE disease damage, as measured by the SA-BILD (transformed), was significantly associated with work productivity impairment, as measured by the WPAI, $\beta(257) = .12$, $p = .035$. The SA-BILD (transformed) β coefficient was reduced from $\beta = .20$, $p = .001$ (as seen in Table 18) to $\beta = .12$, $p = .035$ (as seen in Table 19). A Sobel test was conducted using an online publicly-available calculator (https://www.danielsoper.com/statcalc/calculator.aspx?id=31) to confirm full mediation. The Sobel test was significant, $t(257) = 2.8$, $p = .006$, confirming that depression acted as a partial mediator between SLE disease damage and work productivity impairment. That is, a higher degree of SLE disease damage resulted in a higher depression rate, which in turn, led to increased work productivity impairment.

Table 20

Hierarchical Linear Regression Models: PHQ-9 (transformed) Depression Predicting Work Productivity Impairment (Model 1), andPHQ-9 (transformed) Depression and SA-BILD (transformed) Disease Damage Predicting WPAI Work Productivity Impairment)(Model 2)(N =257)

Model		*B*	*SE B*	*β*	*P*
1	PHQ-9 (transformed)	52.9	5.8	.50	<.001
2	PHQ-9 (transformed)	50.7	5.9	.48	<.001
	SA-BILD (transformed)	19.1	9.0	.12	.035

Note. Model 1: $F(1, 255) = 82.9$, $p < .001$, $R^2 = .25$
Model 2: $F_{change}(1, 254) = 4.5$, $p = .035$, $R^2_{change} = .01$

The second null hypothesis of the third research question was, "$H_{3.2}$: There is no statistically significant mediational effect of depression on the relationship between SLE disease damage and work productivity impairment among GOAL participants." LR and HLR results showed that depression significantly mediated the relationship between SLE disease damage and work productivity impairment. Due to these findings, the null hypothesis for the third research question was rejected (failed to be retained).

Summary

This quantitative cross-sectional research study was conducted using data from the 2014 GOAL study from 257 participants with SLE who were in the workforce at the time of data collection. A *post hoc* power analysis indicated that the power of the study was excellent, 95%. The majority of participants worked full-time (78.2%), identified as Black (72.8%) and female (94.2%), and had private health insurance (70%). The participants were, on average, 45 years of age, and they had an average of 15.7 years of education, equivalent to some college experience. Almost half (48.6%) of the participants had yearly out-of-pocket health expenses of $500 or higher. Participants reported living with SLE for an average of 14 years. Covariate analyses revealed no significant associations between participants' demographic, work, health, and healthcare factors and their work productivity impairment, as measured using the WPAI. The data were analyzed to determine if they met assumptions for linear regression models. All assumptions were met, with the exception of normality for SA-BILD disease damage and PHQ-9 depression scores. These two variables were logarithmically transformed for use in the linear regression for mediation models, which included one LR and one HLR.

The first purpose of the study was to identify, define, and describe SLE-related issues that impact work productivity among GOAL study participants with SLE who were in the workforce at the time of data collection. The first research question was addressed by calculating descriptive statistics on four additional WPAI variables. Participants reported a mean percentage of almost seven hours, equivalent to slightly less

than one day, of work missed in the past seven days. The WPAI scores denoted mild to moderate levels of work productivity impairment.

This study examined if there were significant relationships between the two predictor variables of SLE disease activity, as measured by the SLAQ, and SLE disease damage, as measured by the SA-BILD (transformed), and the criterion variable of work productivity impairment, as measured by the WPAI. Findings from LRs indicated that both SLE disease activity and damage were significantly associated with work productivity impairment. As such the first set of null hypotheses for the second and third research questions were rejected (failed to be retained). This study further examined whether depression, as measured by the PHQ-9 (transformed) variable, mediated between SLE disease activity, as measured using the SLAQ, and damage, as measured using the SA-BILD (transformed) and work productivity, as measured by the WPAI. LRs and HLRs were conducted in accordance with Baron and Kenny (1986) and Sobel tests were conducted to confirm mediation findings. Statistical findings indicated that depression partially mediated between SLE disease activity and work productivity impairment. Moreover, depression partially mediated between SLE disease damage and work productivity impairment. Due to the findings from the LRs and HLRs, the second set of null hypotheses for the second and third research question was rejected (failed to be retained).

The purpose of this chapter was to provide information on the descriptive and inferential statistical findings of the study. In this quantitative study, three research questions were examined. The first research question was descriptive, and as such, did

not have associated null and alternative hypotheses. The second and third research questions were analytical, and thus had associated null and alternative hypotheses. Based on mediation findings, both sets of null hypotheses for the second and third research question were rejected (failed to be retained). The purpose of the following and last section, Section 4, is to provide interpretations of these findings and to discuss the findings in relation to the guiding theory of the study and prior research studies. Recommendations for application and future empirical work are also discussed in the last section of this dissertation study.

Section 4: Application to Professional Practice and Implications for Social Change

Introduction

SLE (or lupus) is an autoimmune rheumatic disease that often emerges during the most productive years of an individual's life (Lim & Drenkard, 2015; Utset et al., 2015). The clinical course of SLE is chronic and unpredictable, and SLE can affect all systems and organs of the body and cause considerable and, at times, irreversible, organ damage (Alarcón, 2008;; Askanase et al., 2013; Baker & Pope, 2009). The progressive nature of SLE and resultant health impairments can lead to increasing rates of absenteeism and diminished work productivity, both of which can result in job loss and/or permanent disability (Baker & Pope, 2009; Cosatti et al., 2017; Utset et al., 2015). Between 15% and 40% of SLE patients are unemployed within 5 years of diagnosis (Cosatti et al., 2017; Drenkard et al., 2014a; Utset et al., 2015). Because work productivity impairment and unemployment are highly prevalent among individuals with SLE, indirect costs related to loss of income can increase the burden on individuals and society as a whole (Barber & Clarke, 2017).

The empirical literature on SLE-related work impairment and disability is extensive enough to have warranted a review of the literature in 2009 (Baker & Pope, 2009), and there has been ongoing empirical examination of SLE disease activity and damage and work productivity, work impairment, and work disability outcomes in the past 10 years (Abu Bakar et al, 2018; Cosatti et al., 2017; Drenkard et al., 2014a; Garris et al., 2013; Utset et al., 2015; Yelin et al., 2009; Zhang et al., 2010). Furthermore, SLE damage and activity and depression have been examined within the context of work

impairment and disability (Drenkard et al., 2014b; Utset et al., 2015; Zakeri et al., 2012). However, no scholars have examined depression as a mediator between SLE disease activity and damage and work impairment, as this study did. The study, conducted using data from 257 GOAL study participants with SLE who were in the workforce at the time of data collection, had three overarching goals: (a) to identify, define, and describe SLE-related issues that impact individuals' work productivity; (b) to determine whether there were significant relationships between SLE disease activity and damage and work productivity impairment; and (c) to assess if depression mediated the relationship between SLE disease activity and damage and work productivity impairment.

I used 2014 GOAL study data from 257 patients who reported working full- or part-time. Participants were, on average, almost 45 years of age, had almost 16 years of formal education (equivalent to an associate's degree/some college education), and had lived with SLE for an average of 14 years. The majority of participants worked full-time (78.2%), identified as Black (72.8%) and female (94.2%), were above poverty level (77.4%), and had private health insurance (70.0%). Although the majority of patients had health insurance coverage, almost half of them (48.6%) had yearly out-of-pocket health expenses of $500 or higher.

I used gold standard instruments frequently used in SLE studies (e.g., Castrejón et al., 2014; Drenkard et al., 2014a, 2014b; Jordan et al., 2018; Yazdany et al., 2011; Zhang et al., 2010). I found that the SA-BILD measure of SLE damage and the PHQ-9 depression measure were positively skewed, and they were therefore loglinearly transformation for correlational and linear regression analyses. The instrument used to

assess SLE disease activity, the SLAQ, and the WPAI work productivity impairment instrument was not skewed and did not need to be transformed.

A series of preliminary analyses were conducted for covariate testing and testing the assumptions of LR and HLR, used in hypothesis testing. In covariate testing, I found no significant findings. In results from the statistical testing of the assumption of normality, I indicated that the SA-BILD and PHQ-9 variables were positively skewed (denoting that a higher number of participants reported lower scores). Logarithmic transformation of these two variables reduced the skewness to acceptable levels, and the transformed SA-BILD and PHQ-9 were used in LR/HLR analyses. The assumptions of homoscedasticity, linearity, lack of multicollinearity, and independence of errors between predictor variables were met. The results from a series of Pearson bivariate correlations indicated significant relationships among all four study variables, meeting the requirements for mediation.

LRs and HLRs for mediation were conducted for hypothesis testing. I found that both SLE disease activity and damage were significantly associated with work productivity impairment. As such the first set of null hypotheses for the second and third research questions were rejected (failed to be retained). LRs and HLRs for mediation were conducted in accordance with Baron and Kenny (1986), with Sobel tests confirming mediation findings. I found that depression partially mediated the relationship between both SLE disease activity and damage and work productivity impairment. Due to these significant findings, the second set of null hypotheses for the second and third research question was rejected (failed to be retained).

The remainder of this last section of the dissertation project elaborates on the study findings. Section 4 is a review of the study findings within the context of the guiding theory, Bandura's (1986) SCT, and prior SLE literature pertinent to this study. Section 4 continues with a discussion on the study limitations follows, followed by recommendations for future research and implications for professional practice and social change. Section 4 ends with a conclusion.

Interpretation of Findings

Interpretation of Findings: Guiding Theoretical Framework

The theoretical framework that informed this study was Bandura's (1986) SCT. According to the SCT, learning occurring within the social context and behavior is a result of the dynamic and reciprocal interaction between the person and his/her environment. In accordance with Bandura's (1986) concept of reciprocal determinism, environmental stimuli and reinforcement contingencies influence and interact with personal attributes of personality characteristics; cognitive factors; and skills to influence the nature, frequency, and intensity of behavior. SCT has been used extensively in the public health and epidemiological empirical literature to examine a range of topics, from chronic disease to ongoing prevention efforts (Glanz & Bishop, 2010). Moreover, depression is a key barrier "to behavior change that negatively affects chronic disease" outcomes (Sell, Amella, Mueller, Andrews, & Wachs, 2016, p. 2).

Findings from this study provided support for SCT, in that both SLE disease activity and SLE disease damage are products of the person interacting with her/his environment and influence work-related behavior, specifically, work productivity

impairment. According to the SCT, individual beliefs, social cues, and reinforcement techniques can be employed to achieve goal-directed behavior that can be maintained over time (Bandura, 1986, 2004, 2005). Depression may be highest among individuals who perceive their SLE symptoms to be severe and unmanageable, and these individuals may be more likely to experience work impairments and have lower work productivity levels (Nowicka-Sauer et al., 2018). The actual objective disease activity and damage (as assessed, for example, by a physician) may be less important than the patient's own beliefs about his/her SLE disease activity and damage (as measured via self-report) in predicting depression and subsequent work outcomes. This idea is suggested in the study findings: significant relationships were found among SLE disease activity and damage, depression, and work productivity impairment despite minimal to moderate levels of dysfunction reported by the patients. In contrast, individuals who seek out resources and supports to manage their SLE symptoms, especially during the earlier stages of the disease, may experience lower levels of depression and enhanced work productivity (Cleanthous, Newman, Shipley, Isenberg, & Cano, 2013; Connolly, McNally, Moran, & Ryan, 2014).

Bandura (1986) identified two key factors shown to minimize behavioral responses: the individual factor of self-efficacy and the environmental factor of social support. Self-efficacy, both general (as an indicator of resilience) and specific to disease management and control treatment adherence, acts to prevent, reduce or buffer against depression and helps to maintain work productivity among SLE patients (Mazzoni & Cicognani, 2011; Mazzoni et al., 2016; Náfrádi et al., 2017; Nowicka-Sauer et al., 2018).

Social support, inclusive of instrumental, informational, and emotional support, plays a protective – buffering – role in the management of SLE while problematic social support plays a detrimental role (Mazzoni & Cicognani, 2011; Mazzoni et al., 2016). This study did not include any self-efficacy or social support variables. However, these participants may have high levels of self-efficacy and/or high levels of social support that has assisted in the management of their SLE and perhaps helped to lessen the negative physical and cognitive sequelae of SLE and enhanced their work productivity.

SCT is often used as a framework for medical interventions (Bandura, 2004, 2005). Within the context of SLE, interventions aimed at improving self-efficacy and increasing social support and resources in relation to disease management, treatment adherence self-efficacy at the time of diagnosis and during the disease adaptation period may be helpful in reducing depression symptoms and lessening the severity of depression, which may reduce work-related problems and impairments (Bandura, 2004, 2005). Health interventions aimed at increasing SLE patients' self-efficacy efforts and mobilization of social support resources have been beneficial in help patients manage the numerous issues that surround SLE and its treatment (Boehmer et al., 2016; Marks, 2014; Náfrádi et al., 2017). As empirical findings with regard to the effects of social support on depression rates among SLE patients have been equivocal (Jordan et al., 2018; Mazzoni et al., 2016), there is a need for future studies to distinguish the effects of self-efficacy and social support with regard to SLE patients' experiences of depression and work productivity.

Interpretation of Findings: Prior literature

This study contributed to and elaborated upon prior empirical work (Abu Bakar et al., 2018; Al Dhanhani et al., 2014; Cosatti et al., 2017; Drenkard et al., 2014a; Jordan et al., 2018; Utset et al., 2015). Many of the previous studies used the same instruments, especially the WPAI (Abu Bakar et al., 2018; Bertolli et al., 2018; Cosatti et al., 2017), or conducted their study using GOAL study data (Drenkard et al., 2014a; Jordan et al., 2018). These empirical similarities allowed for better comparisons of findings, contributed to a deeper understanding of topics, and enhanced the external validity (i.e., generalizability) of the study.

One issue that was immediately evident in this study was the high disability-related unemployment rate among SLE patients. The specificity of this study to employed SLE patients required the removal of data from 63.5% of the GOAL study cases. Only 36.5% of the 2014 GOAL study participants worked, and of the participants who did work, almost a quarter (21.8%) worked part-time. The loss of participants and the relatively high rate of part-time workers in this study are both indicative of higher rates of work disability in patients with SLE, which is supported in prior literature (Abu Bakar et al, 2018; Baker & Pope, 2009; Bultink et al., 2008; Drenkard et al., 2014a).

This study utilized gold standard instruments to assess study constructs: (a) the SLAQ measure of SLE disease activity; (b) the SA-BILD measure of SLE disease damage; (c) the PHQ-9 measure of depression, and (d) the WPAI measure of work productivity impairment. The common use of these measures in SLE studies (Castrejón, 2014) allowed for excellent comparisons of findings in this and other studies. In this

study, SLAQ and SA-BILD descriptive statistics indicated that participants reported mild-to-moderate levels of SLE disease activity and damage. Reports of mild-to-moderate SLE disease activity and damage is not uncommon in studies, especially those conducted with working individuals, and the SLAQ and SA-BILD mean, median, and range of scores reported in this study were similar to those denoted in prior empirical literature on SLE (e.g., Katz, Trupin, Rush, & Yazdany, 2014; Wolfe et al., 2010; Yazdany et al., 2009, 2011, Yelin et al., 2017).

The PHQ-9 was used to measure depression in this study. PHQ-9 scores can range from 0 to 27, and a score of 10 is considered to be indicative of clinical depression (Kroenke et al., 2001). In this study, the PHQ-9 mean score was 6.24, indicative of mild depression; this score was similar to PHQ-9 mean scores reported in studies conducted with patients with SLE (Moldovan et al., 2012) and other autoimmune disorders (Amtmann et al., 2015; Milette, Hudson, Baron, & Thombs, 2010). It was also found that almost 25% of participants had PHQ-9 scores of 10 or higher and thus would be considered to have clinical depression. The percentage of 24.2% of patients with clinical depression was higher than the average percentage of 15% for the primary care population (Mitchell, Yadegarfar, Gill, & Stubbs, 2016) bur similar to the 25% prevalence rates of depression reported in studies on SLE (Jordan et al., 2019; Zhang et al., 2017) and other chronic diseases, such as cancer, cystic fibrosis, and inflammatory bowel disease (Hartung et al., 2017; Neuendorf, Harding, Stello, Hanes, & Wahbeh, 2016; Quon, Bentham, Unutzer, Chan, Goss, & Aitken, 2015).

This study examined different elements of work productivity impairment using the WPAI. One component examined was percentage of work missed. While 61.5% of the working participants did not miss any percentage of work in the past seven days, the average percentage of work missed (i.e., absenteeism) was slightly less than half a day, which is quite considerable. The approximately 3 hours of work missed in the past seven days reported by the participants in this study was very similar to the average of 3 hours of worked missed as reported by Cosatti et al. (2017) and the 2.7 hours of work missed as reported by Utset et al. (2014). These consistent findings support the argument that SLE patients who work experience relatively high rates of absenteeism even if, as indicated in this study, SLE disease activity and damage is somewhat moderate (not severe) (Cosatti et al., 2017; Utset et al., 2014). Participants had relatively moderate work productivity impairment, with an average WPAI percent score of 38.9% (of a possible 100%). This finding adds to the existing literature denoting moderate to high work productivity impairment among SLE patients (Abu Bakar et al., 2018; Cosatti et al., 2017; Drenkard et al., 2014a; Utset et al., 2014).

Study results showed that SLE disease activity and damage significantly influenced work productivity impairment directly and indirectly, through the mediator of depression. Simply stated, as SLE disease activity and damage levels increased, so did depression, and in turn, work productivity impairment. These findings suggest that even moderate levels of SLE disease activity and damage can negatively impact SLE patient's mental health and behavioral outcomes. This and prior studies (Abu Bakar et al., 2018; Bertoli et al., 2018; Cosatti et al., 2017; Dhanhani et al., 2014; Drenkard et al., 2014a;

Garris et al., 2013; Utset et al., 2015) have confirmed a significant link between SLE disease activity and damage and work productivity impairment. This is a consistent finding in literature, despite differences across studies with regard to the survey instruments used (e.g., Abu Bakar et al., 2018; Cosatti et al., 2017; Dhanhani et al., 2014; Utset et al., 2015) and patient country of origin (e.g., Abu Bakar et al., 2018; Bertoli et al., 2018; Dhanhani et al., 2014). No studies to date had examined depression as a mediator between SLE disease activity and damage and work impairment, as this study did. However, a few scholars have examined potential and found support for significant associations between SLE disease activity and damage and higher rates of depression (Jordan et al., 2018; Utset et al., 2015) and similar constructs, such as quality of life (Abu Bakar et al., 2018) and cognitive functioning and fatigue (Utset et al., 2015). Previous studies have also shown that SLE patients with higher (as opposed to lower) rates of depression and fatigue were the most work-impaired (Drenkard et al., 2014a; Utset et al., 2015), lending empirical support for the mediational findings in this study.

It is also important to discuss the lack of significant findings with regard to associations between patient demographic and health variables, healthcare variables, and work productivity impairment. One of the more consistent findings in the SLE literature is the significant association between education level and work productivity impairment (Abu Bakar et al., 2018, Cosatti et al., 2017; Utset et al., 2014). This association was not found to be significant in this study. This study did not find any racial/ethnic differences with regard to work productivity impairment. Significant associations between Black ethnicity and higher likelihood of work impairment have been reported in prior studies

(Cosatti et al., 2017; Drenkard et al., 2014a; Utset et al., 2015; Yelin et al., 2009). Moreover, SLE disease duration was not significantly associated with work productivity impairment, despite a quite high mean disease duration length of 14 years. Abu Bakar et al. (2018), who reported similar median disease duration of 12 years, did find significant associations between disease duration and work productivity impairment. However, like this study, Al Dhanhani et al. (2014), Cosatti et al. (2017) and Drenkard et al. (2014a) found no significant associations between disease duration and work productivity impairment.

Limitations of the Study

As with all empirical studies, this study had both strengths and limitations. The power of 95% was exceptionally strong in this study, and it likely improved the internal validity of the study. The testing and use of covariates minimized the potential of confounding effects, in which the presence of an extraneous variable distorts the true relationships between examined variables (Kumar & Acharya, 2014; Pannucci & Wilkins, 2010). The use of GOAL study data – recognized for its rigorous recruitment and data collection procedures and use of gold-standard instruments – enhanced both the internal and external validity of the study. Taken together, the internal and external validity were especially strong for a correlational study.

The use of cross-sectional correlational research design not only precluded the ability to determine causality, it introduced certain threats to internal validity into the study. One internal validity threat was the inability to ensure temporal precedence (i.e., that the predictor and mediating variables preceded the criterion variable) (Grijbovski &

Ivanov, 2015; Nardi, 2018). It may have been that low work productivity contributed to depression, or that depression contributed to more negative SLE symptom expressions and experiences in the study participants. Moreover, this study did not examine work impairment rates over time; impairment rates likely would have increased as the disease progressed among participants.

As noted by Drenkard et al. (2014a), GOAL study participants were "not a true incident cohort," and the use of a convenience sampling may have increased the likelihood of certain methodological biases, including recall bias and social desirability bias (p. 885). The low reported rates of SLE disease activity and damage, depression, and work impairment are not only suggestive of these biases, they indicate that the *healthy worker effect (HWE)* is a concern in this study. The HWE refers to the fact that employed study participants are more likely to be healthy, and they tend to have lower morbidity and mortality rates than non-employed participants (Chowdury, Shah, & Paval, 2017; Shah, 2009).

This study had some external validity issues. The study was not inclusive enough to overcome the *threat of population validity*, or inability to generalize findings beyond the population represented by the study sample (Ioannidis et al., 2014; Woodward, 2013). While study findings were similar to those found in studies conducted with SLE patients from different countries (e.g., Argentina: Bertoli et al., 2018; Malaysia: Abu Bakar et al., 2018; Canada: Dhanhani et al., 2014), study findings cannot be generalized to ethnic minority populations (e.g., Asian, Hispanic, Native American) who were not well-represented or included in this study. The small percentage of Caucasian/White SLE

patients (24.9%) in this study reduced the ability to generalize study findings to Caucasian/White populations, and the use of GOAL study data limited generalizability to SLE patients residing in American states other than Georgia.

Recommendations

The results from this study open many new avenues for empirical research and contribute to the knowledge base concerning patients' perceptions and management of SLE and the relationship between depression and work productivity impairment among SLE patients. This was the first study to examine if depression mediated the relationship between SLE disease activity and damage and work productivity impairment. Replication studies are needed to validate the findings in this study. Longitudinal studies that examine the pathways examined in this study would be especially beneficial; they would address problems regarding lack of temporal precedence inherent to correlational studies and would provide important information regarding the progression of disease activity, depression, and work impairment over time. Multi-year longitudinal studies can yield important findings from intrapersonal, community, and organizational points of view.

The low-to-moderate levels of disease activity and damage and depression were intriguing, as they suggest the presence of buffers and resources not assessed in this study. There is a need to examine interpersonal (i.e., social support and social resources) and intrapersonal (i.e., self-efficacy, coping, religiosity) factors that may play pertinent roles in promoting SLE patients' physical and mental health. No studies to date have examined if the SLE disease activity and damage, depression, and work impairment

pathways differ across different gender, ethnic, or socioeconomic groups. Such studies would advance understanding of the role that these factors play with regard to SLE symptomatology, organ damage, mental health concerns, and work outcomes. There is also little examination of work productivity impairment across different employment positions and types. As there is some empirical evidence supporting the link between job-related factors (i.e., type of position, work-related stress) and work productivity impairment among SLE patients (Al Dhanhani et al., 2014; Cosatti et al., 2017), additional research is needed in this area.

This study utilized data from SLE patients who were employed part- and full-time, and point biserial correlation results indicated that part- versus full-time employment status did not differentially influence work productivity impairment. However, this study may have overlooked aspects of part- versus full-time work that do influence work productivity impairment. SLE patients who work part-time as opposed to full-time may do so for different reasons, both positive and negative, and studies are needed to distinguish these potential differences. Alternatively, the moderate rates of impairment may indicate stress-buffering aspects of the participants' position or place of employment. Prior literature has shown that the type of job and the psychological and physical demands of a job are significantly associated with increased work productivity impairment (Al Dhanhani et al., 2014; Cosatti et al., 2017). It may be that the participants in this study had relatively low-stress and less physically demanding jobs, implemented certain procedures into their workday that allowed them to better function at work, had supervisors who were more understanding of the stressors related to chronic

disease and adjusted work schedules as needed, or worked in organizations that were more health and wellness focused. Organizational-based studies utilizing data from SLE practitioners would be of value, especially those that examine if and how practitioners assess, diagnose, treat, and manage depression among SLE patients, and if and how such practices influence SLE patient health and work outcomes. Lastly, from the policy perspective it is important to consider how local legislation can protect rights of working individuals diagnosed with SLE in Georgia as well as other states.

Implications for Professional Practice and Social Change

This section provides recommendations to professional practice and positive social change implications relevant to GOAL study participants and the broader SLE population. After reviewing GOAL study data, there is an opportunity to reach out to a wider population for finding effective ways to deal with work productivity challenges for individuals with SLE. The findings contribute to the existing information on how patients with SLE cope with the impact of damage and activity on work productivity, depression and potentially job loss (Bertoli et al., 2007). Additionally, applying the findings has the capacity to enhance awareness and understanding on how effective management of depressive symptoms such as through treatment and medication adherence can alter SLE disease outcomes and impact the quality of life of patients coping with the disease (Lin, Caton, & Von Corff, 2003). Ongoing research using the GOAL dataset will also contribute to the body of SLE-related depression literature where depression is multi-factorial and directly or indirectly related to SLE (i.e. stress, finances, and medication side effects).

From the policy perspective, the knowledge gained from this study can be used to influence local, state, and federal healthcare policy makers towards developing more comprehensive support services for individuals with SLE. Currently, there is limited legislation protecting workers rights if an individual is diagnosed with lupus in the state of Georgia. Work productivity is hampered when individuals with SLE are coping with active symptoms, which can be influenced by depressive episodes (Nery et al., 2007). Current policy also does not protect against job loss due to chronic illness such as SLE and associated depressive symptoms which can create an occupational challenge for diagnosed patients.

After the proposal defense is completed, I intend to disseminate the results of this study through multiple venues including presentations, professional conferences, and peer-reviewed journals. I will share the results of the study with Emory University, Division of Rheumatology SLE investigators where the GOAL study is housed. Also, I will discuss with the Emory investigators how the study can be expanded for further analysis outside of the capstone project.

The GOAL Co-Investigator, Dr. Drenkard, has already reviewed the study findings to ensure quality assurance standards. With permission from Dr. Drenkard, I will also share the results of the study with the GOAL participants. The results will be presented in at least two professional conferences. Abstracts will be submitted to the American Public Health Association Conference and the American College of Rheumatology Conference. Lastly, the results will be distributed via publication in an open-access peer-reviewed journal, likely either SLE or occupational-specific. By

disseminating the research to a broader audience, I will also bring greater awareness of the barriers encountered by individuals with SLE in Georgia and associated work productivity challenges. Addressing barriers that depression creates in SLE patients may allow for opportunities for program development leading to positive social change in Georgia communities, which includes broader SLE educational awareness efforts and furthering mechanisms for social support.

Conclusion

This study enhanced the empirical understanding of SLE in relation to work disability and functioning as well as depression and expanded upon the empirical work on these topics (Abu Bakar et al, 2018; Cosatti et al., 2017; Drenkard et al., 2014a; Garris et al., 2013; Karol et al., 2013; McCormick et al., 2018; Utset et al., 2015; Yelin et al., 2009; Zhang et al., 2010, 2017). This study confirmed that SLE patients experience high rates of unemployment: the specificity of this study to employed SLE patients required the removal of data from 63.5% of the GOAL study cases. Study findings further confirmed that SLE disease activity and damage have a "sizeable impact" on work productivity among SLE patients in the workforce (Drenkard et al.., 2014a, p. 885), even when patients report having mild-to-moderate levels of SLE disease activity and damage. For example, study findings showed that participants missed an average of a half a day of work every seven days and had moderate levels of work productivity impairment, as indicated by WPAI scale scores. While absenteeism and work impairment rates were disconcerting, they were nonetheless similar to findings reported in previous SLE studies (Abu Bakar et al, 2018; Baker & Pope, 2009; Cosatti et al., 2017; Drenkard et al., 2014a).

Findings from this study advanced the empirical study of depression among SLE patients, especially with regard to SLE disease activity and SLE damage and work productivity impairment. There is consistent and considerable evidence that SLE patients experience high rates of depression, with depression rates being higher among unemployed than employed individuals with SLE (Zhang et al., 2017). While findings in this study showed that SLE patients had a PHQ-9 mean score of 6.2, indicative of mild depression, almost a quarter (24.2%) of SLE patients had moderate to severe levels of depression. The rates of depression found in this study were similar to those reported in previous SLE studies (Jordan et al., 2019; Zhang et al., 2017).

This study examined if SLE disease activity and SLE damage influenced work productivity impairment directly, and indirectly, by increasing depression levels. This study added to the existing literature that has documented that significant links exist between SLE disease activity and damage and work productivity impairment (Jordan et al., 2019; Zhang et al., 2017). This was, however, the first study to examine and confirm that depression mediated the relationships between SLE activity, SLE damage, and work productivity impairment. That is, higher levels of SLE disease activity and damage led to higher rates of depression, which in turn led to increased work productivity impairment.

This study contributed to the empirical study of SLE and its effect on depression and work productivity and advanced understanding of the effects of SLE disease activity and damage on depression and work productivity impairment. It is hoped that this study acts as a catalyst for future empirical work that not only further examines the dynamic relationship of these study constructs, but also examines these relationships over time and

in relation to such constructs as self-efficacy and social support. It is important that study findings are translated into practice, to improve the health and mental health as well as the quality of life among individuals with SLE.

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