Spatial-Relational Learning and Memory Deficits Associated with NMDAR Autoantibodies in Systemic Lupus Erythematosus

Brittany L. Bascetta

The Graduate Center, City University of New York

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Spatial-Relational Learning and Memory Deficits Associated with NMDAR Autoantibodies in Systemic Lupus Erythematosus

By

Brittany Bascetta

A dissertation submitted to the Graduate Faculty in Psychology in partial fulfillment of the requirements for the degree of Doctor of Philosophy, The City University of New York

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Abstract

Individuals with Systemic Lupus Erythematosus (SLE) experience inflammation that may target any organ within the body, including the central and peripheral nervous systems. Additionally, these individuals often demonstrate psychological dysfunctions including emotional and cognitive deficits; however, research is inconsistent as to the nature and cause of these dysfunctions. While there are multiple factors that may increase risk for variability in cognitive function, such as population differences, socioeconomic status (SES), mood disorders (depression and anxiety), medication effects, and disease activity, these factors do not reliably predict the severity and extent of cognitive deficits. A growing body of animal research associates autoantibodies (Abs) in SLE with cognitive impairment. A specific Ab that targets N-methyl-D-aspartate receptors (NMDAR) and neuronal DNA, referred to as DNRAb, is associated with hippocampal damage and spatial memory deficits in mice. The goal of this project was to examine the relationship between the DNRAb and cognitive deficits specific to spatial-relational learning and memory within a sample of SLE patients. Two cohorts of healthy controls (HCs) and SLE patients were recruited. Cohort A included 33 HCs and 39 SLE participants (23 DNRAb−, 11 DNRAb+, 5 unknown Ab status). Cohort B included 11 HCs and 21 SLE participants (11 DNRAb−, 10 DNRAb+). All participants completed measures of emotional functioning and neuropsychological measures of visuospatial learning and memory, processing speed, and executive function. Cognitive testing of spatial memory and relational learning was evaluated by two laboratory-developed computerized tasks. Antibody status was determined after participants were recruited in the study. Overall, compared to SLE patients, HCs were significantly more accurate, and thus had better performance on the Spatial Memory and Relational Learning Tasks. The DNRAb+ group was significantly less accurate than HC on both computerized measures. However, the DRNAb− did not differ from either groups on the Spatial Memory Task, and performed similarly to the DRNAb+ group on the Relational Learning Task. This work provides a foundation for future research to analyze how SLE and Ab status could influence cognitive functioning. Future studies can continue to examine subtle cognitive deficits in SLE, consider the possibility of other brain areas to compensate for cognitive deficits in SLE, and provide additional breakthroughs to guide interventions for SLE patients.
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CHAPTER I

Introduction Part I: About Systemic Lupus Erythematosus

Patients with Systemic Lupus Erythematosus (SLE or lupus) experience a progressive, remitting-relapsing autoimmune disorder that is comprised of a heterogeneous constellation of physical and cognitive symptoms. Although there are studies that focus on cognitive profiles of SLE, research has failed to fully distinguish the etiology and manifestation of cognitive dysfunction among SLE patients primarily due to the variable presentation of SLE. In addition, other factors contribute to the broad range of physical and cognitive symptoms, including diagnostic variability, demographic factors, physical health, disease activity, medication, neuropsychiatric (NP) involvement, and autoantibodies. The primary focus of this dissertation is the association between autoantibodies and cognitive dysfunction. Autoantibodies (Abs) are one of the defining features of SLE (Mackay, Uluğ, & Volpe, 2011), however, they are diverse both in type and number. Each Ab may differentially affect any organ; the most commonly affected physiological systems include: muscles, joints, the central & peripheral nervous systems (CNS, PNS), lungs, heart, kidneys, skin, serous membranes, and vascular systems (Lahita, 2011b). Even within the CNS, in particular the brain, the Abs may differentially affect the cells and receptors and different classifications of cell types. Thus, identifying and examining specific Abs may provide better insight regarding cognitive profiles in SLE.

The N-methyl-D-aspartate receptor (NMDAR) Ab, commonly found in SLE, has been hypothesized to play a role in the pathology of the disease, including psychological and cognitive pathologies. Approximately 30-60% of SLE patients have elevated levels of NMDARs Abs (Chang et al., 2015; González-Albo & DeFelipe, 2000). More often than not these Abs are present mostly in the PNS, but research using animal models has found that when the blood brain barrier (BBB) is compromised, NMDAR Abs infiltrate the brain and can cause apoptotic cell death in the hippocampus (among other areas of the brain), which has been linked to
deficits in spatial learning (Chang et al., 2015; Mackay et al., 2011; Kowal et al., 2006).
Understanding the relationship in humans among NMDAR Abs, hippocampal atrophy, and cognition remains limited. In human studies, hippocampal atrophy is linked with cognitive impairment, specifically in spatial and relational learning and memory domains (Appenzeller, Carnevalle, Li, Costallat, & Cendes, 2006). The hippocampus has a high affinity for NMDARs, suggesting that damage to such receptors may impair the formation of spatial and relational memory (Kumaran & Maguire, 2005). Therefore, the aim of this research is to examine whether a relationship exists between concentration levels of anti-NMDAR Abs and impairment in spatial and relational memory in people diagnosed with SLE.

**Diagnosis**

SLE is often difficult to diagnose, as there are 11 possible criteria that are used to classify symptoms of SLE as determined by the American College of Rheumatology (ACR). As described in Table 1, the symptoms include: malar (butterfly) rash, discoid rash, photosensitivity, oral ulcers, arthritis, serosistis (heart or lung inflammation), renal (kidney) disorder, neurological disorder (seizures and/or psychosis), hematologic disorder, immunologic changes, and abnormal titer of antinuclear antibody (ANA). In addition to the diagnostic criteria, patients may experience other symptoms such as pain, fatigue, fevers, and weight loss (Sultan, Begum, & Isenberg, 2003; Tench, McCurdie, White, & D'Cruz, 2000). Oftentimes, these same symptoms are commonly found in other diseases, which must be ruled out before they are given the SLE diagnosis. To be diagnosed with SLE, patients must satisfy at least 4 of the 11 classification criteria either simultaneously or during consecutive observations and test positive for ANA (Petri, 2002; Gill, Quisel, Rocca, & Walters, 2003). Patients can also receive a diagnosis of “probable”, “incomplete”, or “mild” SLE if they do not meet all of the diagnostic
criteria. Therefore, there is great variability in the presentation of SLE and this often true across research studies as well.

The individual diagnostic criteria outlined by the ACR may implicate multiple autoimmune disorders, including SLE. For instance, a positive ANA titer most accurately serves as an indicator for immune system dysfunction. Even though an abnormal ANA titer is critical for diagnosis, as over 90% of SLE patients test positive for ANAs, this antibody is not specific to SLE (Manson & Rahman, 2006). Thus, an abnormal ANA titer may be present in other autoimmune diseases, including rheumatoid arthritis (RA), Sjögren’s Syndrome, scleroderma, and even fibromyalgia (Gill et al., 2003). In order to increase a physician’s certainty regarding an SLE diagnosis in the context of an abnormal ANA titer, the other physical symptoms must be present.

Some researchers argue that since an abnormal ANA titer is not specific to SLE, tests for additional antibodies may increase the specificity and sensitivity of an SLE diagnosis. In addition to ANAs, a test for double-stranded DNA antigen (anti-dsDNA) and anti-Smith nuclear antigen (anti-Sm) antibodies may be particularly helpful to make a more conclusive diagnosis when patients have a positive ANA test but do not fulfill SLE diagnostic criteria (Gill et al., 2003; Kavanaugh, Tomar, Reveille, Solomon, & Homburger, 2000). Both are highly specific to SLE and are uncommon in other autoimmune disorders (Gill et al., 2003). By testing for ANA in addition to anti-dsDNA and anti-Smith antibodies, physicians may increase their confidence in an SLE diagnosis.

Prevalence and Clinical Presentation

Only a small portion of the population may receive an SLE diagnosis within their lifetime. Approximately one per 1000 people, or less than 1% of the population is diagnosed with SLE and estimated prevalence varies depending on whether “probable”, “incomplete”, or “mild”
cases are included (Helmick et al., 2008; Mason & Rahman, 2006; Petri, 2002; Pons-Estel, Alarcón, Scofield, Reinlib, & Cooper, 2010; Uramoto et al., 1999). Despite the low prevalence of SLE, the disease differently affects demographically diverse individuals.

There are multiple factors that contribute to demographic composition of SLE, such as age, gender, race, and ethnicity, and they have implications for symptom manifestation and presentation (Danchenko, Satia, & Anthony, 2006). The frequency of SLE diagnoses varies dramatically by age. For instance, more than half of SLE patients are typically diagnosed between the ages of 20 to 40 years old; however, it is possible to receive the diagnosis at earlier or later ages (Petri, 2002). About 15-20% of patients are diagnosed during childhood (Livingston, Bonner, & Pope, 2011), and a smaller percentage of patients are diagnosed as an adult older than 40 years old (Petri, 2000, 2002). Unfortunately, patients diagnosed with SLE at a younger age often have more severe, active physiological symptoms including NP involvement and increased mortality rates (Costallat & Coimbra, 1994; Livingston et al., 2011). Moreover, children receive more intensive immunosuppressive or steroid treatments than adults and subsequently accumulate more detrimental side effects due to such treatments (Brunner, Gladman, Ibañez, Urowitz, & Silverman, 2008).

Gender is a second demographic factor that appears to influence SLE prevalence. The difference between males and females with SLE is quite striking. SLE occurs approximately 10 times more often in women than in men (Danchenko et al., 2006; Hochberg, 1985; Lim et al., 2014; McCarty et al., 1995; Petri, 2002). This gender difference has led researchers to suspect there is a hormonal contribution (estrogen) to the pathology of SLE (Danchenko et al., 2006; Petri, 2002). Although SLE occurs less frequently in men, they tend to have greater incidents of morbidity, more seizures and increased thrombosis, hypertension, renal insufficiency, and frequency of lupus anti-coagulant (Petri, 2002). Therefore, it appears that gender is associated not only with prevalence but also with disease presentation.
The prevalence of SLE disproportionately affects specific racial and ethnic groups. Within most age groups, SLE is reported more frequently in patients of African, Asian, Hispanic, and Caribbean descent, compared to Caucasians. Across research studies, SLE presents 3-4 times more often in non-Caucasian as compared to individuals from Caucasian or European descent (Cooper et al., 2002; Lau, Yin, & Mok, 2006; McCarty et al., 1995; Michet, McKenna, Elevack, Kaslow, & Kurland, 1985; Seigal & Lee, 1973). However, despite the fact that African American women represent the highest ethnic group with SLE diagnoses, this figure is also dependent upon age. Compared to Caucasian patients, African American and Hispanic SLE patients tend to receive a diagnosis at younger ages, have a more severe course of the disease, and often come from lower socioeconomic status (SES) backgrounds than Caucasian patients (Alarcón et al., 2001; Fernández et al., 2007). Interestingly, while more African American women are diagnosed, on average, 6 years younger than Caucasian women, there is a higher prevalence of Caucasian women in older patient groups (Krishnan & Humbert, 2006; Lahita, 2011a; Pons-Estel et al., 2010). The reason for this is not entirely clear. One possibility for this oddity is that African American SLE patients may have a higher mortality rate than Caucasian patients (Fernández et al., 2007; Krishnan & Humbert, 2006). A second possibility is that that the older SLE patients are a distinct group compared to younger patients with SLE, with a later age of onset and severity/expression of symptoms (Pons-Estel et al., 2010).

As the disease progresses, social factors become important predictors and contributors to disease outcome. Patients with lower SES may have fewer resources available to manage their disease. Furthermore, lower SES may be associated with maladaptive coping strategies, fewer years of formal education, higher levels of poverty, lack of health insurance, and inadequate social support, factors which are all related to SLE symptom severity (Fernández et al., 2007). The contribution of these social factors interacts with ethnicity and may result in more severe courses of SLE particularly for minorities lower in SES.
The Presence of Cognitive Impairment

Cognitive impairment refers to significant deficits in any or all of the following domains: processing speed, attention, language (including fluency), visuospatial abilities, executive functioning (planning, organizing, sequencing), learning and memory, and psychomotor speed. The types of cognitive deficits are variable within SLE patient groups. Furthermore, although cognitive deficits have been identified in SLE patients, researchers have yet to pinpoint the etiology of their cognitive dysfunction (Ad Hoc Committee on Lupus Response Criteria: Cognition Sub-committee, 2007). While the specific causes of cognitive dysfunction in SLE remain unclear, some possible contributors include autoimmune and disease activity and psychosocial, affective/psychological, and biological factors.

In general, cognitive deficits are common among autoimmune disorders. Studies have shown that SLE patients have comparable deficits to patients with other autoimmune disorders, such as rheumatoid arthritis (RA) or multiple sclerosis (MS). Overall, patients with autoimmune disorders are found to have deficits in attention (e.g., sustained attention), processing speed, working memory, and memory (Benedict, Shucard, Zivadinov, & Shucard, 2008; Hanly et al., 2010; Kozora, Thompson, West, & Kotzin, 1996; Kozora et al., 2001; Kozora, Ellison, & West, 2004; Shucard et al., 2004). Moreover, MS patients have more profound deficits than SLE patients on measures of verbal fluency and visuospatial memory (Covey, Shucard, Shucard, Stegen, & Benedict, 2012). Although the mechanisms that contribute to cognitive dysfunction may be different between the autoimmune disorders, for instance atrophy of white matter tracts in MS versus the elevated risks of cerebrovascular disease in SLE (Benedict et al., 2008), the widespread and nonspecific cognitive deficits that these autoimmune disorders have in common may suggest that abnormal immune function may interact with cognitive function.

Researchers have attempted to identify the factors that contribute to the emergence of cognitive dysfunction in SLE. SLE-related disease activity has been proposed to account for
cognitive impairment and is often associated with changes in brain pathology. The disease activity has the potential to be focal (for instance, in the example of a stroke) or more diffuse, both of which can differently impact cognitive deficits. As such, researchers hypothesize that cognitive deficits may arise from antibody activity, NP events (stroke, lesions, or other direct CNS injury), comorbid systemic illnesses (hypertension and thyroid dysfunction), certain medications (beta blockers, antihistamines, antiepileptics, and antidepressants), mood disorders (anxiety, mania, and psychosis), and/or sleep disturbances (fatigue and/or sleep apnea; Hanly, 2011; Kozora, Ellison, & West, 2006; Mackay et al., 2011; Tomietto et al., 2007). These CNS events may produce either acute or persistent physiological and cognitive changes.

Studies that examine the relationship between cognitive deficits and SLE disease activity (type of disease activity and severity) and disease duration have provided mixed results. Disease activity, measured by the Systemic Lupus Erythematosus Disease Activity Index (SLEDAI), is described as the degree of involvement of a single organ or organ system or serologic activity (amount of a certain autoantibody; Bombardier et al., 1992; Kozora et al., 2012). Maneeton, Maneeton, & Louthrenoo, (2010) found that disease activity at the time of SLE diagnosis correlated with scores on a cognitive impairment on a psychological screening battery, however, disease activity at the time of a physical and psychological evaluation and disease duration were not correlated with cognitive impairment. Conversely, another study found that a longer duration or course and more severe disease activity were associated with increased cognitive deficits (Kozora et al., 2012). Older participants, particularly those with longer durations of SLE diagnosis, have been shown to have higher rates of cognitive impairment as compared to similarly aged Healthy Controls (HC; Carlomagno et al., 2000; Glanz et al., 1997). There is even some evidence supporting a relationship between accumulated damage from a history of CNS SLE disease activity and cognitive impairment (Appenzeller et al., 2006; Tomietto et al., 2007). Thus, a history of active CNS disease and
duration of disease, as well as the accumulated disease burden, negatively impacted cognitive abilities; however, current active disease has not been thoroughly investigated. In general, it appears that while disease duration may be negatively associated with cognitive functioning, disease activity may less likely be related to cognitive findings.

In contrast, other studies have not found a relationship between disease activity and cognitive impairment (Ad Hoc Committee on Lupus Response Criteria: Cognition Subcommittee, 2007; Hanly et al., 1992; Monastero et al., 2001). Cognitive deficits have even been observed during periods of inactivity or mild disease activity (Benedict et al., 2008; Glanz et al., 1997; Hanly et al., 1992; Petri et al., 2008). Some groups suggest that cognitive impairment is related to age but unrelated with disease activity (Diamond et al., 2006; McLaurin et al., 2005). One proposed hypothesis to explain why cognitive impairment is not consistently found among SLE patients is that the existing rating scales used to measure disease activity and cognitive impairment are not sensitive enough to detect a correlation between the level of disease activity and cognitive impairment, just as other markers of disease activity such as imaging or serology require further evaluation to assess their sensitivity (Ad Hoc Committee on Lupus Response Criteria: Cognition Subcommittee, 2007). Current measurements of disease activity and cognitive dysfunction have not conclusively determined the cause of cognitive deficits or account for the variability in cognitive deficits.

Since disease activity and duration have not reliably been associated with cognitive impairment in SLE, alternative factors have been proposed to account for variability in SLE cognitive impairment. The factors, which have been explored to determine their contributions to cognitive impairment in SLE, include psychosocial factors and/or environmental factors. Psychosocial factors are often indirectly associated with cognitive impairment, including psychological or affective distress, coping and social support. For instance, patients with SLE may experience a change in emotional functioning. Adjusting to a chronic medical condition or
CNS events may be associated with a change in psychological/affective distress or increased stress. Studies that examine the interaction between affective distress and cognitive dysfunction provided mixed results (Kozora, Ellison, Waxmonsky, Wamboldt, & Patterson, 2005). However, there is at least some support from cross-sectional studies using regression modeling that depression maybe associated with poor cognitive functioning, particularly in SLE patients with NP involvement (see below for discussion of NPSLE; Covey et al., 2012; Kozora et al., 2006; Kozora, Hanly, Lapteva, & Filley, 2008b; Monastero et al., 2001). Some studies have found that SLE patients with concurrent depression, compared to non-depressed SLE patients and HCs, often demonstrate impairments for verbal fluency, visual abilities, attention, visuospatial abilities, and verbal learning and memory (Denburg & Denburg, 1999; Hay et al., 1992; Kozora, Arciniega, Zhang, & West, 2007). Another study found that depression and fatigue were related to deficits in working memory in SLE patients (Covey et al., 2012). Thus, symptoms of depression in SLE have been associated with cognitive deficits.

Environmental factors, such as location, may also play a role in cognitive functioning in SLE. For example, a study that compared cognitive deficits between SLE patients who lived in New York or Colorado discovered that New York patients had more frequent and severe impairment than their Colorado peers (Kozora et al., 2013). With the exception of disease duration and a higher rate of medical complications (in the NY patients), other factors (such as subject selection, test selection, definition of cognitive impairment, age, education, ethnicity, disease severity, level of depression) were controlled, suggesting that it is quite possible environmental or socioeconomic factors (i.e., city living) influenced cognitive impairments.

Despite researchers examining how different factors may be associated with cognitive deficits, some studies do not include patients during current active diseases flares in order to avoid potential confounds associated with an active, inflamed state (Watson, 2014). For instance, Kozora and colleagues (2001) published a study examining inflammation in SLE and
cognitive impairment. One of the exclusionary criteria for this study was current Axis I disorder, including depression, anxiety, or psychosis. Another exclusionary criteria included neurological illness such as seizures or vascular disease, which are common neuropsychiatric symptoms. However, this strict inclusion/exclusion process could also result in variability of cognitive impairment with more severe cognitive impairment under lenient inclusion criteria or subtle or decreased impairment under stricter exclusion criteria.

Altogether, SLE patients experience broad cognitive impairment that may be associated with a variety of biological, affective, and even environmental demands. However, there is a lack of empirical longitudinal studies investigating these potential factors as causes of cognitive impairment in SLE. Despite the probable contributors, there is still a great deal of variability in cognitive function that is not accounted for by these factors.

**Inflammation and Cognitive Impairment**

Inflammation, defined as the body’s response to “foreign” substances, is a commonly occurring symptom in SLE. Inflammation refers to a nonspecific reaction by the body that is initiated by chemical signals in response to potentially harmful activity with the intention of eliminating damaging substances and repairing injury. In autoimmune disorders, however, the body misinterprets innocuous organisms as harmful and targets them. More specifically, in SLE the body generates antibodies that target molecules and cells from the human body.

One of the challenges in studying SLE is that the inflammatory illness produces general or nonspecific systemic responses and may target various areas of the CNS, including the brain, and result in nonspecific cognitive deficits. Inconsistent findings regarding cognitive impairment in SLE may also occur because of the diffuse inflammatory nature of the disease. Although there is a scarcity of research directly comparing cognitive performance and inflammation in SLE, one study comparing SLE patients to RA patients and HCs reported that
inflammatory processes in SLE patients were related to poorer attention and lower learning scores (Kozora et al., 2001). Some studies have limited their participant pool to exclude changes in disease activity or medication dosage for a period of 4-weeks prior to evaluation (Chang et al., 2015; Mackay et al., 2015). Even so, memory impairment continues to be present in neuropsychiatric SLE (NPSLE) even during periods without inflammation (Diamond et al., 2006). Therefore, it is possible that periods of inflammation contribute to greater levels of dysfunction.

**SLE and Medication**

Corticosteroids and nonsteroidal anti-inflammatory medications are among the most commonly prescribed treatments to reduce inflammation during acute disease flare. Corticosteroids are anti-inflammatory steroid hormones that act within cells to produce physiological changes. They work at the intracellular level to prevent the production of cytokines and thus incur immediate anti-inflammatory effects and later result in immunosuppression (Almawi, Beyhum, Rahme, & Rieder, 1996; Chatham & Kimberly, 2001). SLE patients are chronically treated with these medications to relieve their symptoms, and they are commonly thought to influence cognitive function. For instance, they may target the hippocampus and result in transient, reversible changes in memory (Lupien & McEwen, 1997).

However, a majority of studies found that cognitive dysfunction in SLE is not associated with corticosteroid use or dose (Denburg, Carbotte, & Denburg, 1987a; El-Shafey, Abd-El-Geleel, & Soliman, 2012; Emori et al., 2005; Hay et al., 1992; Hanly, 2011; Monastero et al., 2001), while other studies point to both positive and negative effects. A small study found that a low dose of corticosteroid medication might actually improve SLE symptoms and cognition (Denburg, Carbotte, & Denburg, 1994). Conversely, another study found that corticosteroid use may be associated with deficits to verbal memory and retrieval of information (Fisk, Eastwood,
Sherwood, & Hanly, 1993). Other studies have identified that a specific anti-inflammatory corticosteroid, Prednisone, has been associated with cognitive deficits (Kozora et al., 2006; McLaurin, Holliday, Williams, & Brey, 2005). The cognitive effects related to Prednisone were found to be most frequent in middle-aged adults as opposed to younger and older adults, and the cognitive effects seemed to target declarative memory associated with the hippocampus (Keenan et al., 1996). In sum, while there is no strong evidence to suggest SLE medications contribute to cognitive deficits, when impairments are observed they often are associated with memory.

**Neuropsychiatric Systemic Lupus Erythematosus (NPSLE)**

A subgroup of SLE patients will develop symptoms that affect the nervous system. It is estimated that NPSLE occurs in 37-95% of SLE cases (Hanly, 2011). This considerably large range represents the variability in the prevalence and expression of NPSLE and its symptoms, which may depend on the populations recruited for NPSLE studies, sample size, and tools/criteria used to measure dysfunction. As outlined in Table 2, NPSLE is characterized by 19 distinct possible syndromes, which occur in either the PNS or CNS. The syndromes can be focal (e.g., stroke) or more generalized and diffuse (psychosis, mood disorders, cognitive dysfunction). Each of these 19 manifestations may frequently occur in the general population as well; therefore, they are not specific to SLE. Similar to the diagnosis of SLE, alternative causes for NP syndromes must be ruled out before a diagnosis of NPSLE is given to patients.

The diagnosis of NPSLE varies as a function of the classification criteria and methodology. To receive a diagnosis of NPSLE, one must meet 3 or more of the ACR diagnostic criteria for SLE plus an additional case definition of NPSLE (American College of Rheumatology Ad Hoc Committee on Neuropsychiatric Lupus Nomenclature, 1999). However, not all research studies implement this approach, as some have considered even a single focal
symptom from the case definition as an indicator of NPSLE (Carlomagno et al., 2000). Therefore, despite formal criteria outlined by the ACR, studies may vary in the diagnosis of NPSLE.

There is no single laboratory test to establish a diagnosis of NPSLE, and, as such, a multi-modal approach has been used to evaluate the different NP symptoms. Thus, the process to establish a diagnosis of NPSLE may include imaging, including magnetic resonance imaging (MRI) or computed tomography (CT) scans, to assess structural abnormalities, or a positron emission tomography (PET) scan to assess for cerebral blood flow and metabolism), examining cerebral spinal fluid (CSF) to exclude infection, and neuropsychological testing to address specific concerns about cognitive ability (Bruns & Meyer, 2006; Hanly, 2011). However, the selection and effectiveness of these tools varies as a function of the symptoms (Hermosillo-Romo & Brey, 2002). For instance, an MRI may appear normal for depression or other diffuse conditions. Further, studies analyzing blood serum of Abs has provided mixed results; some indicate that the presence of Abs in serum is not related to NP involvement (Fragoso-Loyo et al., 2008), yet others indicate there is a relationship between serum and NPSLE (Kowal et al., 2006; Mackay et al., 2015; Watson, 2014). In fact, some studies have used elevated NMDAR Abs in blood serum to diagnose NPSLE (Omdal et al., 2005). However, other studies did not find strong associations between NMDAR Abs in blood serum and NPSLE (Harrison, Ravdin, & Lockshin, 2006; Hanly Robichaud, & Fisk, 2006; Lapteva et al., 2006). Despite inconclusive research regarding blood serum and diagnosis of NPSLE, the presence of NMDAR Abs in CSF does appear to be associated with NPSLE diagnosis (Arinuma, Yanagida, & Hirohata, 2008).

Cognitive dysfunction is one of the possible diffuse CNS syndromes that are frequently found in NPSLE. In fact, of the 19 different NPSLE manifestations, cognitive dysfunction was the most commonly occurring syndrome, in up to 80% of patients (Bruns & Meyer, 2006), and cognitive dysfunction occurs more frequently and with greater severity for NPSLE patients than
compared to SLE patients without overt NP involvement (non-NPSLE; Loukkola et al., 2003; Monastero, 2001; Nowicka-Sauer, Czusynksa, Smolenska, & Sierbert, 2011). Although, there is a higher frequency of cognitive deficits in NPSLE as compared to non-NPSLE, this difference may not be statistically significant (Vogel et al., 2011). Even with a diagnosis of NPSLE, it is still unclear how and which cognitive deficits will manifest (Emori et al., 2005) and such deficits could be related to medication, psychiatric status, an acute state of confusion, vascular abnormalities, or a comorbid illness (Ainiala et al., 2001). Mood disorders, for example, are quite prevalent in NPSLE. NPSLE patients report greater depressive symptoms and more fatigue and pain than their healthy peers (Kozora et al. 2006). Each of these symptoms may contribute to the cognitive dysfunction experienced by NPSLE patients.

**Autoantibodies in SLE**

In order to further explore differences in cognitive impairment that are seen among SLE patients, research has turned its focus to Abs. Since Abs are one of the defining features of SLE, it has been posited that investigating Abs may provide insight into the source of cognitive impairments in SLE patients (Mackay et al., 2011). It is thought that over 100 Abs (or proteins that attack the antibody proteins within the immune system) may be responsible for the inflammation and damage associated within blood cells, endothelial cells, the nervous system, and other systems in patients with SLE (Sherer, Gorstein, Fritzler, & Shoenfeld, 2004). However, it remains unclear how each Ab or set of Abs affect the central and peripheral nervous system organs, including the brain. Specific to the brain, the Abs may differentially affect areas, matter, and neuron and cell types. Thus, researchers suspect that disruption to these different areas may independently influence cognitive abilities.

Since multiple Abs are related to SLE inflammation, it is difficult to determine how each of these independently affects cognitive function. Researchers have identified some of the most
common Abs and examined their associations with cognitive dysfunction, including Antiphospholipid antibodies (aPL), double stranded anti-DNA (Anti-dsDNA), anti-Sm, and antiribosomal P antibodies (Lahita, 2011b; Tomietto et al., 2007). One of the goals of this research is to begin to identify the relationship between specific Abs and cognitive dysfunction by focusing on the relationship between NMDARs that target specific neuroanatomical locations (with our specific focus on the hippocampus). We suspect that damage to this neuroanatomical location may impair the cognitive functions associated with that area. A broader discussion with respect to NPSLE, Abs, and cognitive dysfunction can be found in Part V.
Part II: Neuropsychology and Neuroscience of SLE

SLE patients with and without NP involvement often show cognitive dysfunction in the domains of attention, language, visuospatial ability, processing speed, learning and memory for verbal and nonverbal material, and/or motor dexterity (Table 4; Benedict et al., 2008; Kozora et al., 2013; Kozora, 2008; Vogel et al., 2011; Watson, Storbeck, Mattis, & Mackay, 2012). However, such cognitive deficits are often classified as mild rather than severe (Nowicka-Sauer et al., 2011). Although there are some studies that focus on cognitive profiles of SLE, research has failed to clarify the cause and nature of cognitive dysfunction, and these studies are inconsistent or provide contradictory findings. The reason for this is unclear, however, factors related to disease activity, such as inflammation, may provide greater insight regarding some of these cognitive findings. Thus, foundational questions, such as the prevalence of specific cognitive symptoms, remain unanswered (Kozora et al., 2008a; Monastero et al., 2001; Vogel et al., 2011). Below, the course of cognitive impairment will be detailed and recent research findings regarding dysfunction within specific cognitive domains will be summarized.

Course of Cognitive Deficits

Cognitive deficits often emerge soon after SLE diagnosis and are present and stable throughout the course of the disease. Newly-diagnosed SLE patients often score significantly lower than HCs on measures of cognitive efficiency (a composite measure of accuracy, consistency, omissions, and reaction time, RT) in the domains of vigilance/sustained attention, visuospatial working memory, and simple RT/processing speed (Kozora et al., 2011; Petri et al., 2008). Over a short (12-month period) duration, it may appear that these cognitive deficits are transient and, to a certain degree, remit (Hanly, Fisk, Sherwood, & Eastwood, 1994). However, longitudinal studies that review cognitive functioning over a 12- to 60-month period of time have reported that cognitive functioning in SLE is relatively stable and not cumulative with disease...
(Hanly et al., 1997; Waterloo et al., 2002). A 5-year longitudinal study by Waterloo and colleagues (2002) found that SLE patients had mild cognitive impairment in attention, psychomotor function/speed, executive function, and intellectual functions, which remained stable over a 5-year period despite disease progression. In fact, cognitive impairment is thought to be persistent in SLE patients and may increase with age, but may not be related to disease activity (Diamond et al., 2006; McLaurin et al., 2005). Furthermore, the severity and pattern of cognitive impairment remains stable in patients with non-NPSLE and NPSLE (Carlomagno et al., 2000). Therefore, both SLE groups with and without NP involvement have demonstrated a relatively steady course of impairment.

Specific Domains of Cognitive Dysfunction: Subjective Concerns

**Subjective Concerns.** Subjective concerns about cognitive dysfunction are usually common among non-NPSLE and NPSLE patients. The subjective concerns are often nonspecific in nature, and may be subtle, thus, not often detected in a clinical setting (Ad Hoc Committee on Lupus Response Criteria: Cognition Subcommittee, 2007). There is a relationship between the perception of cognitive dysfunction and demonstration of cognitive impairment on objective assessments (Kozora et al., 2006). Kozora and colleagues (2006) found that when compared to non-NPSLE patients, NPSLE patients had both higher levels of subjective concerns as well as higher objective impairment identified using cognitive assessments. Additionally, subjective concerns regarding poor concentration have been associated with marginal performance on the Mini-Mental State Examination (MMSE; Carlomagno et al., 2000). Thus, subjective concerns or perceptions of cognitive impairment may correlate with objectively measured performance.

Although some research has emerged indicating there is a relationship between subjective concerns and cognitive impairment, the availability of such research is limited and
has some contradictory findings. For instance, in another study conducted by Kozora and colleagues, researchers did not find an association between perceived impairment and objectively measured impairment (2007). Similarly, Vogel and colleagues found that while some non-NPSLE patients have low levels of subjective cognitive complaints, the complaints were not correlated with cognitive impairment (2011). Interestingly, while these patients did demonstrate cognitive deficits in attention, planning, and mental speed, the deficits were associated with affective status and not perceived deficits (Vogel et al., 2011). Thus, while it does appear that SLE and NPSLE are associated with mild cognitive impairments, subjective reports do not always accurately predict the degree of a patient's cognitive dysfunction.

**Attention.** Attention is a broad construct that includes concepts such as simple or basic attention and complex attention that includes both auditory and visual modalities of sustained attention, concentration, and vigilance. Attention is one of the most studied cognitive domains in SLE and has the most empirical support for impairment in non-NPSLE and NPSLE patients throughout the course of the disease (Ad Hoc Committee on Lupus Response Criteria: Cognition Subcommittee, 2007). When compared to HCs, SLE patients have been found to have lower cognitive efficiency (a construct that measures speed and accuracy) on three tasks of sustained attention/vigilance (Petri et al., 2008). Overall, it does appear that attention is often deficient within SLE and NPSLE populations and that decreased attention may be more severe within the NPSLE population (Emori et al., 2005; Kozora, 2008; Monastero et al., 2001; Sabbadini, 1999; Watson et al., 2012).

**Working Memory.** Working memory is a form of complex attention that consists of the ability to temporarily encode, maintain, and manipulate information over a short time span. Working memory may be measured by tests such as the n-back task, which may range in difficulty from simple attention (mentally holding and updating 1 item at a time) to more complex working memory (mentally holding and updating 4 items). Shucard and colleagues
demonstrated that with simpler attention/working memory tasks (0-back), SLE patients performed similar to HCs, but, as the task became more challenging (2-back), performance for the SLE group became less accurate (2011). Interestingly, when controlling for processing speed, non-NPSLE and NPSLE patients continued to demonstrate comparable working memory deficits. In another study by Shucard and colleagues, the researchers observed that individuals with SLE adopt compensatory strategies (e.g., chunking) sooner than HCs suggesting inefficiencies in working memory (2004).

Although the above effects examined verbal working memory, visuospatial working memory has yielded mixed results. Similar deficits have been found on a visuospatial working memory Automated Neuropsychological Assessment Metrics (ANAM) task where individuals were asked to select a matrix to match to a previously displayed matrix (Petri et al., 2008). Neuropsychologically, visuospatial working memory has been assessed with a block-tapping test, which requires only gross motor skills and reduces the demand for fine motor skills often associated with visuospatial tasks. Using this modality, researchers have not found visuospatial working memory deficits in NPSLE or non-NSPLE patients (Denburg et al., 1987a; Monastero et al., 2001). Overall, auditory, but not visuospatial, working memory deficits often present in both SLE and NPSLE populations, however, more research is needed to characterize deficits in this aspect of complex attention.

**Processing Speed and Motor Speed.** Processing speed is a basic test of cognitive efficiency assessed through psychomotor speed. Often, processing speed is related to multiple neural networks, including perception, attention/working memory, decision-making, and motor performance. Therefore, poor processing speed is a nonspecific finding that is often associated with multiple neural networks (frontal, cerebellar) and/or white matter integrity (Eckert, 2011). Depending on the methodology used to measure speed, actual motor deficits can sometimes confound performance on psychomotor tasks. The interaction between processing speed and
motor speed is particularly relevant for SLE and NPSLE populations when compared to HCs, as deficits have been found on a variety of motor and processing speed tasks (Benedict et al., 2008; Denburg et al., 1987a; Ferstl et al., 1992; Glanz et al., 1997; 2005; Kozora, 2008; Loukkola et al., 2003; Shucard et al., 2011; Vogel et al., 2011). Fine motor speed is not consistently impaired, whereas research more frequently supports deficits in processing or psychomotor speed. In a composite of motor functioning measured by finger tapping and grip strength, SLE patients appear impaired relative to controls (Glanz et al., 1997). When assessing finger tapping alone, one study by Kozora and colleagues found SLE patients were slower than HCs (Kozora et al., 2004), however, subsequent studies have not supported this conclusion (Glanz et al., 2005; Kozora et al., 2004). Yet, SLE patients have evidenced poorer psychomotor speed. In fact, the speeded numerical sequencing task, Trailmaking (TMT) A, has frequently been impaired across many of these studies (Denburg et al., 1987a; Emori et al., 2005; Glanz et al., 2005; Kozora et al., 2004). The TMT A task has been described as a measure of visual search (attention) and motor speed (Crow, 1998). Additionally, studies that included a comprehensive neuropsychological battery incorporating both oral and/or written psychomotor speed have found that NPSLE patients consistently performed significantly worse than non-NPSLE patients and/or HCs (Glanz et al., 1997, 2005; Loukkola et al., 2003; Nowicka-Sauer et al., 2011; Vogel et al., 2011). Only one study failed to find differences between SLE patients and HCs when using a simple processing speed task (El-Shafey et al., 2012). Overall, the research appears to most consistently support the notion that processing speed is impaired in SLE patients, even when controlling for motor speed deficits.

**Language Abilities.** The language domain consists of knowledge for words (vocabulary), fund of knowledge, naming/identifying objects (from pictures or description), and higher-order verbal fluency. In general, language abilities (naming and vocabulary) for SLE patients usually fall within a satisfactory range (Glanz et al., 1997; Kozora, 2008; Kozora et al.,
However, both non-NPSLE and NPSLE patients demonstrate both phonemic and semantic fluency deficits when compared to HCs (Kozora et al., 2004; Denburg et al., 1987a). Therefore, individuals with SLE may experience higher-order language-processing deficits, while general language abilities often remain unimpaired.

**Visuospatial Abilities.** Visuospatial abilities involve visuoperception (identifying figures or matching line orientations), visuoconstruction (ability to copy a figure, using executive functioning to plan, or construct complex designs using blocks), and visuospatial fluency (fluently create multiple visual stimuli and also relies on executive functions). Basic visuoperception, measured by a line orientation task, was not impaired in NPSLE and non-NPSLE patients when compared to MS patients and HCs (Covey, Shucard, Shucard, Stegen, & Benedict, 2012).

Although basic visuospatial abilities appear to be intact, findings regarding higher-level visuospatial abilities are inconsistent. Deficits have been found amongst patients with SLE, and specifically with NPSLE, in a computerized higher-level visual perception task that included a working memory demand (Petri, 2008), as well as on tasks of visuoconstructional ability and visuospatial fluency (Denburg et al., 1987a; Kozora, 2008; Kozora et al., 1996, 2004; Monastero et al., 2001; Nowicka-Sauer et al., 2011; Roebuck-Spencer et al., 2006). However, not all studies consistently support these deficits. Kozora and colleagues (1996) found that even after adjusting for IQ, SLE patients demonstrated significant deficits in the fluency domain (comprised of both the Controlled Oral Word Association test for verbal fluency and the Ruff Figural Fluency Test for design fluency). Yet, in another study in 2004, Kozora and colleagues did not observe similar group differences on this same figural fluency task. In addition, while a few studies found that NPSLE patients often perform worse than non-NPSLE peer groups on visuoconstruction
ability during a complex figure copying task (Monastero et al., 2001; Vogel et al., 2011) and a block design task (Denburg et al., 1987a; Kozora et al., 2004; Nowicka-Sauer et al., 2011); other studies have failed to find differences between non-NPSLE and NPSLE patients on these same tasks (Denburg et al., 1987a; Glanz et al., 1997, 2005; Kozora et al., 2008; Sabbadini et al., 1999). Taken together, simple visual perception is not impaired in patients with SLE; however, greater variability of deficits are seen in more complex visuospatial tasks that rely on additional cognitive abilities. Although it is unclear why there is such variability among these functions, speculatively, deficits in other areas such as attention, processing speed, and motor functioning could contribute to this variability.

**Executive Functioning.** Executive functions include the ability to plan/organize, reason or problem-solve, and switch between tasks or mind-sets (cognitive flexibility). SLE patients are impaired on a variety, but not all, executive functioning tasks. Most SLE patients do not have difficulty with planning/organization and problem-solving tasks (Emori et al., 2005; Loukkola et al., 2003). Research regarding reasoning deficits has been inconsistent. While recent studies have found no differences on a verbal reasoning task among non-NPSLE patients, NPSLE patients, and HCs (Glanz et al., 2005; Loukkola et al., 2003), an earlier study found a small subset of both non-NPSLE and NPSLE patients may have difficulty with verbal reasoning (Denburg et al., 1987a).

SLE patients do consistently show executive function deficits for cognitive flexibility. When assessed by an alphanumeric sequencing task (Trailmaking B), impairment is often found in set-switching (El-Shafey et al., 2012; Emori et al., 2005; Loukkola et al., 2003; Nowicka-Sauer et al., 2011; Vogel et al., 2011). Interestingly, this finding remains consistent regardless of whether or not SLE patients are deficient on the simpler psychomotor/numeric sequencing measure (Trailmaking A; El-Shafey et al., 2012; Emori et al., 2005, Nowicka-Sauer et al., 2011).
Set-switching abilities may also differentiate NPSLE from non-NPSLE peers as NPSLE patients demonstrate greater impairments (Loukkola et al., 2003; Nowicka-Sauer et al., 2011). This finding is also supported on other tasks of cognitive inhibition, such as the Stroop task, which differentiated NPSLE from non-NPSLE and HCs (Loukkola et al., 2003). However, not all studies have found deficits in cognitive flexibility (Denburg et al., 1987). In sum, cognitive flexibility appears to be the most impaired executive function, whereas other functions, such as planning/organization and reasoning, appear mostly intact.

**Learning and Memory.** In neuropsychological measures, learning and memory are always grouped together. Individuals are assessed on their initial acquisition of information, learning slope, recall after a brief delay (immediate recall), their retention upon a delay (short or long delayed recall), and recognition of material. The information that is presented to individuals can also be divided between verbal (linguistic) and nonverbal (visual, spatial-relational) methods. Memory deficits are among the most frequent complaints and objectively observed deficits in SLE (Ad Hoc Committee on Lupus Response Criteria: Cognition Subcommittee, et al. 2007). Many studies have demonstrated below average learning in both verbal and nonverbal (visual) information when SLE patients are compared to HCs and even to patients with RA (Ferstl et al., 1992; Glanz et al., 1997; Kozora et al., 1996, 2001, 2004, 2008a, 2011; Monastero et al., 2001), and corresponding deficits in both immediate and delayed memory for material (Denburg et al., 1987a, Kozora et al., 2011). However, one study failed to find delayed memory impairment on an auditory list-learning task when comparing SLE and HCs (Kozora et al., 2004). It is common for NPSLE patients to suffer greater delayed memory deficits compared to non-NPSLE patients (Monastero, et al., 2001; Nowicka-Sauer et al., 2011); however, this finding is not consistently supported (Denburg et al., 1987a; Loukkola et al., 2003; Sabbadini et al., 1999).
As mentioned above, memory consists of learning, retention, and retrieval. Some studies suggest that memory differences may stem from encoding failures. For instance, SLE patients, who were assessed for memory deficits pre- and post-diagnosis, did not decline in delayed memory function, but, rather, researchers suggested such deficits might be due to failures of attention during learning or testing (Skeel, Johnstone, Yangco Jr., Walker, & Komatireddy, 2000). Thus, care must be taken when assessing memory and interpreting the research studies regarding memory deficits in SLE.

**Verbal Memory.** Although many studies show that SLE patients have impairments on multiple aspects of verbal memory, there are also many studies that do not show these deficits. On both individual and paired rote list-learning tasks, SLE patients have lower scores compared to controls on the learning trials, immediate list recall, delayed list recall, and interference trials (Emori et al., 2005; Kozora et al., 2011; Paran et al., 2009; Roebuck-Spencer et al., 2006; Shapira-Lichter et al., 2013). Although there are differences in verbal memory when comparing non-NPSLE and NPSLE patients, performance across verbal recognition measures has been contradictory and has been attributed to “poor strategy” or problems in encoding and storage (learning) rather than in retrieval (Emori et al., 2005; Fisk et al., 1993). Therefore, it appears that poor acquisition may account for difficulties across aspects of rote verbal learning and memory.

Patients with SLE are also believed to have weaknesses for verbal memory of contextual information, although the evidence is not overwhelming. In one study, NPSLE patients differed significantly than non-NPSLE patients and HCs on their immediate recall of passages, but not on delayed recall (Denburg et al., 1987a). However, another study found that on both immediate and delayed recall of stories, NPSLE patients had clear deficits in immediate and delayed recall, whereas non-NPSLE patients were not significantly different from NPSLE patients or HCs (Loukkola et al. 2003). In sum, deficits are present across initial acquisition and
recall of both rote and contextual verbal information in both non-NPSLE and NPSLE patient groups. Although these patient groups appear to have similar deficits in verbal learning memory, the greater deficits observed in NPSLE groups suggest that they may experience more difficulty with inattention during learning, which likely impairs storage/retrieval of information.

**Visuospatal Memory.** Although basic visuospatial abilities are intact, there has been some variability on more complex measures of visuospatial functioning (such as those that are timed like block design tasks, those requiring higher-ordered executive abilities such as fluency, or planning and organization on the RCFT copy). Visuospatial memory measures one’s ability to recall visuospatial material that was previously encoded. Few tasks have been designed to measure visuospatial memory. Oftentimes, visual memory is assessed following an individual’s ability to draw a figure, series of figures, or an array and then reproduce the figure, figures, or array after delays. Impairment has frequently been observed in the visuospatial (sometimes called nonverbal) memory domain of cognitive functioning in SLE patients. Although there does not appear to be a significant difference in SLE patients’ ability to copy a complex figure (Denburg et al., 1987; Emori et al., 2005; Glanz et al., 1997, 2005; Monastero et al., 2001), deficits are prevalent for visuospatial free recall after a delay (Coín-Mejías et al., 2008; Ferstl et al., 1992; Monastero et al., 2001; Nowicka-Sauer et al., 2011; Vogel et al., 2011) and recognition (Kozora et al., 2011). Additionally, NPSLE patients often make more errors for the delayed recall compared to non-NPSLE patients (Nowicka-Sauer et al., 2011). Thus, although SLE patients are typically able to copy a complex figure without any error, their delayed recall of this type of figure is poor.

Researchers have used the complex figure approach to assess visuospatial memory in SLE patients; however, this particular task does not directly assess an individual’s ability to recall spatial and relational aspects of items. Computerized visual memory tasks have deficits in
patients’ ability to match visuospatial items to a display after a delay (Brey et al., 2002; Petri et al., 2008). Using a different laboratory-designed computerized task, Chang and colleagues (2015) presented a series of line-drawn objects in an array to SLE patients and to HCs. After each array, the participants were asked either a question about the spatial arrangement of the objects within the array or a nonspatial question (i.e., “was an item present?” or “were there 4 images on the screen?”). The researchers determined that SLE patients had difficulty remembering the spatial arrangements for items, but not for recalling the presence of items. Further, NPSLE patients with (patients with elevated NMDAR Abs) exhibited worse performance on this task, whereas NPSLE patients without elevated NMDAR Abs performed more similarly to HCs. Therefore, preliminary research appears to support deficits in aspects of visuospatial memory, particularly in patient groups with NMDAR Abs.

Overall, short- and long-term visuospatial memory tasks consistently demonstrate nonverbal memory deficits in NPSLE and non-NPSLE patients. Despite these findings, research has been limited when characterizing the nature of visuospatial memory in SLE patients.

**Brief Review of Cognitive Deficits**

In sum, it appears that there is variability in the types of deficits faced by SLE patients, yet the most consistent areas of intact functioning include basic fund of knowledge, vocabulary, and visual perception. In contrast, the research regarding other areas of cognitive functioning have found deficits in at least some aspects of attention, working memory, processing speed, and learning and memory. Furthermore, the deficits in learning and memory are present in both the verbal and visuospatial domains. While verbal learning and memory have been reviewed comprehensively, this is not the case for nonverbal learning and memory. To our knowledge, different aspects of nonverbal memory, such as learning, recall, and recognition of spatial memory and spatial-relational memory have not been methodically investigated.
Part III: Limitations and Considerations in the Assessment of Cognitive Dysfunction in SLE

Although it is clear that SLE patients exhibit deficits within cognitive domains, the variability and limited research in these domains have made it difficult to identify the exact nature of cognitive dysfunction. Studies have varied in the type of measures they use (neuropsychological batteries, computerized batteries, and self-report rating scales), as well as their definitions of cognitive impairment. The variability in research procedures and measures, outlined below, has resulted in several limitations that must be considered when interpreting the results of these studies.

Assessment of Neuropsychological Functions

Researchers have implemented a variety of instruments to characterize cognitive functions in SLE. While some studies incorporate screening measures, others include a comprehensive neuropsychological battery, and others use computerized cognitive measures. Each of these approaches has both benefits and disadvantages. However, broad comparisons across studies must be considered cautiously.

Screening measures, such as the MMSE (Folstein, Folsetin, & McHugh, 1975; Leritz et al., 2000) or the Montreal Cognitive Assessment (MOCA; El-Shafey et al., 2012; Nasreddine et al., 2005), are brief tools used to identify gross cognitive decline. Use of a screening measure greatly reduces time-consuming process of a comprehensive neuropsychological evaluation. The drawback of using these methods is that they do not detect subtle cognitive deficits, and as such, often under-emphasize cognitive deficits in SLE patients. However, despite this limitation, there have been significant findings regarding cognitive deficits in SLE groups. For instance, on the MMSE, non-NPSLE and NPSLE individuals were more likely than HCs to show deficits in the areas of attention/calculation, auditory comprehension, visuospatial abilities, and executive
function (Maneeton et al., 2010). The findings were more pronounced in the NPSLE group as compared to the non-NPSLE group on the MMSE. As for the MOCA, SLE patients tend to reveal deficits in executive function and visuospatial ability, when compared to controls; however, other covariates, such as education, may have a greater impact on performance than the disease itself (El-Shafey, Abd-El-Geleel, Soliman, 2012). Nonetheless, although the MMSE and MOCA have been validated as brief screening measures to assess cognitive impairment in dementias but not in autoimmune disorders, and they have limited validity when examining individual subscales as predictors of specific cognitive impairment, they are not intended to discriminate between more subtle or nuanced cognitive impairment (Moafmashadi & Koski, 2012). Therefore, these screening tools have limited utility in characterizing the cognitive deficits that SLE patients may experience.

The American College of Rheumatology (ACR) Ad Hoc committee has developed a standard battery, which was designed to diagnose and characterize NPSLE and the cognitive dysfunction apparent in both NPSLE and non-NPSLE patients (Table 3). The one-hour ACR battery is both valid and reliable in identifying cognitive deficits in a research setting (Kozora et al., 2004). Despite the established validity and reliability of the ACR battery to assess cognitive dysfunction, it has not met with widespread acceptance (Kozora et al., 2008b). The ACR battery is limited to a general overview of cognitive domains, which may not detect subtle cognitive impairment (Kozora et al., 2004; Roebuck-Spencer et al., 2006). Another limitation of the ACR battery is that, like many neuropsychological measures, it sometimes may be insensitive to cultural biases for mixed ethnicities (Holliday et al., 2003), which is particularly relevant to the demographic background of SLE patients.

Some researchers have attempted to use computerized assessment measures to provide more nuanced information than cognitive screening measures and the ACR battery. Of note, the computerized Automated Neuropsychological Assessment Metrics (ANAM) system
has frequently been used as a screening tool, due to ease of administration and accuracy of data collection (Ad Hoc Committee on Lupus Response Criteria: Cognition Subcommittee, 2007). It has higher validity and reliability than brief screening measures like the MMSE, and high specificity and sensitivity in recognizing even subtle cognitive impairments in SLE patients (Ad Hoc Committee on Lupus Response Criteria: Cognition Subcommittee, 2007; Brey et al., 2002; Roebuck-Spencer et al., 2006). Furthermore, subtests on the ANAM are strongly correlated with aspects of attention and cognitive switching (executive function), but not with more complex memory, language, or visuospatial functions (Kozora et al., 2008b). In a review of multiple studies that used the ANAM with SLE patients, impairment is correlated with neuropsychological tests in the domains of attention, processing speed, motor speed, and executive functions, as well as learning and short-term memory; plus, it was considered to be a good predictor of cognitive impairment (Kane, Roebuck-Spencer, Short, Kabat, & Wilken, 2007; Roebuck-Spencer et al., 2006). Overall, the ANAM findings have been considered comparable to neuropsychological findings in SLE patients.

It has been suggested that the use of the ANAM can also reduce the impact of other factors than can influence performance on cognition measures. The ANAM is more robust than certain neuropsychological measures against non-disease-related factors (fatigue, mild depression, or level of education, English language proficiency, and ethnic/cultural differences). In fact, when the ANAM was used to assess and compare cognitive deficits in Hispanic patients with SLE to non-Hispanic SLE patients, the ANAM provided greater reliability than compared to more traditional neuropsychological paper-and-pencil tasks (Holliday et al., 2003). However, the ANAM is susceptible to practice effects (Holliday et al., 2003, Kozora et al., 2008b). Moreover, subtle deficits may go undetected because a trained clinician typically does not administer the ANAM (Ad Hoc Committee on Lupus Response Criteria: Cognition Subcommittee et al., 2007).
For research purposes, the ANAM is a cost-effective, an adequate tool for assessing cognitive deficits, and more sensitive to cultural biases in the SLE population.

**Defining Cognitive Impairment in SLE**

SLE patients vary widely with respect to cognitive impairment, and part of the variability is due to sampling and comparison groups, variability in test measurements, and definitions of SLE. The variability in psychobiosocial factors observed in SLE populations and variability assessment measures have been addressed above. Thus, this section will be devoted to the variability in defining SLE. Across multiple SLE studies, there are inconsistencies in the methods used to define cognitive impairment, including defining decline by domain, the use of “cut-off” points, and comparing cognitive function to their HC peers.

Formally, there is no consistent procedure when defining cognitive impairment across the SLE research studies. An Ad Hoc Committee attempted to set standards for defining cognitive impairment. They examined neurocognitive impairment by compiling a series of SLE clinical trials, and they defined “cognitive decline” as 1.5-1.9 standard deviations (SD) below the mean, while “cognitive impairment” refers to 2 SD below the mean (Ad Hoc Committee on Lupus Response Criteria: Cognition Subcommittee et al., 2007). The Ad Hoc committee also differentiated between focal (one or more measures in a single domain) and multifocal (two or more domains) impairments. However, many studies have not followed the committee’s guidelines when determining whether SLE patients have cognitive deficits.

Research studies vary in their cut-off points for defining cognitive impairment. On the liberal end, researchers select a cut-off score of 1 SD below the mean in defining impairment (Kozora et al., 2012; Julian et al., 2012; Tomietto et al., 2007). The liberal cutoff results in a greater presentation of cognitive impairment, and to reconcile the liberal inclusions, patients must score below 1 SD on multiple tests or indices to be defined as cognitively impaired. Other
groups use a more conservative approach, only labeling patients as cognitively impaired if they are 1.5 or even 2 SD below the mean (Katz et al., 2012; Nowicka-Sauer et al., 2011). The differences in cut-off points may account for the inconsistencies between studies when reporting frequency and severity of cognitive deficits.

Instead of using cut-off scores to define cognitive impairment, alternative approaches have been devised, such as comparing raw scores from SLE patients to HCs (El-Shafey et al., 2012; Kozora, 2008) or by comparing the current scores to the patients’ estimated premorbid functioning (Nowicka-Sauer et al., 2011). One approach even used a regression model to determine whether observed scores were different than expected scores (derived from demographically corrected age, education, years, and intellectual ability based on the Vocabulary subtest from the WAIS; Vogel et al., 2011). Therefore, it is clear that the various definitions of cognitive impairment could contribute to inconsistent findings across studies.
Part IV: Models of Learning and Memory and the Hippocampus

Learning and memory are part of a multi-step process. The most commonly accepted theory of the process of learning and memory has four stages: (a) information acquisition and encoding, (b) consolidation of information, (c) storage, and (d) retrieval. However, the specific models of memory, as well as the role of the hippocampus within these models, continue to be areas of debate.

Models of Learning and Memory

There are multiple theories and models used to describe different aspects of long-term memory. For instance, memory may be considered explicit (or direct) or implicit (or indirect), relating to the conscious ability to recall information, with explicit memory pertaining to conscious learning/retrieval and implicit pertaining to unconscious learning/retrieval (Schacter, 1987). Another model considers knowledge for facts and events as declarative memory, whereas learning of skills or habits are considered procedural memory (Cohen & Squire, 1980). Declarative memory has been further divided into episodic memory (self-referential information) or semantic memory (fact, non-self-referential information). Lastly, relational learning is described as the ability to learn the associations between 2 or more seemingly unrelated objects, tasks, situations, etc. Although these models parse different characteristics related to memory, they all overlap to a certain degree. For instance, one may have conscious awareness of facts and events (explicit, declarative), thus these constructs may be supported by the same system.

Learning and Memory within the Hippocampus

The theories surrounding the main functions of the hippocampus are many and quite varied. The hippocampus is thought to be involved in multiple aspects of the learning and
memory process, from encoding through retrieval. At the simplest level, the hippocampus is thought to be critical for the learning, storing, and recalling of novel stimuli or environments (Kumaran & Maguire, 2007; Lisman & Grace, 2005). Beyond encoding, the hippocampus is also involved in (nonverbal) object and spatial recognition (Broadbent, Squire & Clark, 2004). More notably, the hippocampus plays a role in the consolidation of information into long-term memory, but the storage occurs elsewhere in the brain. Specifically, the hippocampus is associated with long-term potentiation (LTP), a process by which information is consolidated and later transferred to permanent “storage” in other cortical areas (Squire, 1992). However, LTP will be discussed in more detail at the cellular level in Part V. It is quite clear that the hippocampus remains involved in multiple stages of the learning and memory process.

In addition to being involved in various aspects of the process of learning and memory, there are varying theories on the types of memories in which the hippocampus is involved. The hippocampus has been associated with verbal and nonverbal memory and the formation of declarative memories (Eichenbaum, 1999; Konkel & Cohen, 2009; Scoville & Milner, 1957; Squire, 1992). In addition, the hippocampus has been associated with a unique form of memory known as relational memory (Konkel & Cohen, 2009). Relational memory is believed to integrate different elements (items, locations, and temporal order) into a single memory code. Spatial-relational memory, then, is specific to the encoding, consolidation, and storage of visual-spatial information and to related elements (features, location, and spatial representations).

There is no consensus on how the hippocampus is involved in spatial-relational memory. One theory, based mostly on the rat/mouse hippocampus, has suggested that the hippocampus is critical for mapping the spatial environment through the use of place-cells in a map-like representation that updates constantly based on the person/animal’s position in space (Nakazawa, McHugh, Wilson, & Tonegawa, 2004; O’Keefe & Nadel, 1978). O’Keefe and Nadel believe that the hippocampus developed spatial representations known as a “Cognitive Map.” In
this theory based on a rodent model, the hippocampus “creates” and “maintains” a map-like representation of the environment, which is activated by the firing of location-specific “place cells” in the hippocampus, thereby mapping out the environment. The Multiple Trace Theory claims to include aspects of both O’Keefe and Nadel’s Cognitive Map Theory and a consolidation model, which proposes that the hippocampus is involved in re-experiencing both episodic and spatial memories, as well as the formation of semantic memories and spatial maps (Moscovitch, Nadel, Winocur, Gilboa, & Rosenbaum, 2005). Extending beyond the cognitive map theory, Eichenbaum (1999) has suggested that the hippocampus is involved in producing relational representations both of the environment and the stimuli within the environment. The benefit of such a system can result in encoding information from multiple sensory systems to create more holistic memory representations of environments and stimuli (Eichenbaum, 1999). However, our overall understanding of the hippocampus and its role in supporting memory, particularly spatial-relational memory, is still debated.

**Hippocampus Anatomy and Projections**

The hippocampus is a neuroanatomical structure with distinct defining features. It is located within the medial temporal lobes, curved in an “s”-like shape (Blumenfeld, 2002). The hippocampus, together with the dentate gyrus (DG) and subiculum, is part of the “hippocampal formation.” The hippocampus is a complex structure comprising multiple subsections, known as cornu Ammonis (CA1-CA4) and connections (Blumenfeld, 2002; Moscovitch et al., 2005; Squire, 1992). Section C4 is closest to the DG, and C1 is closest to the subiculum. Each of these structures depend upon one another within the brain to serve different functions related to learning novel information, and then to encoding, consolidating, storing, and retrieving it.

There are two main pathways within the hippocampus: the perforant and the alvear pathways. The first is known as the perforant pathway, which is a “loop” that begins in the
subiculum and moves across the hippocampal sulcus to the DG, where they project from the hippocampal formation through the fornix (Blumenfeld, 2002). In the perforant pathway, granule cells from DG project their axons on CA3 cells, which have direct connections to the cortex and the Schaffer collaterals, and eventually synapse on the dendrites of CA1 cells. From CA1, there are projections to the fornix and to the subiculum. Cells in the perforant pathway also project from the subiculum to the fornix and back to the entorhinal cortex. This pathway has been hypothesized to be involved in LTP and associative learning. Alternatively, in the alvear pathway, neurons project from the entorhinal cortex, to both the CA1 and CA3, to the subiculum (Blumenfeld, 2002). Both the perforant pathway and the alvear pathway are integral to hippocampal functioning.

The hippocampus works as part of a network in collaboration with other neuroanatomical structures. After information leaves the association areas of the cortex, where they are initially processed, they project to the entorhinal cortex, which serves as the primary input source to the hippocampus (Blumenfeld, 2002). Once information reaches the hippocampus and travels along either the perforant or alvear pathways, it projects back to the entorhinal cortex or to the fornix via the subiculum, and then across to other neuroanatomical structures (Blumenfeld, 2002). The connections between the hippocampus and other neuroanatomical structures are necessary in order to process different aspects of memory (Banich & Compton, 2011; Montaldi et al., 1998; Moscovitch et al., 2005; Strauss, Sherman, & Spreen, 2006). For instance, the hippocampus has bidirectional connections with the amygdala and cortical areas. These bidirectional connections may allow for the hippocampus to play a role in integrating information across cortical areas to form a more holistic memory of an event (Mayes, Montaldi, Migo, 2007). Therefore, we will briefly review the role of the hippocampus within the context of its networks.

The hippocampus has multiple projection networks that serve different types of memories. There are two major distinct pathways, the ventral pathway and the dorsolateral
pathway, which each serve distinct memory functions (Jung, Weiner, & McNaughton, 1994). It has been hypothesized that the ventral pathway mediates emotional learning (such as fear) through Papez’s limbic circuit, whereas the dorsal pathway is responsible for spatial learning and memory without the context of emotion and fear (Fanselow & Dong, 2010). The pathway for visuospatial information begins with reciprocal connections from the DG and projects to other areas of the brain such as the medial and lateral mammillary nuclei and the anterior thalamic complex. The distinction between these two major pathways is particularly relevant to our understanding of the role of the hippocampus in spatial-relational learning and memory.

Damage to the dorsal hippocampal pathway has been known to impact spatial cognition. Researchers have reported that a disruption in the pathway that connects the hippocampus with mammillary bodies and anterior thalamic nuclei may result in spatial relational deficits (Moscovitch et al., 2005). Moreover, disruption of the dorsal pathway using NMDAR Abs does not affect the ventral tract, suggesting independence of the two pathways (Fanselow & Dong, 2010). Thus, the dorsolateral hippocampal pathway is a site with multiple NMDARs and is responsible for spatial learning and declarative memory.

### The Hippocampus, Lateralization, and Spatial Memory

Spatial memory is likely lateralized to the right hemisphere, with greater representation for spatial activity. Unilateral lesions to right hippocampal areas are reported to cause deficits in visuospatial forms of learning and memory (Banich & Compton, 2011; Blumenfeld, 2002). However, this lateralization is particularly nuanced. In a comprehensive review of the literature on spatial memory, Burgess, Maguire, and O’Keefe (2002) reported that the right hippocampus is particularly responsible for memory of locations of items whereas the parahippocampus is involved in spatial processing, and the left hippocampus is associated with context-dependent
episodic or autobiographical memory. Despite the complexities, it is generally assumed that the right hippocampus is implicated in spatial memory.

There is a growing body of support for lateralization and subsequent functional impairment of spatial memory in the right hippocampus. In fact, physiological, lesion, and imaging studies using an SLE population have implicated right laterialized hippocampal structures in spatial memory impairment, extending to both encoding and delayed recall (Kozora, 2011; Ploner et al., 2000; Reber, Wong, & Buxton, 2002). The PHC has long been shown to also play a significant role in spatial memory, specifically in recall of contextual and spatial information (Eichenbaum, Yonelinas, & Ranganath, 2007). Lateralization of spatial memory function is also found in SLE research on delayed visual memory. Kozora (2011) compared non-NPSLE patients with HCs and discovered that although both groups had mild impairments in both verbal and visual memory, there was a notable correlation between visual memory deficits and right hippocampal neuronal damage. Although the right hippocampus is dominant in processing spatial information, it is also involved in nonspatial visual memory. A functional magnetic resonance imaging (fMRI) lesion study found that the right hippocampus and the right parahippocampal cortex (PHC) were essential for encoding of novel images (Reber, Wong & Buxton, 2002). Thus, not only is visual memory an important function of the hippocampus, it appears to be lateralized to the right hemisphere.

**Neuroanatomical Correlates of SLE**

The neuroanatomical structures and networks that are related to changes in cognitive dysfunction in SLE are quite heterogeneous. Although neuropsychological assessments are useful for identifying the functional impairment, they do not assess the presence and degree of SLE disease activity and associated neuroanatomical changes (Denburg & Denburg, 2002; Denburg et al., 1987a, 1987b). Therefore, researchers often integrate neuropsychological
findings within the context of a variety of animal and human models, including imaging and lesion studies. Integrating these multiple sources of information are valuable tools to understand the underlying mechanisms that likely contribute to the cognitive dysfunction found in particular disorders on neuropsychological testing.

In general, SLE patients experience multiple neuroanatomical insults. In addition to inflammation and other SLE associated symptoms, NPSLE patients may experience seizures, mood disorders, anxiety, psychosis, demyelination, and cerebrovascular disease; each of which could result in damage. SLE patients are frequently found to have global cerebral atrophy, infarcts, hemorrhages, venus sinus thrombosis, and white matter hyperintensities (WMHI; Appenzeller et al., 2007; Graham et al., 2003; Kozora et al., 2011; Watson et al., 2012). Some researchers believe the cognitive deficits observed in these patients are directly linked to anatomical changes within the brain (Kozora et al., 2011). It is highly likely that there is a direct link between these anatomical changes and the cognitive impairment seen in SLE.

Consistent with various neuropsychological studies that find cognitive deficits in multiple domains, it is suspected that SLE patients have multifocal or global damage in both cortical and subcortical areas. Not surprisingly, global atrophy and damage in subcortical, frontal, parietal, and temporal areas are commonly related to cognitive impairment (Jung et al., 2010; Sabbadini et al., 1999; Tomietto et al., 2007; Zhang et al., 2007). In addition, white matter tracts that connect cortical and subcortical areas are also affected by SLE, and often result in cognitive deficits most notably in processing speed (Emmer et al., 210; Jung et al., 2012). Tract damage may be due to a variety of factors, and it is currently unclear whether they are due to antibody activity on myelinated axons, vascular insults, or diminished white matter tracts resulting from grey matter lesions. Regardless, it is evident that SLE patients experience changes across multifocal cortical and subcortical regions.
Neuroanatomical changes in cortical regions are frequently implicated in SLE research. Monastero and colleagues (2001) argued that the diffuse pattern of neuropsychological deficits in both NPSLE and non-NPSLE participants are likely associated with cortical damage in the temporal, frontal, and parietal lobes. The diffuse nature of these difficulties, they argue, reflects frontotemporoparietal dysfunction, which is supported through PET/ single-photon emission computerized tomography (SPECT) findings. SPECT scans, which are similar to PET scans but use antibodies to link to a radioactive substance, suggest that microinfarction (tissue death due to lack of blood supply) and hypofusion (damaged due to decreased blood flow) most frequently occur in NSPLE patients in the parietal, frontal, and temporal lobes (Lin, Wang, Yen, & Lan, 1997). Frontal structures, in particular, are likely related to SLE deficits on neuropsychological tasks that require free-recall, preserved recognition, and organizational strategies (Paran et al., 2009; Wheeler, Stuss, & Tulving, 1997). Functional imaging furthermore found increased blood flow in bilateral frontoparietal regions for NPSLE patients as compared to patients with RA or HCs during a working memory task (Fitzgibbon et al., 2008). This finding suggests that patients with NPSLE use compensatory mechanisms and require more cognitive effort to complete a simple working memory task. Overall, it appears that SLE patients experience changes to cortical areas and have cognitive deficits associated with those changes.

Subcortical brain regions are also affected by SLE disease. When the type of errors produced by SLE patients on a basic cognitive screening measure was analyzed, their mistakes were more indicative of subcortical impairment rather than cortical impairment (Leritz, Brandt, Minor, Reis-Jenson, & Petri, 2000). Among subcortical regions, there has been convincing empirical support for hippocampal damage in SLE patients. Researchers have discovered strong associations among hippocampal atrophy and disease duration, NP involvement, corticosteroid use, antiphospholipid antibodies, and cognitive impairment in SLE patients (Appenzeller et al., 2006). Both non-NPSLE and NPSLE patients, compared to HCs, had
cerebral micro-infarcts (tissue death due to disruption of blood supply) and hyperintense areas (tissue abnormalities) related to hippocampal atrophy (Appenzeller et al., 2006). Moreover, the hippocampal volume loss observed in SLE patients progressed over a period of 19 months and was related to greater cognitive impairment, particularly in the memory domain (Appenzeller et al., 2006). Thus, a history of CNS disease activity that was related to hippocampal atrophy assessed at the start of the study predicted cognitive impairment at the 19-month follow-up assessment. It is evident, then, that the hippocampus is affected by SLE disease duration, and, as such, deficits in learning and memory are likely related to this damage. This supports some research suggesting a history of disease activity and accumulated disease burden may be related to the course and contributing factors of cognitive impairment in SLE. Particularly, that cognitive impairment is related to accumulated disease burden, a history of SLE disease activity that affects the CNS, as well as longer disease duration.

**Course of Neuroanatomical Changes in SLE**

The atrophy found within neuroanatomical structures is often present early on in diagnosis and increases as the disease progresses. Further, the damage observed in brain regions is often associated with concordant cognitive deficits. Through brain MRI studies, global cerebral atrophy was identified in 25% of newly diagnosed SLE patients (Petri et al., 2008). In this sample, focal lesions were associated with the presence of abnormal levels of anti-dsDNA antibodies. It appears that both focal and multifocal or global cortical atrophy is found early within the course of the disease.

Brain atrophy in SLE patients is not limited only to newly diagnosed patients. As the disease progresses, neuroanatomical damage also progresses (Appenzeller et al., 2007). Follow-up imaging after a period of 19 months found greater reduction in gray matter in the dorsolateral areas, frontal, and medial temporal lobes, including the hippocampus. In fact, MRI
and diffusion tensor imaging (DTI) findings from other aforementioned studies support a similar pattern of findings, with damage in frontal, occipital, and temporal lobes, the corpus callosum, and the cerebellum (Appenzeller et al., 2007). Appenzeller and colleagues discovered that white and grey matter reduction was more pronounced and related with the severity of cognitive function and number of cognitive domains impaired (2007). Furthermore, they were able to identify specific domains of cognitive function and corresponding neuroanatomical damage. General memory deficits were related with medial temporal lobes and frontal lobe damage. Finally, the researchers observed that longer disease duration was related to more pronounced cognitive deficits in patients. Therefore, it seems that damage to the brain is related both to severity of cognitive impairment and progression over time.
Part V: Antibodies and SLE

Research suggests that antibody involvement in SLE is associated with cognitive impairment; however, the specific relationship has yet to be identified. One goal of researchers is to identify specific cognitive deficits that are associated with Abs in SLE. Many patients with SLE have an Ab that causes damage against NMDAR, which are found in high density within the hippocampus and have been hypothesized to play a role in spatial and relational memory. With respect to SLE, NMDAR Abs cause cell death in the hippocampus (and other brain areas) and the resulting loss of these cells is also associated with cognitive decline. Therefore, it is critical to understand the relationship among NMDAR receptors, Abs, and the hippocampus to see if they are associated with specific spatial and relational learning and memory deficits.

The Immune System and Antibodies

In order to understand the specific mechanisms of Abs, it is important to understand the role of a healthy immune response. At the most basic level, when an individual is exposed to foreign molecules, often referred to as an antigen, white blood cells (lymphocytes) are activated to protect the body (Alberts et al., 2002; Campbell & Reece, 2002). Typically in a healthy immune system, B-lymphocytes (B cells) produce a type of protein, or antibody, to react with specific antigens (Aranow, Zhou, & Diamond, 2011; Campbell & Reece, 2002). Antibodies are comprised of a class of proteins known as immunoglobulins (IGs). When an antibody binds to an antigen-binding site on the surface of an antigen, it causes cell disposal by a variety of means depending on the IG classification. This process allows an individual to fight off infections or other foreign particles.

Autoantibodies are another classification of protein antibodies that are produced by the immune system and bind to receptors (autoreactive) within the body (self-antigens), including brain tissue or proteins in the body. Abs may serve an adaptive function and, often, serve to
regulate immune system function. Like antibodies, Abs are similarly produced by B-cells and, within a healthy immune system, they usually are expunged through apoptosis or other breakdown mechanisms within the immune system (Aranow et al., 2011). Some of the processes in which these Abs are destroyed or eliminated are not yet understood. In SLE, there are multiple failures in the body’s natural ability to purge these Abs. The possible explanations as to why these failures occur may be related to reduced B-cell receptor signal strength, high levels of B-cell activating factors, low levels of B-cells due to damage from medications or disease (Aranow et al., 2011). Undoubtedly, SLE patients experience poor regulation of Abs, which further exacerbates their disease.

**Autoantibodies in SLE**

Within SLE, the number and type of Abs are numerous, and there is some conjecture that these Abs are associated with cognitive impairment. A meta-analysis of Abs in SLE found that there are possibly 116 types of Abs, although the frequency of each type of antibody varies immensely (Sherer et al., 2004). Of the most frequently occurring Abs, almost all SLE patients tend to have antinuclear Abs (ANA), and a large portion of patients have high levels of anti-DNA Abs. Elevated levels of anti-double stranded DNA (dsDNA) Abs are found in approximately 30-60% of SLE patients and are thought to be specific to SLE (Chang et al., 2015; González-Albo & DeFelipe, 2000). NPSLE, specifically, is also associated with a high number of Abs. Some of the commonly found Abs in SLE include the antiphospholipid (aPL; anticardiolipin, aCL; and lupus anticoagulant, LAC), antineuronal (anti-NA), antiribosomal-P, antiganglioside, and antisynaptosomal antibodies (Abbott Mendonça & Dolman, 2003). Although research in this field is limited, there appears to be some support for the relationship between the presence of specific antibodies and cognitive impairment. For example, cognitive impairment is frequently associated with anti-NMDA, aCL, LAC, and several other Abs (Denburg, Carbotte, & Denburg, 1987b; Zandmann-Goddard, Chapman, & Shoenfeld, 2007). Given the numerous Abs
associated with SLE and the immense fluctuations in conjunction with disease activity, a review of the literature regarding each of these Abs and possible cognitive impairment is beyond the scope of this project. Therefore, we will briefly review the three most frequently studied Ab groups before focusing specifically on Abs that target the NMDAR.

Although there has been some evidence linking Abs to cognitive impairment, not all data supports this relationship. Petri and colleagues (2008) suggested that there is no relation between cognitive performance and anti-dsDNA or aPL antibodies early in the disease. Similarly, it is debated whether the presence of cognitive impairment fluctuates in the presence of current disease activity or whether the presence of cognitive impairment is a response to cumulative damage and activity. In fact, some researchers have found that patient age and disease duration, but not activity, are related to cognitive impairment (Diamond et al., 2006; McLaurin et al., 2005). Thus, it may be likely that cognitive deficits in SLE could be related to the cumulative, rather than an acute, response of Ab damage (Matus et al., 2007). Regardless of whether cognitive impairment is related to an acute or cumulative effect, it appears that there is at least some association between Abs and cognitive impairment. In light of the complex relationship between the major Ab classes and their influence on cognition, a brief background of some prominent Abs may help to provide more insight into their relation with cognitive deficits.

**Antineuronal Antibodies**

Antineuronal antibodies (ANAs) are a group of Abs that react to neuronal components (specifically nuclei) but are generally not well defined (Zandmann-Goddard et al., 2007). Overall, they are more frequently found in NPSLE than non-NPSLE patients (Colasanti et al., 2009; Denburg et al., 1987b). Some evidence suggests ANAs are related to cognitive impairment (Denburg et al., 1987b). However, the evidence is not overwhelming. Moreover, ANAs are nonspecific when binding to receptors, and, therefore, ANAs may serve as poor informants to
predict specific cognitive impairment. However, a subset of ANAs may be more specific when binding to neuroanatomical receptors, and, as such, may provide insight on cognitive impairment (see Anti-dsDNA antibodies).

**Antiphospholipid, Anticardiolipin, and Antiribosomal P Antibodies**

Among the most studied of all the Abs are aPL, aCL, and Antiribosomal P Abs. The aPL Abs are related to thrombosis of intracranial vessels and are related to focal events such as headache, chorea, strokes, and seizures that may be related to diffuse cognitive dysfunction (Hanly, 2011). Overall, many studies observed an association between aPLs and cognitive impairment in NPLSE (Zandmann-Goddard et al., 2007). The greatest impairment was in verbal memory (Paran et al., 2009). Other research observed that aPL Abs was associated with lesions, brain atrophy, and impaired executive functions (Appenzeller et al., 2007; Tomietto et al., 2007). However, not every study found aPL antibody presence predicting cognitive deficits (Emori et al., 2005; Petri et al., 2008). Consequently, the aPL Abs are not consistent predictors of the cognitive impairments found in SLE.

There has been some evidence suggesting aCL Abs is associated with cognitive impairment. The aCL Abs are related to an increase in processing speed and executive dysfunction (Hanly, Hong, Smith, & Fisk, 1999). Yet, these cognitive domains do not account for poor spatial memory.

Further, it is possible that Antiribosomal P Abs have an indirect influence on cognitive function. There is some evidence to suggest that Antiribosomal P Abs may be associated with psychosis and depression in SLE (Hanly, 2011). Thus, these Abs are both poor predictors of specific cognitive dysfunction that SLE patients experience.

**Anti-DNA antibodies**
The anti-DNA antibodies refer to a large class of ANAs that is correlated with disease activity and is cross-reactive with different receptors within the brain. Cross-reactivity refers to when an antibody reacts with similar antigens on different proteins. Within the anti-DNA antibodies classification, there are single-stranded DNA antibodies (ssDNA) and double-stranded DNA antibodies (dsDNA). A subset of anti-dsDNA antibodies, known as DNRAbs, is thought to react against both dsDNA and N-methyl D-aspartate receptors (NMDAR) Abs, and, in turn, is related to impaired cognitive ability in SLE patients (DeGiorgio et al., 2001, Lapteva et al., 2006, Omdal et al., 2005). Thus DNRAbs and NMDAR Abs are both terms that are used to describe a subclass of anti-dsDNA antibodies.

The Structure, Function, and Role of Anti-N-methyl-D-aspartate Receptors.

The mechanism of action for NMDARs is a function of their specific structure. NMDA receptors are ionotropic (or fast-acting), excitatory, and glutamatergic receptors (Meyer & Quenzer, 2005). This receptor is structured into a tetramer or four subunits, typically GluN1 (also called NR1) and GluN2A-GluN2D (also called NR2A-NR2D) or GluN3 (NR3). An NMDA receptor includes two GluN1 subunits and two GluN2 or GluN3 subunits (Furukawa, Singh, Mancusso, & Gouaux, 2005). While GluN1 occurs throughout the brain, GluN2A and GluN2B are most prevalent in the CA1 unit of the hippocampus, as well as in the amygdala (Aranow, Diamond, & Mackay, 2010; Danysz & Parsons, 1998; Omdal et al., 2005). These different subunits include specific binding sites to glutamate or a co-agonist and are a prerequisite for the ion channel to open.

NMDA receptors are a unique protein receptor complex that requires several important steps to function. In order for NMDAR channels to open, the amino acid glutamate must be present and bind to the binding site on GluN2 (Furukawa et al., 2005; Meyer & Quenzer, 2005; Nestler, Hyman, & Malenka, 2009). They also have a binding site for the amino acids glycine or D-serine (a co-agonist with glutamate), which bind to GluN1. Even in the presence of these
amino acids, NMDAR channels typically remain blocked due to binding with magnesium ions (Mg^{2+}). However, zinc ions (Zn^{2+}) also serve as to block this channel (Amico-Ruvio, Murthy, Smith, & Popescu, 2011). Since the NMDAR are voltage dependent, the Mg^{2+} (or Zn^{2+}) ions will continue to block the channel unless the cell membrane is depolarized by another source of excitation (stimulation will typically occur on another receptor such as AMPA). Thus, NDMAR channels only become active in the presence of glutamate, a co-agonist, and a depolarized membrane.

Activation of NMDAR channels leads to the movement of ions within the neuron as well as secondary effects. Once the NMDAR channels open, NMDAR channels become permeable to sodium (Na^+), potassium (K^+), and Calcium (Ca^{2+}; Meyer & Quenzer, 2005; Nestler et al., 2009). Due to their ability to conduct Ca^{2+}, they are also able to function as a second messenger system in the post-synaptic cell, in which intracellular receptors move into the post-synaptic membrane, which, in turn, increases the post-synaptic cell’s signal strength the next time it experiences excitatory stimulation (Meyer & Quenzer, 2005). In fact, it is through persistent stimulation during this process that NMDA receptors located in the hippocampus play an important role in a form of synaptic plasticity known as long-term potentiation (LTP) and the formation of memory. LTP facilitates the consolidation of memories to a more permanent store through structural changes in other cortical areas (Squire, 1992). Therefore NMDAs hold a complex role within the brain and are implicated in learning and memory.

Although NMDAR stimulation produces a beneficial role in the formation of memory, excessive glutamate has detrimental effects. Prolonged exposure to glutamate and Ca^{2+} influx results in excitotoxicity, or cell death. In particular, CA^{2+} targets the mitochondria found within the neuron (Aranow et al., 2010; Meyer & Quenzer, 2005). Given the NMDAR’s integral role in enhancing signal strength to glutamate and subsequent learning and memory, damage to cells with NMDARs could potentially lead to serious cognitive consequences.
**NMDARs in the Hippocampus.**

Within the hippocampus, there are high-density areas of NMDA receptors. Some of the highest levels of NMDARs are found in the CA1 in the hippocampus (Ozawa, Kamiya, & Tsuzuki, 1998). However, other areas of the hippocampus also have high levels of NMDARs, such as the CA3 area (Lee & Kesner, 2002), which is less studied as compared to the other subunits of the hippocampus. It is suggested that the CA3 area is essential in spatial working memory of a new environment whereas CA1 combined with the DG are involved in the retention of information for longer delay periods (Lee & Kesner, 2002). Therefore, the CA1 and CA3 areas of the hippocampus are likely to be susceptible to NMDAR Ab damage along with impaired long-term memory formation and spatial/relational memory.

**Anti-N-methyl-D-aspartate Receptor Antibodies**

The NMDAR Abs are referred to by several names. Anti-NMDAR antibodies are autoantibodies that bind to NMDAR. More specifically, they are sometimes referred to as Anti-NR2 antibodies in the literature due to their tendency to bind to the GluN2 subunits of NMDAR. Moreover, DNRAbs are cross-reactive antibodies and, therefore, are likely to target both DNA and NMDAR (DeGiorgio et al., 2001). In the following section, these terms are each used in order to describe the NMDAR Abs that are implicated in SLE pathology.

DNRAbs are thought to produce an antagonistic response against the NMDA receptors. The process by which they do this is by binding to the GluN2A or GluN2B subunit of the NMDAR. Binding to this region results in an influx of $\text{Ca}^{2+}$, which, at low levels, promotes excitation of these glutamatergic receptors. Animal models of NMDAR-mediated neurotoxicity suggest a dosage-dependent causal role of NMDAR Abs and cell death (DeGiorgio et al., 2001; Faust et al., 2010). At high concentrations, they continue to permit the flow of $\text{Ca}^{2+}$ into the neuron, which leads to mitochondrial damage and apoptosis (cell death) in the CA1
hippocampus (the highly dense NMDAR area of the hippocampus; Faust et al., 2010; Kowal et al., 2006; Mackay et al., 2011). Moreover, animal model studies demonstrate that the cell death caused by NMDAR Abs within the hippocampus cause spatial memory impairment (Chang et al., 2015; Kowal et al., 2004; Mackay et al., 2011). As a result, these animal models support the hypothesis that DNRAbs may play a role in cognitive dysfunction by causing cell death and disrupting brain areas with high affinity NMDARs. Therefore, animal models support the hypothesis that NMDAR Abs target NMDAR in the hippocampus, cause cell death, and result in functional impairment.

The NMDAR Abs are frequently present in brain regions where NMDA receptors are highly concentrated. In SLE, the NMDAR Abs are known to bind with NR2 subunits, which are commonly found in the hippocampus (Kowal, DeGiorgio, Nakaoka, Diamond, & Volpe, 2004; Zandman-Goddard et al., 2007). Studies utilizing a variety of imaging procedures, including PET and proton magnetic resonance spectroscopy (or MRS), find high concentrations of anti-NR2 antibodies in visuospatial and memory regions of the brain, such as the hippocampus, of SLE patients (Lapteva et al., 2006; Mackay et al., 2015). Thus, NMDAR Abs in SLE preferentially target the hippocampus due to the high affinity of receptors, which may result in cognitive deficits specific to hippocampal-related functions.

Researchers are still developing hypotheses to explain how NMDAR Abs access the brain. It is unclear yet whether these Abs are produced locally in the brain or cross the blood-brain barrier (BBB), as there is evidence for both occurrences (DeGiorgio et al., 2001). Speculatively, the recent discovery of a lymphatic system within the brain may explain how immune cells and molecules access the CNS (Louveau et al., 2015). However, due to the recency of this discovery, it is unclear yet how this system is affected in SLE.

There is a greater body of research examining NMDAR Abs within blood serum and whether they cross the BBB. If the BBB is intact, NMDAR Abs from blood serum are unable to
penetrate the BBB, and, therefore, fail to enter the brain. However, NMDAR Abs may cross the BBB when there is insult. BBB insult may be transient, in which case the Abs may cross the BBB during episodes of disease activity or NP manifestations that result in an insult to the BBB. It is suggested that this inflammatory autoimmune disease activates endothelial cells of the BBB, which would allow Abs to access brain regions that have been previously restricted by the BBB (Abbot et al., 2003). It is also possible that Abs could penetrate the BBB after SLE-related damage through an inflammatory process or an event such as vascular deterioration, stroke, stress, nicotine exposure, or elevations in epinephrine (Abbot et al., 2003; Hanly et al., 1992; Huerta et al., 2006). SLE patients are likely to have repeated insults against the BBB over the course of their disease, particularly for NPSLE individuals, allowing Abs to access the brain and potentially cause cell death and subsequent cognitive impairment (Abbott et al., 2003). It may even be possible that NMDAR Ab directly react to and damage the endothelium of the vessels that protect the BBB, causing a breach in the BBB (Brimberg et al., 2015). Although the exact mechanism by which NMDARs access the brain is unclear, overall, there are multiple factors that could contribute to a BBB breach in SLE patients.

Although there is an established relationship between NMDAR Abs, cell death, and resulting functional impairment, researchers seek to explore the potential role of NMDAR Abs in other aspects of SLE. DNRAbs may be present in blood serum (in which they can affect organs throughout the body, such as the kidney causing permanent kidney damage; DeGiorgio et al., 2001) and in CSF, where they affect the brain. Once present in CSF, NMDAR Abs are found in areas such as the amygdala (affecting emotional regulation), hypothalamus, and hippocampus and cause structural and functional deficits related to their targeted areas. Their presence has been associated with NPSLE symptoms including seizures, acute confusional state, mood and anxiety disorders, psychosis, and severe cognitive dysfunction (Arinuma et al., 2008; Mackay et al., 2011). For example, when NMDAR Abs targets the amygdala, they can induce anxiety-like
symptoms in addition to the known cognitive deficits in learning and memory (Huerta, Kowal, DeGiorgio, Volpe, & Diamond, 2006). In summary, NMDAR Abs may produce different symptoms depending on the targeted area. For the purpose of this research, we will look specifically at NMDAR Abs within the hippocampus and its known role in spatial and relational memory.

**Spatial/Relational Learning and Memory Deficits Associated with NMDAR Autoantibodies in Systemic Lupus Erythematosus**

Although research on the cognitive implications of NMDAR Ab in SLE patients is in the preliminary stages, there have been a few noteworthy animal and human studies. These studies using mice models find that NMDAR Abs often bind with receptors located in the forebrain and hippocampus (DeGiorgio et al., 2001; Hanly, 2011). Human studies also find promising results with regard to association between anti-NMDAR and cognitive dysfunction, most specifically in learning and memory domains. Studies are beginning to support the relationship between cognitive deficits (particularly in visuospatial and learning and memory domains) and the presence of NMDAR Abs (Lapteva et al., 2006, Omdal et al., 2005). Thus, the association between NMDAR Ab positivity and cognitive deficits in the learning and memory domain appears promising.

Research is also beginning to emerge associating the presence of NMDAR Abs with cognitive impairment, while controlling common factors (e.g., age) associated with cognitive decline. A recent PET study published by Mackay and colleagues (2015) finds an association between an increase in glucose metabolism in the hippocampus and serum DNRAb titres, as well as memory impairment and mood alterations. They further conclude that impairment is not related to disease duration and supported previous reports that brain atrophy is involved in the early stages of the disease. In a separate proton magnetic resonance spectroscopy (MRS)
imaging study, decline in expected cognitive performance frequently occurred in visuospatial and memory domains in SLE patients with anti-NR2 antibodies (Lapteva et al., 2006). Interestingly, in this study there is no association between cognitive dysfunction and aCL, anti-P, or other anti-dsDNA antibodies. Thus, there appears to be sufficient evidence linking SLE Abs, NMDAR, the hippocampus, and spatial impairment to each other (Omdal et al., 2005). Therefore, there is at least some support that anti-NR2 antibodies and DNRAbs are associated with memory decline in SLE patients.

To date, only one study to date by Chang and colleagues (2015) reports spatial memory deficits in an SLE population combining both murine models and human research. In this study, 46 SLE (22 NPSLE) and 27 HCs completed a battery of neuropsychological assessments, including a laboratory-developed computerized measure of spatial recall. In the computerized measure, all subjects observed drawings of objects in a 2 x 2 array. After studying the array, participants answered a spatial (i.e., “Was object A above/below/left/right of object B?”) or a nonspatial question (i.e., “Was there a banana in the picture?”). This study demonstrates that NPSLE patients with positive serum titres of DNRAb perform worse on spatial items than HCs and SLE patients with negative DNRAb titres. Accuracy for the non-spatial items does not vary by group, however, suggesting DNRAb titres are uniquely associated with spatial impairment. While this study examines the relationship among SLE, DNRAb presence, hippocampal damage, and spatial memory deficits, it is only a preliminary evaluation. The current project aims to look further examine the relationship between DNRAb and spatial memory deficits and to explore relationships between DNRAb and relational memory deficits within an SLE population.

**Summary**
In summary, there appears to be a developing body of evidence linking NMDAR Abs to hippocampal damage and to cognitive impairment, specifically in the learning and memory domain. By better understanding spatial and relational learning and memory cognitive deficits in NMDAR Ab positive and negative SLE patients, we will have a better understanding of the damage inflicted on NMDAR Abs. It is critical to further investigate the mechanisms of SLE, particularly with regards to Ab activity in NPSLE, in order to develop more reliable biomarkers for disease and resulting deficits, including cognitive deficits, in order to provide the most appropriate and personalized therapeutic tools.

**Specific Aims:**

**Aim 1:** To examine if there were differences between SLE and HC participants for spatial memory deficits (Aim 1a) and whether DNRAbs are uniquely associated with spatial memory deficits (Aim 1b).

1a. The first goal was to examine the presence and extent of spatial memory deficits in SLE patients versus HCs. The literature supports the possibility that SLE patients have deficits on measures of visuospatial memory and recognition. More specifically, SLE patients exhibit deficits in their ability to accurately duplicate a previously drawn complex figure (Brey et al., 2002; Coín-Mejías et al., 2008; Ferstl et al., 1992; Monastero et al., 2001; Nowicka-Sauer et al., 2011; Petri et al., 2008; Vogel et al., 2011), visuospatial recognition (Kozora et al., 2011), and memory the spatial arrangement of various objects (Chang et al., 2015).

Data were collected using the computerized measure from Chang and colleagues’ paper (2015). The outcome measure, correct recall, examined whether the type of question (spatial or nonspatial) and task difficulty (2x2 array—easier—or 3x2 array—harder—) influenced performance by participant group (SLE and HC). The analysis consisted of a 2 x 2 x 2 mixed ANOVA, Group (HC and SLE) x Memory Type (spatial and nonspatial) x Item Difficulty (easier
and harder), exploring the main effects and interaction of these variables as a function of the proportion of correct responses. It was hypothesized that SLE patients would perform worse overall on measures of spatial memory as compared to HCs. It was predicted that there would be no difference between groups on nonspatial questions. There was also a consideration regarding whether the difficulty of the task would affect performance. It was predicted that, overall, all participants would have higher accuracy on the 2x2 array as compared to the 3x2 array, but that the HCs would perform better than the SLE patients for both levels of difficulty.

1b. The second part of this aim was to examine whether any observed spatial memory deficits were associated with DNRAbs. DNRAbs are a class of dsDNA antibodies that cross-react with dsDNA and NMDA receptors. In SLE, NMDAR Abs often bind to receptors located in the forebrain and hippocampus (DeGiorgio et al., 2001; Hanly, 2011). The hippocampus is thought to be involved in verbal and nonverbal memory, particularly spatial working memory (Konkel & Cohen, 2009). Furthermore, there is some support suggesting a relationship between visuospatial learning and memory domains and NMDAR Ab presence (Chang et al., 2015; Lapteva et al., 2006, Omdal et al., 2005). In animal models, spatial memory has also been associated with hippocampal place cells in the CA1 area of the hippocampus, an area that is dependent on NMDARs (Nakazawa, McHugh, Wilson, & Tonegawa, 2004). Because of the apparent relationship among NMDAR, hippocampal function, and spatial memory within mouse models, this project examined whether there were deficits in hippocampal-dependent spatial memory within a SLE population.

Previously Chang and colleagues (2015), reported that the DNRAb+ SLE group performed worse than HCs however, the DNRAb- SLE group did not differ significantly from HCs. Given this finding, the current project also investigated whether DNRAb status was related to performance on this spatial memory task. Therefore, analyses were conducted using a 2 x 2
x 3 mixed ANOVA, for Group (HC, DNRAb-, and DNRAb+) x Memory Type (spatial and nonspatial) x Item Difficulty (easier and harder), to explore the main effects and interaction of these variables as a function of the proportion of correct responses. It was hypothesized that HCs would have a significantly higher performance than the SLE patients on spatial questions; however, it was expected that this relationship would be more pronounced for DNRAb+ for both the easy and hard sets. Again, it was predicted that the groups would have similar accuracy for nonspatial questions.

Aim 2: To examine if there were associations between relational learning deficits and individuals diagnosed with SLE (Aim 2a) and whether DNRAbs status specifically predicted relational learning deficits (Aim 2b). An exploratory analysis assessed whether group (Aim 2c) and DNRAb status (Aim 2d) influenced type of errors participants made.

2a. A few studies have examined visuospatial learning and memory in SLE patients; however, to our knowledge, none to date have specifically investigated relational learning. Relational learning is important because it encompasses encoding of different elements (items, locations, and temporal order) into a single memory code and is a hippocampal-dependent process. However, few tasks are designed to specifically measure relational learning. Because preliminary research indicates that SLE patients have deficits in visuospatial memory and experience hippocampal damage over the course of their disease, it is important to examine the full range of memories that are, in part, hippocampal dependent to fully understand deficits that might arise for individuals with SLE.

A novel computerized measure was developed to evaluate relational learning among unrelated shapes for which people had to remember specific shapes (e.g., a square and a circle) and their spatial-relationship to each other (e.g., the square was above the circle). In this task, 2 shapes were paired with each shape occupying a single location within a 2 x 2 array.
Participants were asked to study 12 sets of these shape pairs and to identify the learned sets from non-learned (foil) sets. The foil sets included three different types: 1) relational recognition (i.e., one of the shapes had been moved), 2) object recognition (i.e., one of the shapes had changed), and 3) spoiler items (i.e., a novel shape was introduced that had not been presented earlier). The task was repeated over three Blocks with the same 12 target variables but different foils over each Block. A 3 x 2 repeated measures ANOVA, for Group (HC and SLE) x Block (1, 2, and 3), was conducted. It was hypothesized that HC s would have higher accuracy on the relational learning task than SLE patients. It was also expected that performance would improve over the course of the three Blocks equally for both groups. An exploratory analysis assessed whether the two groups differed in the type of errors (i.e., the type of foil sets) that were made.

2b. Furthermore, as DNRAb levels are associated with hippocampal damage, the present study investigated whether this factor influenced performance on the relational task. A 3 x 3 repeated measures ANOVA was conducted for Group (HC, DNRAb-, and DNRAb+) x Block (1, 2, and 3). It was hypothesized that DNRAb+ participants would have more pronounced deficits than the DNARb- and HC groups. It was further hypothesized that performance would improve with each successive Block across all groups. However, it was hypothesized that the degree of improvement would vary as a function of group. It was predicted that performance would improve with Block equally for the HC and DNRAb- groups. However, due to suspected hippocampal damage in the DNRAb+ group, it was predicted that the DNRAb+ group would demonstrate less improvement across the blocks as compared to the HC and DNRAb- groups. An exploratory analysis assessed whether the group and DNRAb status influenced the type of errors (i.e., the type of foil sets) that participants made.
Aim 3: To determine whether other factors were related to performance on the performance for the spatial memory task (Aims 3a-c) and relational learning task (Aims 3d-f). The factors that were considered included background demographics (age, education), SLE disease characteristics (i.e., disease duration), affective functioning (such as scores on depression and anxiety inventories), and neuropsychological assessments (including the paper-and-pencil tasks and the ANAM).

The purpose of this aim was to determine the extent to which such factors (demographic, psychological, or cognitive) are related to spatial and relational memory deficits found in participants with SLE. Moreover, presence of DNRAbs were investigated to determine whether they uniquely predicted relational learning and spatial memory deficits. The relationships between these factors and performance on the relational learning and spatial memory tasks were analyzed through correlations among the continuous variables (i.e., age, disease duration, scores on depression/anxiety inventories, and neuropsychological measures). Significant correlations were further explored in multiple regression analyses to assess the degree to which those significant variables predict performance on the Relational Learning and Spatial Memory tasks. As disease duration is typically associated with cognitive impairment, it was predicted disease duration would be correlated with performance on the tasks. Furthermore, since performance on the RCFT and Trailmaking Tests, as well as the ANAM tasks, are often poorer in SLE patients, it was predicted performance on these neuropsychological measures would be correlated with performance. However, it was predicted that basic visuoperception is typically intact in SLE patients, performance on the Judgment of Line Orientation test, would not be related to performance on either the Relational Learning or Spatial Memory test.
CHAPTER II

Methods

Cognitive assessment used traditional neuropsychological measures and behavioral measures, which were completed on the same day and took approximately 60 minutes to complete. Clinical evaluations of the patients' disease activity were completed within 2 weeks of the cognitive assessment. SLE patients were tested during periods of stable disease activity and medication use.

Participants

Two cohorts of SLE patients and HCs were included in this study. A comparison of recruitment, inclusion, and exclusion criteria for the cohorts A & B is displayed in Table 5 (See Appendix). Data from Cohort A was previously collected. Data from Cohort B was collected from newly recruited participants, who were participating in a 3-year longitudinal study. The data for Cohorts A and B were combined for the Spatial Memory Task in order to increase the statistical power for the spatial memory task (Chang et al., 2015). Cohort B only completed the relational memory task to directly assess relational memory. For both cohorts, SLE patients were divided into two groups: those with high circulating levels of NMDAR Abs (DNRAb+) and those with low circulating levels of anti-NMDAR Abs (DNRAb-). However, the investigators remained blind to the anti-NMDAR Ab status until data collection was completed, which resulted in unequal Ab status groups in Cohort A. However, Ab status groups were similar in Cohort B. Antibody status was determined through serum analysis, which was collected from SLE patients and HCs. To determine DNRAb status, Ab serum levels were obtained from HC participants. The mean level of Abs in HC participants was used to determine Ab status in the SLE groups. Participants in the DNRAb+ group had Ab levels greater than two SD above the mean Ab levels of the HC group.
After the recruitment of SLE participants, HCs were recruited and matched for gender, age (within three years of the SLE group members), and education at the group level.

Across both cohorts, there were a total of 104 participants. Of these participants, two participants were excluded because their group status data were missing. Thus, a total of 102 participants were available for analyses comparing HC and SLE groups. The sample included 42 HCs and 60 SLE patients. Of the SLE participants, 34 were DNRAb-, and 21 were DNRAb+. Five SLE patients from Cohort A were run in the study but were not included in the Ab analysis, because Ab data could not be analyzed due to error on the part of the laboratory that conducted the assay. Therefore, when analyses included Ab status, the data from only 97 participants were available. Descriptions regarding the participants recruited in Cohort A (n = 72) and Cohort B (n = 30) are provided below. The sample was representative of the demographic groups affected by SLE. It was both racially and ethnically diverse (see Tables 6 and 7 for descriptive demographics). The SLE groups were comprised of 23 African Americans (54.76%), 2 Asian individuals (4.76%), 6 Hispanic participants (14.28%), 8 Caucasian individuals (19.04%), and 3 individuals who indicated “other” or left the background history form unmarked (7.14%). The HC group included 39 African Americans (65%), 1 Asian individual (1.67), 11 Hispanic participants (18.33%), 7 Caucasian individuals (11.67%), and 2 individuals who indicated “other” or left the background history form unmarked (3.33%).

The Cohort A group included 74 female participants whose data was collected during a previous study. Of these 74 Cohort A participants, two potential participants within the SLE group provided consent but could not complete the neuropsychological and behavioral testing due to an increase in disease activity. Of these participants, 33 were HCs and 39 were SLE patients (11 DNRAb +, 23 DNRAb -, 5 unknown Ab status). A third SLE patient was excluded from analysis because her lack of proficiency with the English language prohibited her ability to
understand task instructions. One HC was excluded from analysis due to impaired performance across multiple neuropsychological measures, which suggested impairment in cognitive functioning. Another healthy control was excluded from analyses because of their high education level. This Cohort also included a group of 10 HC and four SLE that were added to increase the power following the conclusion of the initial study. Participants in Cohort A completed the computerized spatial memory task but not the neuropsychological evaluation.

Cohort B included a total of 32 participants. Among these participants, 21 were diagnosed with SLE (2 male; 11 DNRAb-, 10 DNRAb+) and 11 were HC (0 male). Data from one HC were not included in the analyses due to impaired performance across multiple neuropsychological and cognitive measures, suggesting impaired cognitive functioning. A second healthy control withdrew from the study prior to beginning neuropsychological testing. Due to a computer error, the data from the relational learning task were not recorded for one DNRAb- participant. Another computer program error resulted in the exclusion of the ANAM Sleep Scale for 6 HCs.

Both Cohorts - Measures

Neuropsychological Assessment

Both cohorts were administered a neuropsychological evaluation; however, the evaluations were quite different between Cohort A and B. Two of the measures administered to Cohort A were also administered to Cohort B because the measures revealed significant group differences between SLE and HCs. The two measures included for both cohorts were: (1) the Rey Osterrieth Complex Figure Test (Meyers & Meyers, 1995), and (2) the Trail Making Test (A and B; Bowie & Harvey, 2006).
The Rey-Osterrieth Complex Figure Test (RCFT): This task examines visuospatial constructional ability and visual memory. Participants were asked to copy a complex geometrical figure onto a blank sheet of paper. Following a 20-minute delay, during which they completed the computerized Spatial Memory Task (see description below), they were asked to draw the figure from memory. The raw score was calculated based on the accuracy and placement of each component of the figure by using the Meyer's and Meyer's manual (1995). Additional information was obtained using a separate, second scoring procedure, the Boston Qualitative Scoring System, but will not be reported on within this dissertation (Stern et al., 1999).

Trail Making Test, Parts A & B (TMT): The TMT is used as a measure of visual scanning, attention, processing speed, and rapid sequencing. In Part A, participants were required to quickly scan a page of randomly arranged numbers (1-25) and draw lines connecting numbers in sequential order. In Part B, participants again were asked to quickly draw lines connecting numbers and letters in sequential order alternating between numbers and letters (i.e., 1-A-2-B…13.). The number of seconds it took to finish the task was recorded. Errors were immediately corrected and also recorded.

Behavioral assessment

Spatial Memory Task: This research laboratory developed the Spatial Memory task (Chang et al., 2015). It was designed to assess memory for spatial arrangements of items (spatial memory) or presence of items (nonspatial memory). The task consists of participants viewing 4 or 6 line drawings of common objects (apple, horse) arranged in an unseen 2x2 (easier) or a 3x2 (harder) matrix and then answering either a spatial or nonspatial question.
At the start of the trial, participants were given five seconds to study a displayed matrix. Then, one of two questions was displayed: a) assessing non-spatial memory (e.g., Was an airplane present?) or b) assessing spatial memory (e.g., Was the tiger above the orange?). Participants responded by pressing the "1" or "2" button on the numeric pad of their keyboard, which corresponded to the answer on the monitor. For example, if the question "Was an airplane present?" appeared on the screen, below the question, a prompt will read "Press '1' for yes, Press '2' for no." Participants had 6000 ms to respond to the stimuli. There were 2 practice trials followed by 64 test trials. Both accuracy and reaction time were recorded and analyzed.

**Mood Assessments**

All subjects completed self-report mood scales, including the Beck Depression Inventory – second edition (BDI-II; measures self-reported presence of current depressive symptoms; Beck, Steer, & Brown, 1996; Gladman et al., 1997) and the State-Trait Anxiety Inventory, Form Y (STAI; measures self-reported state and trait levels of anxiety; Julian, 2011; Spielberger, Gorsuch, Lushene, Vagg, & Jacobs, 1983.). The STAI is a 40-item self-report questionnaire that has two subscales: 20-items regarding current (state) anxiety and 20-items regarding overall (trait) levels of anxiety. The scores range from 20-80, with higher scores suggestive of higher levels of anxiety.

**Cohort B – Specific Measures**

The remaining measures were only assessed with Cohort B. The Judgment of Line Orientation (Form V) measures was included to ensure that SLE patients had normal visuospatial ability in perceiving angles (Benton, Sivan, Hamscher, Varney, & Spreen, 1994). Additional neuropsychological assessment was assessed using ANAM, a standard neuropsychological battery often used in SLE research (Ad Hoc Committee on Lupus Response
Criteria: Cognition Subcommittee, 2007). Moreover, as part of the ANAM, a mood assessment was administered. Finally, a relational learning task was also included to further assess hippocampal-dependent learning.

**Judgment of Line Orientation (JLO):** The JLO is used as a measure of simple visuospatial ability (Benton et al., 1994). Participants observed a set of lines arranged in a semi-circle from 0 to 180 degrees. The lines were labeled from one to eleven, and were equally spaced spanning a full 180 degrees. Above the semi-circle, two lines were presented and participants had to match the appropriate angle the two lines made using the lines within the 180-degree semi-circle. Responses were recorded as correct when both lines are identified.

**Automated Neuropsychological Assessment Metrics (ANAM):** The ANAM is a computerized measure designed to assess cognitive functions related to visuospatial ability, attention/working memory, executive functions, information processing speed, fine motor speed, coordination, mood, and fatigue. The ANAM was selected because it is commonly used within the SLE community to assess cognitive deficits due to its validity, ease of use, and cost-effectiveness (Ad Hoc Committee on Lupus Response Criteria: Cognition Subcommittee, 2007). The following subtests were selected from the ANAM battery: Computer Experience Scale (a self-report measure regarding exposure to computers and frequency of use), Handedness Scale (self-report of dominant hand), Mood Scale (self-report of current mood on dimensions of happiness and sadness), Sleep Scale (self-report of fatigue and alertness), Simple Reaction Time, Matching Grids, Matching to Sample, Running Memory Continuous Performance Task (CPT), and Go/No-Go. The Simple Reaction Time, Matching Grids, Matching To Sample, Running Memory CPT, and Go/No-Go tasks each relied on the use of a mouse pad. Participants were instructed that they must respond by pressing 1 (left button on the standard computer mouse) and/or 2 (right button on the standard mouse) according to the instructions on
the screen for each task. The ANAM provided visual instructions indicating which button was 1 (left) or 2 (right).

**Simple Reaction Time (RT):** The Simple RT task measures simple attention through reaction time and vigilance. A large symbol (*) appeared in the center of a computer display. Participants were instructed to respond as quickly as possible by pressing only the 1 (“left”) mouse button each time the * appeared. Otherwise, they did not respond.

**Matching Grids:** The Matching Grids task measures visuospatial information processing. Two grids were presented side by side and the participants are asked to press their mouse pad when they appeared, with the buttons corresponding to whether the grids matched (1 mouse button) or did not match (2 mouse button).

**Matching to Sample:** The Matching to Sample task measures of spatial information processing and visuospatial working memory. A 4x4 grid appeared on the screen with a various number of boxes shaded in. After it disappeared, two new 4x4 grids appeared side by side. The participants were asked to select which one of the new grids matched the previous display by selecting the left grid (1 mouse button) or right grid (2 mouse button).

**Running Memory CPT:** The Running Memory CPT measures attention, concentration, and working memory using the design of a 1-back task. A series of numbers appeared on the screen. For each number that appeared, the participant was asked to press the 1-mouse button if it matched the previous number or the 2-mouse button if it did not.

**Go/No-Go Task:** The Go/No-Go Task measures of attention and response inhibition. On this task, individuals pressed the 1 mouse button for each time the symbol “**” appeared on the display, but they were instructed to inhibit their response when the letter “o” appeared on the screen.
Behavioral Assessment

**Relational Learning Task**: This task was designed to assess the ability to remember the relational configuration of spatial-oriented stimuli. A set included two shapes, and each shape was located in a unique quadrant of an unseen 2 x 2 array creating a particular relational configuration. The 12 sets appeared one at a time and memory for these configurations were tested three times using a recognition test. The possible set of configurations included the two objects being presented either next to each other (e.g., top left and top right), on top of each other (e.g., top left and bottom left), or diagonal from each other (e.g., top left and bottom right).

In the first Block, 12 object sets appeared one at a time for 10 seconds each, and the participants were asked to study the objects and their configuration. After the 12 object sets were shown, a new group of 24 object sets (12 from the first phase and 12 foils, previously unseen sets) were then presented to assess recognition. The 12 foils consisted of three different recognition types: 1) relational recognition (i.e., one of the shapes had been moved), 2) object recognition (i.e., one of the shapes had changed), and 3) spoiler items (i.e., a novel shape was introduced that had not been presented earlier). The participants were asked to identify the sets that were previously shown by pressing the ‘A’ key (match) or the sets that were new by pressing the ‘L’ key (no match). The key codes were continuously presented on the screen during the recognition test. Participants had 3000 ms to respond. After a response or the 3000 ms past, there was a 500 ms delay before the next set was presented. This learning and test phase was repeated two more times for the same original 12 sets, however, the 12 foils were always new. Both accuracy and reaction time were recorded and analyzed.

**Procedure**

**Screening**: After signing an IRB-approved informed consent form*, participants were screened for entry into the study and given a study ID number. The screening visit for SLE

*Note: Institutional Review Board (IRB) approval for this project was obtained by both the Feinstein Institute of Medical Research (FIMR) at Northwell Health and the Queens College, City University of New York. However, only the FIMR consent form was used to reduce duplication.
patients included a complete history and physical examination conducted by a rheumatologist. Identification of ACR criteria, date of diagnosis, other co-morbid illnesses, blood, and history of CNS disease was also collected and documented. Current disease activity and stability of symptoms was determined at that time.

Testing: Cognitive and behavioral testing were conducted within two weeks of the screening in order to ensure disease stability. For all participants, demographic information, including level of education, zip code, occupation, and ethnicity, was documented. The testing procedures began with the collection of demographic information, including self-report of cognitive dysfunction, and then the neuropsychological testing. Several of the neuropsychological measures were used across the two cohorts, and those measures were: the Rey Complex Figure, TMT, and Spatial Memory Test. Other measures were specific to Cohort A, which followed the ACR-recommended battery of neuropsychological tests and will not be reviewed in the current project. The procedures were taken from Watson (2014), and the descriptive and inferential statistics are also reported in Watson (2014). Behavioral testing from the Spatial Memory Test was administered during the delay from the Rey Complex Figure. For Cohort B, TMT and JLO were administered following the RCFT Delay. Finally, the Relational Learning Task was administered after the neuropsychological testing was completed, followed by the ANAM. An additional computer laboratory task assessing spatial navigation through a complex fictional city was administered upon the completion of the ANAM; however, this task was not part of the dissertation.

Anti-NMDAR Autoantibody: Blood serum was drawn by one of two research assistants to determine the presence of the anti-NMDAR Ab. The assays were performed in the Center for Autoimmune and Musculoskeletal Diseases laboratory at the Feinstein Institute for Medical Research using an ELISA with the DWEYS consensus sequence as the substrate. The antigen
was absorbed onto high binding, half-area 96 well plates (Costar #3690, Corning, NY) at 15 μg/ml in 0.1 M NaHCO₃ pH 8.6, and overnight at 4° C. The serum was tested at 1:100 dilution in 0.2% BSA/PBS at 37° C for 1 hour following 1-hour blocking with 1% BSA/PBS also at 37° C. The bound Abs were detected with AP-labeled goat anti-human-IgG (Southern Biotech, Birmingham, AL) followed by AP substrate (SIGMA, St. Louis, MO). Individuals involved with data acquisition remained blind to participant Ab status until after the data collection period had ended.
CHAPTER III

Results

Statistical Analysis: Descriptive statistics for demographic data were assessed. Independent sample student t-tests were used to compare age, education, disease duration (between DNRAb- and DNRAb+), and scores on the mood inventories. Chi-square was used to compare gender, handedness, race, family history of autoimmune disease, and self-reported history of cognitive dysfunction. Mixed ANOVAs were used to compare performance on the spatial and relational tests between or among groups (SLE patients versus HCs; DNRAb- patients versus DNRAb+ patients versus HCs). Bonferroni post hoc tests were used to compare significant differences. Correlation analyses and multiple regression analyses were performed to assess relationships among demographic and neuropsychological variables and performance on the spatial and relational working memory tasks.

Descriptive Characteristics: A comparison of the HCs and SLE groups (N=102) is presented in Table 6. Continuous variables were compared using a t-test. The two groups differed significantly from one another on age, education, BDI-II depression, and STAI trait anxiety. There was no significant difference between the groups on STAI state anxiety, \( t(93) = -1.17, p = .25 \). On average, the SLE participants were older than the HCs, \( t(100) = -2.33, p = .01, r = .23 \) (effect size), had higher levels of BDI-II depression, \( t(99) = -4.54, p < .01, r = .42 \), and STAI trait anxiety, \( t(94) = -3.58, p < .01, r = 0.01 \), and were less educated, \( t(98) = 3.04, p < .01, r = .29 \).

Categorical variables were compared using Chi-Square, unless frequencies were under 5 in a cell; in which case, Fisher’s exact test was implemented. Overall, there were no group differences between HCs and SLE participants on gender (\( p = .51 \), two-tailed Fisher’s exact
test, Cramer’s $V = .12$), handedness ($p = .29$, two-tailed Fisher’s exact test, Cramer’s $V = .26$), or race/ethnicity, $\chi^2 (1) = 3.12$, $p = .56$. There was a significant group difference in reported family history of autoimmune disease, $\chi^2 (1) = 5.26$, $p = .03$, as individuals diagnosed with SLE had a 2.80 times higher likelihood of reporting a family history of autoimmune disease than did HC. The SLE group were also more likely to report having experienced cognitive dysfunction, $\chi^2 (1) = 18.87$, $p < .01$, as the SLE group was 8.56 times more likely than the HC group to report a history of cognitive dysfunction.

Cohort B only included individuals who were between 18 to 55 years of age, whereas Cohort A included individuals above 18 years of age. Furthermore, Cohort B included an individual with a reported 24 years of education; therefore this individual was also excluded to determine whether the high level of education influenced the data. When the data were restricted to only include individuals with a known level of education that was less than 21 years and an age that was less than 56, six HCs (new $N = 36$) and 5 SLE participants (new $N = 55$) were eliminated from the study. However, the relationships among age, education, scores on the BDI-II and trait STAI, family history of autoimmune disease, and history of cognitive dysfunction remained significant at the $\alpha = .05$ level. Therefore, the full sample is included in the analyses below.

Descriptive analyses were also conducted to compare demographic data using HCs, DNRAb-, and DNRAb+ groups (total $n = 97$; see Table 7). Continuous variables were compared using a one-way ANOVA. Levene’s test was conducted to test whether the variances of the three groups were significantly different on the ANOVA measures. The only variable that failed the Levene’s test was BDI-II, $F(2, 94) = 6.71$, $p < 0.01$. Thus, for BDI-II, alone, Welch’s F-test will be conducted. The other variables had similar variances: age, $p = .43$; education, $p = .78$; disease duration, $p = 0.24$; STAI trait anxiety, $p = 0.15$; and STAI state anxiety, $p = 0.16$. 
There was a significant main effect of age, $F(2, 94) = 3.24, p = 0.04$, however, none of the post hoc analyses reached significance. There was a significant effect of education, $F(2, 93) = 4.77, p = 0.01$, and post hoc analyses indicated that HCs had a significantly higher level of education than did DNRAb+, $p = 0.01$. DNRAb- did not differ significantly from HCs, $p = 0.10$, or DNRAb+, $p = 0.97$. There was a significant effect of BDI-II depression using Welch’s $F(2, 42.04) = 12.80, p < 0.01$, with post hoc analyses demonstrating that HCs scored significantly lower than DNRAb-, $p < 0.01$, and DNRAb+, $p = 0.07$ (marginal). There was a significant effect of STAI trait anxiety, $F(2, 90) = 8.37, p < 0.01$, wherein HCs scored significantly lower than DNRAb-, $p < 0.01$, but not DNRAb+, $p = 0.76$. Disease duration was not significantly different between DNRAb- and DNRAb+, $F(1, 53) = 0.57, p = 0.45$. STAI state anxiety scores were not significantly different between groups $F(2, 88) = 0.71, p = 0.50$.

Categorical variables were compared using Chi-Square, unless frequencies were under 5 in a cell; in which case, the Fisher’s exact test was implemented. Overall, there were no group differences between HCs and the two SLE groups on gender ($p = .17$, two-tailed Fisher’s exact test, Cramer’s $V = .20$), handedness ($p = .51$, two-tailed Fisher’s exact test, Cramer’s $V = .26$), or race/ethnicity ($p = .12$, two-tailed Fisher’s exact test, Cramer’s $V = .26$). There were significant group differences in terms of reported family history of autoimmune disease, $\chi^2 (2) = 6.85, p = .03$. Overall, HC reported significantly lower family history of autoimmune disorder compared to the DNRAb- group, $p = 0.01$, whereas the DNRAb+ group did not significantly differ from either groups, $p$'s > 0.05. There was also a group difference in self-reported cognitive dysfunction, $\chi^2 (1) = 18.96, p < .01$. Compared to the HC group, both the DNRAb- and DNRAb-groups reported significantly higher histories of cognitive impairment, $p < 0.01$. However, the DNRAb groups did not differ from one another, $p = 0.47$. 
When the data were restricted to only include individuals with a known level of education that was less than 21 years and age less than 56 years, six HCs (new n = 36) and four DNRAb-participants (new n = 30), and no DNRAb+ participants (n = 21) were eliminated from the study. Another set of analyses was conducted and only restricted education, but not age (HCs n = 36, DNRAb- n = 34, DNRAb+ n = 21). However, in both sets of analyses, the relationships among HCs, DNRAb-, and DNRAb+ groups on age, education, scores on the BDI and TAI-Y, family history of autoimmune disease, and history of cognitive dysfunction remained the significant at the $\alpha = .05$ level. Therefore, the full sample was included in the analyses below.

**Aim 1:** To examine if there were differences between SLE and HC participants for spatial memory deficits (Aim 1a) and whether DNRAbs are uniquely associated with spatial memory deficits (Aim 1b).

To address this aim, a series of 2 x 2 x 2 mixed ANOVA analyses were conducted to compare performance between SLE and HC groups on the spatial memory task. Independent variables were the Difficulty Level (2 x 2 array = easy or 3 x 2 array = hard) and type of memory question posed (spatial or nonspatial), as a function of group. The dependent variable was the proportion of correct responses. Memory and difficulty served as within-subject factors and group served as a between-subjects factor. To analyze Aim 1b, a second mixed 3 x 2 x 2 mixed ANOVA was conducted to further explore the contribution of Ab status, Group (HC, DNRAb-, DNRAb+) x Item Difficulty (easy or hard) x Memory (spatial or nonspatial), with the only difference being the level of group analysis. Table 10 summarizes the means and SDs for the group performances on this task.

**Aim 1a. Spatial Memory Results for SLE and HCs Participants**

It was predicted that the SLE group would have reduced memory for the spatial items compared to the HCs, but both groups would have similar performance for the nonspatial items.
The 2 x 2 x 2 mixed ANOVA found significant main effects for Group, $F(1, 100) = 10.79, p < 0.01, \eta^2 = 0.97$, Item Difficulty, $F(1, 100) = 194.38, p < 0.01, \eta^2 = 0.66$, and Memory Type, $F(1, 100) = 63.06, p < 0.01, \eta^2 = 0.39$, as well as a significant interaction effect of Group and Item Difficulty, $F(1, 100) = 6.89, p = 0.01, \eta^2 = 0.06$ (Figure 1). These effects resulted in higher accuracy rates for the easy compared to hard matrix, nonspatial compared to spatial items, and the HCs compared to SLE group. As for the interaction, post hoc analyses revealed that the HCs as compared to the SLE had a higher proportion of correct responses on the easy array as compared to the hard array (all $p$s < .01; Figure 2). The remaining interactions were all found not to be significant: Group by Memory Type, $F(1, 100) = 1.09, p = 0.30, \eta^2 = 0.01$, Item Difficulty by Memory Type, $F(1, 100) = 0.05, p = 0.82, \eta^2 < 0.01$, and Group by Item Difficulty by Memory Type, $F(1, 100) = 0.20, p = 0.65, \eta^2 < 0.01$.

Chang et al. (2015) focused exclusively on the easy matrix of the Spatial Memory Task, given the low accuracy across all groups for the hard matrix. In order to replicate those results, a 2 (Group) x 2 (Memory Type) mixed ANOVA was conducted using only the easy items. The significant effect of Group, $F(1, 100) = 5.58, p = 0.02, \eta^2 = 0.05$, revealed that the HC group was more accurate than the SLE group. The significant effect of Memory Type, $F(1, 100) = 119.22, p < 0.01, \eta^2 = 0.54$, revealed that participants had more accurate responses on the nonspatial items as compared to the spatial items. Of note, there was a significant interaction between Group and Memory Type, $F(1, 100) = 5.87, p = 0.02, \eta^2 = 0.06$; and post hoc analyses revealed that the HCs as compared to the SLE patients had a higher proportion of correct responses on the spatial questions (all $p < .01$; Figure 3), however, the groups did not significantly differ on the nonspatial questions ($p = 0.54$; see Figure 3).

Thus, the HC group, overall, performed better than the SLE group in terms of the proportion of correct responses on the entire task. In general, all participants did better on the
easy compared to hard items and nonspatial compared to spatial items. When looking at the
task in its entirety, as predicted, there was no interaction between the HC and SLE groups and
Memory Type. However, when only analyzing the easy questions, the HC group was more
accurate for spatial items compared to the SLE group, but both groups had similar rates of
accuracy for the nonspatial items, thereby, replicating the results in Chang et al. (2015).

**Aim 1b. Spatial Memory Results for HC, DNRAb-, and DNRAb+ Participants**

It was predicted that the DNRAb+ group would have reduced memory for the spatial
items compared to the HC and DNRAb- groups but that all groups would have similar
performance for the nonspatial items. A 3 x 2 x 2 mixed ANOVA was conducted. There were
significant effects for Group, \(F(2, 94) = 6.18, p < 0.01, \eta^2_p = 0.12\), Item Difficulty, \(F(1, 94) = 201.80, p < 0.01, \eta^2_p = 0.68\), Memory Type, \(F(1, 94) = 69.11, p < 0.01, \eta^2_p = 0.42\), as well as a
significant interaction of Group by Item Difficulty, \(F(2, 94) = 3.13, p = 0.05, \eta^2_p = 0.06\), and a
three-way interaction among Group, Item Difficulty, and Memory Type, \(F(2, 94) = 4.65, p = 0.01, \eta^2_p = 0.09\) (see Figure 4). However, the interactions for Group by Memory Type, \(F(2, 94) = 0.87, p = 0.42, \eta^2_p = 0.02\), and Item Difficulty by Memory, \(F(1, 94) = 0.76, p = 0.39, \eta^2_p < 0.01\), were
not significant. Individuals were more accurate for the easy compared to hard matrix and
nonspatial compared to spatial items. The HC group was more accurate compared to the
DNRAb+ patients, \(p < 0.01\), but not the DNRAb- group, \(p = 0.10\), and the DNRAb groups had
similar accuracy levels, \(p = 0.44\).

The Group by Item Difficulty interaction revealed that on the easy array the HC group
had a significantly higher proportion of correct responses as compared to the DNRAb+ group, \(p = 0.04\); however, the DNRAb- group did not differ from either the HC, \(p = 0.57\), or DNRAb+
groups, \(p = 0.57\). As for the hard array, the HC group had a higher proportion of correct
responses as compared to the DNRAb+ group, \( p = 0.01 \) (Figure 4). Again, the DNRAb- group did not differ from either the HC group, \( p = 0.07 \), or DNRAb+ group, \( p = 0.71 \).

To understand the significant three-way interaction among Group, Item Difficulty, and Memory Type, two separate 3 x 2 mixed ANOVAs, for Group (HC, DNRAb-, and DNRAb+) and Memory type (nonspatial or spatial), were conducted, separating the ANOVAs based on Item Difficulty (Figure 5). On the easy array, again, individuals were more accurate for nonspatial compared to spatial items, \( F(1, 94) = 140.97, p < 0.01, \eta^2_p = 0.60 \). A significant effect of Group was observed, \( F(2, 94) = 3.22, p = 0.05, \eta^2_p = 0.06 \). As expected, the HC group was more accurate than the DNRAb+ group, \( p = 0.04 \). The DNRAb- group did not differ significantly from the HC group, \( p = 0.57 \), or the DNRAb+ group, \( p = 0.57 \). Importantly, there was a significant Group by Memory Type interaction effect, \( F(2, 94) = 5.66, p < 0.01, \eta^2_p = 0.11 \). For the nonspatial items, there were no group differences, all \( p \)'s \( \geq 1.00 \). However, for the spatial items, the HC group had a significantly higher proportion of correct responses as compared to the DNRAb+ group, \( p < 0.01 \). The DNRAb- group did not significantly differ from the HC group, \( p = 0.31 \) or the DNRAb+ group, \( p = 0.09 \).

For the hard array, the nonspatial questions were more accurately answered than spatial items, \( F(1, 94) = 84.58, p < 0.01, \eta^2_p = 0.47 \); and the main effect of Group, \( F(2, 94) = 5.82, p < 0.01, \eta^2_p = 0.11 \), revealed that the HC group was more accurate than the DNRAb+ group, \( p < 0.01 \). The DNRAb- group did not significantly differ from the HC, \( p = 0.07 \) or DNRAb+ groups, \( p = 0.71 \). The Group by Memory Type interaction was not significant \( F(2, 94) = 2.46, p = 0.09, \eta^2_p = 0.05 \).

Thus, the HC group, overall, performed better than the DNRAb+ group in terms of the proportion of correct responses on the task and, in particular, the spatial items for the easy matrix. Overall, participants were more accurate on the easy questions as compared to the hard
questions, and were better at answering correctly the nonspatial items compared to spatial items. Finally, there were some notable significant interactions among the variables. For instance, on the easier items, the HCs as compared to the DNRAb+ group scored significantly higher on spatial questions as compared to nonspatial questions. However, on the hard array the Group by Memory Type interaction was not significant. This supports the previous study by Chang and colleagues (2015) that suggested the hard array was, perhaps, too difficult to differentiate the groups.

**Aim 2: To examine if there were associations between relational learning deficits and SLE (Aim 2a) and whether DNRAbs specifically predict relational learning deficits (Aim 2b). An exploratory analysis assessed whether the group (Aim 2c) and DNRAb status (Aim 2d) influenced the type of errors participants made.**

To address aims 2a and 2b, two repeated-measure ANOVA analyses compared the proportion of correct responses (accuracy) on the Relational Learning Task as a function of Group (aim 2a. 3 x 2 ANOVA for HC vs. SLE or aim 2b. 3 x 3 ANOVA for HC vs. DNRAb- vs. DNRAb+) and Block (3 Blocks). Table 11 summarizes the means and SDs for the performances on this task.

Aims 2c and 2d. The aims of 2c and 2d were included to explore whether the type of errors on the relational task were specific to failure to encode the shape or location of target stimuli. Such errors may be informative to further diagnose any potential failure of learning relational information. To analyze aims 2c and 2d, an additional mixed ANOVA was conducted, as above, and added a third level to the analyses to evaluate the type of errors that were made on the Relational Learning Task (shape, location, or spoiler). Shape indicated that one of the shapes on the monitor changed. Location indicated a single object was moved in the array.
spoiler was the introduction of a novel shape. Bonferroni corrections were included to decrease potential Type I error.

Aim 2a. Relational Learning as a Function of Group Status: Using HC and SLE and Block

It was predicted that the SLE group would have a reduced performance (proportion of accurate responses) for the task as compared to the HCs. A 2 x 3 repeated-measures ANOVA was conducted to compare the performance between HCs and SLE as a function of Block. As predicted, the HC group had a significantly higher accuracy rate than SLE participants, \( F(1, 27) = 12.70, p < 0.01, \eta_p^2 = 0.32 \). There was a significant main effect of Block on the proportion of correct responses, \( F(2, 54) = 20.52, p < 0.01, \eta_p^2 = 0.43 \), such that the first Block was associated with the lowest level of accuracy, the second Block associated with a higher level of accuracy, and the third Block associated with the highest level of accuracy, all \( ps < 0.01 \). The Group by Block interaction was not significant, \( F(2, 54) = 0.06, p = 0.95, \eta_p^2 < 0.01 \). In summary, the HCs performed significantly better than the SLE group.

Aim 2b. Relational Learning as a Function of Group Status: Using HC, DNRAb- and DNRAb+ Groups and Block

It was predicted that the DNRAb+ group would exhibit reduced performance on the task as compared to the HC and DNRAb- groups. A 3 (Group) x 3 (Block) repeated-measures ANOVA was conducted. As predicted, there was a significant main effect of Group, \( F(2, 26) = 6.13, p = 0.01, \eta_p^2 = 0.32 \). The HC group had a higher proportion of correct responses compared to both DNRAb-, \( p = 0.01 \), and DNRAb+, \( p = 0.02 \), groups. However, the DNRAb- and DNRAb+ groups did not significantly differ from one another, \( p = 1.00 \). A main effect of Block was also observed, \( F(2, 52) = 23.20, p < 0.01, \eta_p^2 = 0.47 \), and, specifically, individuals got progressively better with each passing Block, all \( ps < 0.01 \). The interaction effect of Group by
Block was not significant, $F(4, 52) = 0.51, p = 0.73, \eta_p^2 = 0.04$. In sum, although the SLE group performed worse than the HC group, the expected finding of the DNRAb+ performing worse on the task compared to the DNRAb- group did not emerge.

**Aim 2c. The aim was to examine if Group (HC or SLE) differences emerged for the type of errors (Location, Shape, or Spoiler Items) made as a function of Block.**

As an exploratory analysis, the possibility of an association between SLE and a particular type of error was examined. The $2 \times 3 \times 3$ mixed ANOVA was conducted to compare the relationship between Group (HC and SLE), Block, and Recognition Stimuli (relation, shape, or spoiler; see Figure 6). A review of Mauchly’s test indicated that the assumption of sphericity had been violated for the main effect of Block, $\chi^2(2) = 10.07, p < 0.01$, and the interaction of Block and Recognition Stimuli $\chi^2(9) = 30.09, p < 0.01$. Therefore, degrees of freedom were corrected using the Greenhouse-Geisser estimates of sphericity ($\varepsilon = 0.76$ for the main effect of Block and $\varepsilon = 0.91$ for the interaction effect of Block and Recognition Stimuli). A review of Mauchly’s test indicated that the assumption of sphericity was met for Recognition Stimuli, $\chi^2(2) = 2.75, p = 0.25$.

A significant main effect of Block was observed, $F(1.51, 40.88) = 4.83, p = 0.02, \eta_p^2 = 0.15$; post hoc analyses revealed no differences for errors between Blocks 1 and 2, $p = 1.00$, but individuals made fewer errors on Block 3 compared to both Block 1, $p < 0.01$, and Block 2, $p < 0.01$. There was a significant main effect of Recognition Stimuli, $F(2, 54) = 36.84, p < 0.01, \eta_p^2 = 0.58$, and fewer errors were made responding to spoiler items compared to relational and shape items, $ps < 0.01$, whereas no performance difference emerged between relational and shape items, $p = 1.00$. The main effect of Group was not significant, $F(1, 27) = 1.33, p = 0.26, \eta_p^2 = 0.05$. All the interactions were found to be not significant; Group by Block, $F(1.51, 40.88) = 0.22, p = 0.74, \eta_p^2 < 0.01$; Group by Recognition Stimuli, $F(1.82, 49.08) = 0.71, p = 0.48, \eta_p^2 = \ldots$
0.03; Block by Recognition Stimuli, $F(2.54, 58.54) = 2.06, p = 0.12, \eta_p^2 = 0.07$; and Group by Block by Recognition Stimuli, $F(2.54, 68.54) = 0.18, p = 0.88, \eta_p^2 < 0.01$. In summary, the individuals were less likely to make errors on spoiler items and made the least amount of errors on the last Block.

**Aim 2d. Error Responses as a Function of Group (HC, DNRAb-, or DNRAb+), Block, and Recognition Stimuli**

The 3 x 3 x 3 mixed ANOVA was conducted to compare the relationship between Group (HC, DNRAb-, and DNRAb+), Block, and Recognition Stimuli (see Figure 7). A review of Mauchly’s test indicated that the assumption of sphericity had been violated for the main effect of Block, $\chi^2(2) = 11.36, p < 0.01$, and the interaction of Block and Recognition Stimuli $\chi^2(9) = 29.54, p < 0.01$. Therefore, degrees of freedom were corrected using the Greenhouse-Geisser estimates of sphericity ($\epsilon = 0.73$ for the main effect of Block and $\epsilon = 0.75$ for the interaction effect of Block and Recognition Stimuli). The assumption of sphericity was met for the main effect of Recognition Stimuli, $\chi^2(2) = 2.40, p = 0.30$.

There was a significant main effect of Block, $F(1.46, 38.09) = 5.59, p = 0.01, \eta_p^2 = 0.18$, with individuals making fewer errors on Block 3 compared to the other two Blocks, $ps < 0.05$, whereas Blocks 1 and 2 had similar proportion of errors, $p = 0.52$. There was a significant main effect of Recognition Stimuli Type, $F(1.83, 47.64) = 46.47, p < 0.01, \eta_p^2 = 0.64$, such that fewer errors were made for the spoiler items compared to the relational and shape items, $ps < 0.01$. However, performances on relational and shape items were not significantly different from each other ($p = 1.00$). The main effect of Group was not significant, $F(2, 26) = 0.81, p = 0.46, \eta_p^2 = 0.06$. Again, all the interactions failed to reach a level of significance: Group by Block, $F(2.93, 38.09) = 0.43, p = 0.72, \eta_p^2 = 0.03$; Group by Recognition Stimuli, $F(3.66, 47.64) = 0.76, p = 0.54, \eta_p^2 = 0.06$; Block by Recognition Stimuli, $F(2.50, 65.02) = 2.02, p = 0.13, \eta_p^2 = 0.07$; and
Group by Block by Recognition Stimuli, $F(5.00, 65.02) = 0.31, p = 0.90, \eta^2_p = 0.02$. The pattern of findings was identical to that as in aim 2c, and importantly, SLE status was not related to performance for the various types of errors.

**Aim 3:** To determine whether other factors were related to performance on the performance for the spatial memory task (Aims 3a-c) and relational learning task (Aims 3d-f). The factors that were considered included background demographics (age, education), SLE disease characteristics (i.e., disease duration), affective functioning (such as scores on depression and anxiety inventories), and neuropsychological assessments (including the paper-and-pencil tasks and the ANAM).

Pearson correlations were conducted, breaking into 3 separate analyses: demographic factors (age, education, and disease duration), affective functioning, and neuropsychological measures for each of the two learning and memory tasks. All correlations were conducted using 2-tailed significance values. For a discussion of how the different variables within each of these domains were related to one another, please see Appendix A. Following the correlations for each of the two learning and memory tasks, any significant correlations were included in multiple regression analyses.

**Aim 3a.** The first goal was to assess if any relationship exists between demographic factors and accuracy on the spatial memory task for the spatial and nonspatial items (see Table 12).

**Correlations.** Nonspatial Items: Performance on the easy nonspatial questions was significantly correlated with years of education, $r = 0.26, p < 0.01$, such that individuals with higher education performed better, reflecting a positive correlation. Individuals with longer disease duration was associated with worse performance for the hard nonspatial questions, $r = -0.30, p = 0.02$, reflecting a negative correlation. Age, $r = -0.19, p = 0.053$, and education, $r =$
0.20, \( p = 0.0501 \), just missed the cut-off for a significant correlation with performance on the hard nonspatial questions (\( p \)'s > 0.05), such that older individuals and individuals with less education performed worse.

Additional correlations were conducted to analyze the relationships between these variables and the Spatial Memory task, irrespective of item difficulty. Higher accuracy on the nonspatial items were significantly correlated with lower age, \( r = -0.26, p = 0.01 \); higher education, \( r = 0.30, p < 0.01 \); and shorter disease duration, \( r = -0.26, p = 0.05 \).

**Spatial Items**: Older age was significantly correlated with performance on easy spatial items, \( r = -0.27, p < 0.01 \), and hard spatial items, \( r = -0.36 \), with older individuals performing worse on both the easy and hard spatial items. Disease duration was moderately negatively correlated with accuracy on the easy spatial items, \( r = -0.34, p < 0.01 \).

Additional correlations were conducted to analyze the relationships between these variables and the Spatial Memory task, irrespective of item difficulty. Greater accuracy on the spatial items were significantly correlated with lower age, \( r = -0.36, p < 0.01 \); higher education, \( r = 0.43, p < 0.01 \); and shorter disease duration, \( r = -0.26, p = 0.04 \).

**Linear Regression.** A linear regression was conducted to further evaluate the relationships between these demographic variables and whether they predict performance on the Spatial Memory task. The dependent variables for the regressions were (1) proportion of accurate nonspatial responses and (2) proportion of accurate spatial responses, both irrespective of item difficulty. Significant correlations were the independent variables, in addition to group (HC or SLE; or Ab status).

**Nonspatial Items**: Since age, education, and disease duration were each correlated with performance on the nonspatial items, they, therefore, were examined in the linear regression.
When those three factors were entered into a single regression model, in addition to Group as a variable in the second Block (HC or SLE), the model was significant, $F(3, 55) = 3.85$, $p = 0.01$. Education was a significant predictor, $p = 0.02$, whereas age, $p = 0.62$ and disease duration were not, $p = 0.08$. These factors accounted for 18% of the variance. However, in this model, disease duration only applied to SLE populations and restricted the sample to only include the SLE group in the regression. When the disease duration variable was eliminated in order to analyze the entire sample, the model was significant, $F(3, 96) = 6.18$, $p < 0.01$. In this model, both age, $p = 0.01$, and education $p = 0.01$ were significant predictors, whereas group, $p = 0.52$, was not. These factors accounted for 16% of the variance. When Ab status was included in the model (i.e., DNRAb- or DNRAb+), the model was significant, $F(5, 53) = 2.46$, $p = 0.04$ and accounted for 18% of the variance. Education was a significant predictor, $p = 0.02$, whereas age, $p = 0.68$, disease duration, $p = 0.10$, DNRAb- status, $p = 0.79$, and DNRAb+ status, $p = 0.83$, were not. Again, when disease duration was not included in order to increase the sample in these analyses, the model was significant $F(4, 95) = 5.05$, $p < 0.01$. In this model, both age, $p = 0.01$, and education $p = 0.01$ were significant predictors, whereas DNRAb- status, $p = 0.94$ and DNRAb+ status, $p = 0.23$, were not. These factors accounted for 18% of the variance.

In sum, age and education were the most consistent predictors of performance on nonspatial items for the entire sample. When the groups were limited to only SLE participants, only education was a significant predictor of performance. Disease duration and group were not significant predictors of performance.

Spatial Items: Age, education, and disease duration were each correlated with spatial items and, thus, used in a linear regression to examine whether the predicted performance on the task. When all three variables were included in the regression model, in addition, with group as a second Block (HC or SLE), the model significantly predicted performance on the spatial
questions, $F(3, 53) = 7.13, p < 0.01$. Age, $p = 0.05$, and education, $p < 0.01$, were significant predictors, $p = 0.02$, whereas disease duration was not, $p = 0.31$. These factors, then, accounted for 28% of the variance. As before, the same analyses were conducted after the disease duration variable was recoded, and the model was again significant, $F(4, 95) = 12.22, p < 0.01$ and accounted for 34% of the variance. Age, $p = 0.02$ and education, $p < 0.01$, were significant predictors, whereas disease duration, $p = 0.28$ and group, $p = 0.37$, were not. When disease duration was not included in the model, the model was significant, $F(3, 96) = 15.88, p < 0.01$. In this model, age, $p = 0.01$, education $p = 0.01$, and group, $p = 0.05$, were significant predictors. The model accounted for 33% of the variance.

When Ab status was included in the regression, the model significantly predicted performance on the spatial questions, $F(5, 53) = 4.31, p < 0.01$. Age, $p = 0.04$, and education, $p < 0.01$, were significant predictors, whereas disease duration $p = 0.33$, DNRAb- status, $p = 0.59$, and DNRAb+ status, $p = 0.87$, were not. The model accounted for 29% of the variance. When disease duration was excluded from the model, it continued to remain significant, $F(4,95) = 11.70, p < 0.01$. Again, only age and education were significant predictors ($p < 0.01$), and DNRAb- status was not ($p = 0.26$). DNRAb+ status was not a significant predictor either; however, it was trending towards significance, $p = 0.06$. The model accounted for 33% of the variance.

In summary, age and education continued to present as strong predictors of spatial memory items. Disease duration was not a significant predictor of performance. Group was an inconsistent predictor of performance. While group did not typically predict performance on spatial memory items, on analyses where disease duration was removed, HC or SLE groups became predictive of performance, and DNRAb+ status was beginning to trend towards significance but did not significantly predict performance.
Aim 3b. The second goal was to assess if any relationship existed between affective factors and accuracy on the spatial memory task for the spatial and nonspatial items (see Tables 13-15). Depression was measured by the BDI. State and trait anxiety were measured by the STA-Y and TAI-Y, respectively (Table 13). Additional state affective ratings were obtained from Cohort B’s data from the ANAM. Mood measures extracted from the ANAM included self-reports of sleepiness, fatigue, and restlessness (Table 14), and anger, anxiety, depression, happiness, and vigor (Table 15).

**Correlations.** *Nonspatial Items:* Significant correlations were found with only Cohort B, as only factors specific to the ANAM were correlated with performance. Specifically, sleepiness was strongly and positively correlated with performance on the hard nonspatial questions, $r = 0.54, p < 0.01$, such that higher levels of sleepiness were associated with better performance on the hard nonspatial questions. Higher scores on the vigor scale were moderately and negatively associated with performance on the hard nonspatial questions, $r = -0.37, p = 0.04$. Additional correlations were conducted to analyze the relationships between these variables and the Spatial Memory task, irrespective of item difficulty. Greater accuracy on the nonspatial items was not significantly correlated with any of the affective measures, $ps > 0.05$.

*Spatial Items:* There were significant negative correlations between accuracy on the hard spatial items and higher scores on the depression inventory, $r = -0.21, p = 0.03$, such that as depression inventory raw scores increased accuracy decreased. Accuracy on the easy spatial items were negatively correlated with higher scores on the STA-Y, $r = -0.21, p = 0.04$, TAI-Y, $r = -0.24, p = 0.02$, and the ANAM depression scale, $r = -0.43, p = 0.02$, in such a way that higher raw scores on any of these scales were related to poor accuracy on the easy spatial items. Additional correlations were conducted to analyze the relationships between these variables and the Spatial Memory task, irrespective of item difficulty. Greater accuracy on the
spatial items were significantly correlated with lower scores on the depression inventory, $r = -0.22$, $p = 0.03$, and on a measure of trait anxiety, $r = -0.23$, $p = 0.02$, both reflecting negative correlations.

In summary, sleepiness and vigor were associated with performance on hard nonspatial items for Cohort B, whereas none of the affective measures correlated with performance on easy nonspatial items. However, when examining the data irrespective of item difficulty, none of the affective measures correlated with overall performance on nonspatial items. Conversely, performance on easy spatial items was significantly correlated with scores on the STA-Y, TAI-Y, and ANAM depression scale whereas hard spatial items were correlated with scores on the BDI. Yet, when performance on spatial items was evaluated irrespective of item difficulty, only the BDI and TAI-Y significantly correlated with overall spatial item performance.

**Linear Regression**

*Nonspatial Items:* None of the affective scores were significantly correlated with the nonspatial items (irrespective of difficulty). Therefore, a regression was not conducted with these items.

*Spatial Items:* The BDI and TAI-Y were the only affective items that remained significantly correlated with performance on spatial items, irrespective of item difficulty. When both scores from the BDI and TAI-Y were included in the regression model, in addition to including group as a second Block (HC or SLE), the model significantly predicted performance on the spatial questions, $F(3, 92) = 3.85$, $p = 0.01$. Group, $p = 0.02$, was a significant predictor, whereas scores on the BDI, $p = 0.95$, and TAI-Y, $p = 0.35$, were not. This model accounted for 11% of the variance. Interestingly when Ab status was included, the model, again, significantly predicted performance $F(4, 91) = 4.02$, $p < 0.01$; however, only DNRAb+ status significantly
predicted performance, \( p < 0.01 \), whereas scores on the BDI, \( p = 0.80 \), TAI-Y, \( p = 0.24 \), and DNRAb- status, \( p = 0.15 \), did not. The entire model accounted for approximately 15% of the variance in spatial item scores.

In summary, none of the affective measures significant predictors of spatial memory performance in the regression model.

Aim 3c. The third goal was to determine if any relationship exists between scores on neuropsychological and cognitive measures and accuracy on the spatial memory task for the spatial and nonspatial items (see Tables 16-19). Raw scores were used for the JLO (number of items correct), TMT-A (seconds), TMT-B (seconds), RCFT copy (Meyer’s scoring method), and RCFT delay (Meyer’s scoring method) performance (Table 16). The raw scores were chosen to remain consistent between all neuropsychological measures due to the fact that a one-way ANOVA between these measures (Tables 8 and 9) found significant differences between group and performance on the TMT-A, TMT-B, and RCFT copy. Alternatively, using z-scores yielded significant differences between only TMT-B and RCFT copy.

The ANAM output created multiple values, which varied according to task. The output variable frequently included a measure of accuracy and RT, although some tasks also provided a value for hits, omission, and/or commissions. For the reader’s ease, only ANAM “throughput” scores (the efficiency measure that combines accuracy and RT) will be used for correlations on the spatial and nonspatial measures. The only exception to this is the Go/No-Go task, which did not have a “throughput” score, but rather included a measure of signal detection (D’).

Furthermore the correlation tables are broken down for ANAM tasks into visuospatial tasks (Table 17), processing speed (Table 18), and the Go/No-Go inhibition task (Table 19).
Correlations. Nonspatial Items: A longer amount of time to complete the TMT-A task (in seconds) was significantly and negatively correlated with accuracy on the hard nonspatial questions, $r = -0.36$, $p < 0.01$. A longer amount of time to complete the TMT-B task (in seconds) was significantly and negatively correlated with poorer performance on both the easy nonspatial memory questions, $r = -0.29$, $p < 0.01$ and the hard nonspatial questions, $r = -0.28$, $p < 0.01$. Higher raw scores on the RCFT copy task were significantly and positively correlated with greater accuracy on the easy nonspatial memory questions, $r = 0.22$, $p < 0.03$, and hard nonspatial questions, $r = 0.33$, $p < 0.01$. Higher raw scores on the RCFT delay task was significantly correlated with higher proportion of accurate responses on the easy nonspatial memory questions, $r = 0.26$, $p = 0.02$, reflecting a positive correlation. Greater efficiency on the Matching Grids task was significantly correlated with higher accuracy on the easy nonspatial questions, $r = 0.38$, $p = 0.04$, reflecting a positive correlation.

Additional correlations were conducted to analyze the relationships between these variables and the Spatial Memory task, irrespective of item difficulty. Higher accuracy on the nonspatial items was significantly correlated with faster TMT-A completion, $r = -0.36$, $p < 0.01$; faster TMT-B completion, $r = -0.39$, $p < 0.01$; higher raw score on the RCFT copy, $r = 0.39$, $p < 0.01$; higher accuracy on the RCFT long delay, $r = 0.30$, $p < 0.01$; and greater efficiency on the Matching Grids task, $r = 0.40$, $p = 0.03$.

In summary, easy nonspatial item performance was significantly correlated with TMT-B, RCFT copy, RCFT delay, and ANAM Matching Grids performances. Hard nonspatial items were correlated with TMT-A, TMT-B, and RCFT copy performances. However, when the nonspatial were reviewed irrespective of item difficulty, the TMT-A, TMT-B, RCFT copy, RCFT delay, and Matching Grid tasks were all correlated with nonspatial item performance.
Spatial Items: Higher accuracy on the JLO was significantly positively correlated with accuracy on the easy, $r = 0.39$, $p = 0.03$; and hard, $r = 0.56$, $p < 0.01$, spatial memory items. The longer it took to complete the TMT-A task was significantly and negatively correlated with poorer performance on the easy, $r = -0.27$, $p = 0.01$, and hard, $r = -0.42$, $p < 0.01$, spatial items. Completion time on the TMT-B task was significantly and negatively correlated with accuracy on both the easy, $r = -0.42$, $p < 0.01$, and hard, $r = -0.49$, $p < 0.01$, spatial items, such that longer completion times were associated with lower accuracy. The raw score on the RCFT copy task was significantly correlated with accuracy on the easy, $r = 0.29$, $p < 0.01$, and hard, $r = 0.40$, $p < 0.01$, spatial items, such that as one increased so did the other. Similarly, the raw on the RCFT delay task was significantly correlated with accuracy on easy, $r = 0.25$, $p < 0.02$, and hard, $r = 0.36$, $p < 0.01$, spatial items, such that as one increased, so did the other. Greater efficiency on the Simple RT task was significantly correlated with greater accuracy on the easy spatial questions, $r = 0.38$, $p = 0.04$. Greater efficiency on the Matching Grids task was significantly correlated with accuracy on the hard spatial questions, $r = 0.49$, $p = 0.01$, reflecting a positive correlation. Greater efficiency on the Match to Sample task was significantly and positively correlated with accuracy on the easy, $r = 0.52$, $p < 0.01$, and hard, $r = 0.45$, $p = 0.01$, spatial items.

Additional correlations were conducted to analyze the relationships between these variables and the Spatial Memory task, irrespective of item difficulty. Greater accuracy on the spatial items was significantly correlated with higher JLO accuracy, $r = 0.54$, $p = 0.02$; faster TMT-A completion, $r = -0.39$, $p < 0.01$; faster TMT-B completion, $r = -0.52$, $p < 0.01$; higher accuracy on the RCFT copy, $r = 0.39$, $p < 0.01$; higher accuracy on the RCFT delay, $r = 0.34$, $p < 0.01$; greater efficiency on the Matching Grids task, $r = 0.43$, $p = 0.02$; and greater efficiency on the Match to Sample task, $r = 0.56$, $p < 0.01$. 
In summary, easy spatial items were significantly correlated with JLO, TMT-A, TMT-B, RCFT copy, RCFT delay, Simple RT, and Match to Sample performances. In addition to these same measures, hard spatial items were also correlated with performance on the Matching Grids task. When analyzing spatial items irrespective of difficulty, all of these same measures were significantly correlated with overall performance on spatial items.

**Linear Regression. Nonspatial Items:** Performance on the nonspatial items was correlated with TMT-A (sec), TMT-B (sec), RCFT copy (raw), RCFT long delay (raw), and the Matching Grids (throughput) task. It is important to note that the ANAM was only administered to Cohort B. For the multiple regression, two analyses will be conducted (one that includes the Matching Grids task and one that does not, in order to analyze a larger sample). When all of the significantly correlated neuropsychological variables were included in the regression model, in addition to group as a variable in the second Block (HC or SLE), the model did not significantly predict performance on the nonspatial questions, $F(6, 23) = 2.41, p = 0.06$, and all individual variables did not significantly predict performance ($p > 0.10$). When Ab status was included in the analyses, the model trended towards significance but still was not significant, $F(7, 22) = 1.97, p = 0.11$, and all individual variables did not significantly predict performance ($p > 0.10$).

Interestingly, when the Matching Grids task was not included in the regression model (in order to increase the sample size of this analysis and include both Cohorts A and B), the model was significant, $F(5, 83) = 5.08, p < 0.01$, and accounted for 23% of the variance. However, a review of the individual measures (TMT-A, TMT-B, RCFT copy, RCFT delay) and group revealed that none predicted performance ($p > 0.10$). When Ab status was included, the model was significant, $F(6, 82) = 4.62, p < 0.01$. Although, a review of the neuropsychological measures found that they did not significantly predict performance on the nonspatial items ($p > 0.10$).
In summary, neuropsychological measures did not significantly predict the variance on nonspatial items. When analyzing only Cohort B, although the model was significant, there were no significant predictors. When Ab subgroups were included in the analyses, there were no significant predictors of nonspatial item performance. When both Cohorts A and B were included (by eliminating the ANAM Matching Grids variable from the model), none of the models significantly predicted performance on nonspatial items. When Ab status was included in the model, none of the neuropsychological measures significantly predicted performance on nonspatial items.

Spatial Items: Greater accuracy on the spatial items was significantly correlated with the JLO, TMT-A, TMT-B, RCFT copy, RCFT long delay, Matching Grids (throughput), and Match to Sample (throughput) tasks. It is important to note that both the JLO and the ANAM were only administered to Cohort B. Two multiple linear regressions were conducted; the first that included the JLO and two ANAM tasks, the second excluded the JLO and ANAM tasks in order to analyze a larger sample. When all of the variables were included in the regression model, in addition to group as a variable in the second Block (HC or SLE), the model significantly predicted performance on the spatial items $F(8, 21) = 2.43, p = 0.05$, and the model accounted for 48% of the variance. However, none of the variables significantly predicted performance on the spatial items ($ps > 0.10$). When Ab status was included in the analyses, the entire model was not significant, $R^2 = 0.50, F(9, 20) = 2.32, p = 0.06$.

Interestingly, when the JLO and ANAM tasks were excluded from the regression (only TMT-A, TMT-B, RCFT copy, and RCFT delay were included in order to analyze both Cohorts A and B), the resulting model significantly predicted performance on the spatial items, $F(5, 83) = 8.17, p < 0.01$. TMT-B, $p = 0.02$, was a significant predictor, whereas scores on the TMT-A, $p = 0.17$, RCFT copy, $p = 0.75$, RCFT delay, $p = 0.09$, and group, $p = 0.24$, were not. This model
accounted for 33% of the variance. When Ab status was included, the model significantly predicted performance on the spatial items, $F(6, 82) = 7.01, p < 0.01$. TMT-B, $p = 0.01$, was a significantly predictor of performance, whereas TMT-A, $p = 0.30$, RCFT copy, $p = 0.74$, RCFT delay, $p = 0.07$, DNRAb- status, $p = 0.98$, and DNRAb+ status, $p = 0.16$, were not. The entire model accounted for approximately 34% of the variation in spatial item scores.

In summary, when only analyzing Cohort B, the regression models were unable to identify specific predictors to account for the variance on the performance on spatial items. However, when both the ANAM and JLO tasks were excluded from the analyses in order to review performance for both Cohorts, the TMT-B task emerged as a significant predictor for performance on spatial items. SLE or Ab status did not affect the model.

Aim 3d. The first goal was to assess if any relationship exists between demographic factors (age, education, and disease duration) and proportion of accurate responses on the relational learning task by examining: (1) overall accuracy on the whole task, (2) overall learning slope (measured by the maximum accuracy of Blocks 1 or 2 subtracted from the accuracy of Block 3), (3) Block 3, and (4) the three types of recognition stimuli, irrespective of Block (see Tables 20-23). Correlational tables are broken down by Block 1 (Table 20), Block 2 (Table 21), Block 3 (Table 22), and the entire task (Table 23).

**Correlations.** (1) **Whole Task:** Higher education was moderately correlated with a higher proportion of accurate responses on the task as a whole, $r = 0.57, p < 0.01$. (2) **Learning Slope:** The overall learning slope was not significantly correlated with any demographic factors. (3) **Block 3:** Higher education was moderately correlated with a higher proportion of accurate responses in Block 3, $r = 0.47, p = 0.01$. (4) **Recognition Stimuli:** Higher education was moderately correlated with fewer errors on relational items, $r = 0.40, p = 0.03$. **Shape items and Spoilers** were not significantly correlated with any demographic factors.
**Linear Regressions.** Linear regressions were conducted to further evaluate the relationships between these demographic variables and whether they predict performance on the Relational Learning task. The dependent variables for the regressions were (1) proportion of accurate responses on the task as a whole and (2) proportion of accurate responses on Block 3, (3) responses on the Recognition Stimuli (irrespective of Block). Significant correlations were the independent variables, in addition to group (HC or SLE; or Ab status).

Whole Task: Performance on the Relational Learning Task was significantly correlated with higher education. When education and group were included in the regression, the model significantly predicted performance on the relational learning task \( F(2, 26) = 9.85, p < 0.01 \). Both education, \( p = 0.03 \), and group (HC or SLE), \( p = 0.04 \), predicted performance. The model accounted for 43% of the variance. When Ab status was entered into the regression, the model continued to significantly predict performance, \( F(3, 25) = 6.31, p < 0.01 \). However, only education, \( p = 0.04 \), significantly predicted performance, whereas both DNRAb- status, \( p = 0.06 \), and DNRAb+ status, \( p = 0.07 \), predicted performance at a marginal level.

Block 3: Performance on Block 3 was also significantly correlated with higher education. A regression that included education and group (HC or SLE) significantly predicted performance on the third Block of the Relational Learning Task, \( F(2, 26) = 5.49, p = 0.01 \). However, neither education, \( p = 0.12 \), nor group, \( p = 0.10 \), predicted performance. The model accounted for 30% of the variance. When Ab status was included in the model, the model significantly predicted performance on Block 3, \( F(3, 25) = 3.79, p = 0.02 \). However, neither education, \( p = 0.13 \), DNRAb- status, \( p = 0.08 \), nor DNRAb+ status, \( p = 0.23 \) emerged as significant predictors of task performance. Although, DNRAb- status was at a trend level suggesting it was the strongest predictor. The model accounted for 31% of the variance.
Recognition Stimuli: Only performance on the relational items was significantly correlated with high education. The shape and spoiler items were not correlated with age, education, or disease duration. When education and group were included in the regression, the model did not significantly predict errors for the relational recognition items, $F(2, 26) = 2.46, p = 0.10$ (the model accounted for 16% of the variance), even when Ab status was included in the analyses, $F(3, 25) = 1.93, p = 0.15$.

Relational Learning Task Regression Summary: Overall, education and group (HC or SLE) emerged as significant predictors of performance on the Relational Learning Task. However, Ab status was not a significant predictor. Further, when only looking at the third Block of the learning task, group only served as a predictor of performance when education was eliminated from the model. Conversely, on the relational recognition items, none of these variables emerged as significant predictors.

Aim 3e. The second goal was to assess if any relationship exists between affective factors and proportion of accurate responses on the relational learning task on (1) the task as a whole, (2) the learning slope, (3) Block 3, and (4) Recognition Stimuli (see Tables 24-35). Correlation tables among the BDI, STA-Y, and TAI-Y are separated between Block 1 (Table 24), Block 2 (Table 25), Block 3 (Table 26), and the entire task (Table 27). Correlation tables between ANAM sleep scores are separated between Block 1 (Table 28), Block 2 (Table 29), Block 3 (Table 30), and the entire task (Table 31). Correlation tables between ANAM mood scores are separated between Block 1 (Table 32), Block 2 (Table 33), Block 3 (Table 34), and the entire task (Table 35).

Correlations. (1) Whole Task: Higher scores for depression (BDI), $r = -0.41, p = 0.03$, trait anxiety (TAI-Y), $r = -0.41, p = 0.03$, and current level of sleepiness (ANAM Sleep scale), $r = -0.50, p = 0.02$, were negatively correlated with lower proportion of accurate responses overall.
(2) Learning Slope: Higher scores on the BDI were correlated with lower learning scores, $r = -0.39$, $p = 0.34$, reflecting a negative correlation. (3) Block 3: Higher scores on the BDI, $r = -0.48$, $p = 0.01$, and Restlessness Scale, $r = -0.39$, $p = 0.04$, were moderately but negatively correlated with a lower proportion of accurate responses in Block 3. (4) Relational Items: Higher scores on the BDI were correlated with more errors on relational items, $r = -0.41$, $p = 0.03$.

*Shape items and spoilers* were not significantly correlated with any neuropsychological or cognitive scores.

Linear Regression. Whole Task: Performance on the relational learning task was significantly correlated with scores on the BDI, TAI-Y, and ANAM Sleep Scale. The regression model significantly predicted performance on the Relational Learning Task, $F(4,18) = 4.02$, $p = 0.02$. However, group, $p = 0.06$, and the ANAM Sleep Scale, $p = 0.06$, only trended towards significantly predicted performance and BDI, $p = 0.84$, and TAI-Y, $p = 0.13$, scores did not predict performance on the Relational Learning Task. The model accounted for 47% of the variance.

When Ab status was added to the regression, the model was significant, $F(5, 17) = 3.17$, $p = 0.03$. However, the ANAM Sleep scale and DNRAb- status were just above the significance level at $p = 0.053$ and $p = 0.055$, respectively, regarding their tendency to predict performance on the Relational Learning Task. The BDI, $p = 0.73$, TAI-Y, $p = 0.14$, and DNRAb+ status, $p = 0.12$, did not predict performance. The model accounted for 48% of the variance.

Since the ANAM Sleep Scale was missing data from 6 HCs, a multiple regression was conducted without the ANAM. The model included group (HC or SLE), BDI, and TAI-Y and significantly predicted performance on the Relational Learning task, $F(3, 25) = 4.78$, $p = 0.01$. Group, $p = 0.02$, was a significant predictor, whereas scores on the BDI, $p = 0.66$, and TAI-Y, $p = 0.44$, were not. The model accounted for 32% of the variance. When Ab status was included
in the regression, the model significantly predicted performance on the Relational Learning Task, $F(4, 24) = 3.45, p = 0.02$. DNRAb+ status was a significant predictor, $p = 0.03$, whereas DNRAb- status trended towards was marginally significant, $p = 0.051$, and scores on the BDI, $p = 0.65$, and TAI-Y, $p = 0.45$, were not. The model accounted for 37% of the variance.

**Block 3:** Performance on Block 3 of the Relational Learning Task was significantly correlated with scores on the BDI and ANAM Restlessness Scale. A multiple regression was conducted, which included the BDI, ANAM Restless Scale, and group (HC or SLE). The model significantly predicted performance on Block 3 of the Relational Learning Task, $F(3, 25) = 5.09$, $p = 0.01$. However, the BDI scores, $p = 0.11$, ANAM Restless Scale, $p = 0.12$, and group, $p = 0.13$ were not significant predictors. The model accounted for 38% of the variance.

When Ab status was included in the regression, the model significantly predicted performance on Block 3 of the Relational Learning task, $F(4, 24) = 4.00, p = 0.01$. Yet, again none of the variables emerged as significant predictors, including the scores on the BDI, $p = 0.19$, ANAM Restlessness Scale, $p = 0.09$, DNRAb- status, $p = 0.08$, and DNRAb+ status, $p = 0.30$. The model accounted for 40% of the variance.

**Recognition Stimuli:** Only performances on the relational items were significantly correlated with scores on the BDI. The shape and spoiler items were not correlated with scores on any of the affective items. A regression, which included scores on the BDI and group (HC or SLE), did not significantly predict errors on the relational items, $F(2, 26) = 2.73, p = 0.08$. When Ab status was included in the model, it, again, did not significantly predict errors on the relational items, $F(3, 25) = 1.89, p = 0.16$.

**Relational Learning Task Regression Summary:** Scores on the ANAM Sleep Scale predicted performance on the Relational Task in its entirety. However, while the ANAM Sleep
Scale emerged as a significant predictor of performance, the incomplete data for the HCs participants (only 6 HCs had missing data, leaving only 3 HCs with the ANAM) resulted in the removal of that item. Subsequent regressions using the overall performance on the Relational Learning Task yielded only SLE groups and, more specifically, DNRAb+ status as significantly predictors, and not scores on the BDI or TAI-Y. When analyzing only Block 3 and the relational Recognition Stimuli, the affective measures did not significantly predict performance.

Aim 3f. The third goal was to assess if any relationship exists between neuropsychological or cognitive factors and proportion of accurate responses on the (1) whole task, (2) learning slope, (3) Block 3, and (4) Recognition Stimuli (see Tables 36-51). Correlation tables for the neuropsychological tests are separated between Block 1 (Table 36), Block 2 (Table 37), Block 3 (Table 38), and the entire task (Table 39). Correlation tables for ANAM visuospatial are separated between Block 1 (Table 40), Block 2 (Table 41), Block 3 (Table 42), and the entire task (Table 43). Correlation tables for ANAM processing speed scores are separated between Block 1 (Table 44), Block 2 (Table 45), Block 3 (Table 46), and the entire task (Table 47). Correlation tables for the ANAM Go/No-Go inhibition task are separated between Block 1 (Table 48), Block 2 (Table 49), Block 3 (Table 50), and the entire task (Table 51).

Correlations. (1) Whole Task: Higher scores on the JLO, $r = 0.50$, $p = 0.01$, faster TMT-B completion, $r = -0.43$, $p = 0.02$, and greater efficiency on the Match to Sample task, $r = 0.61$, $p < 0.01$, were correlated with a higher proportion of accurate responses overall.

(2) Learning Slope: Faster TMT-B completion was significantly correlated with a higher proportion of accurate responses overall, $r = -0.41$, $p = 0.03$. 
(3) Block 3: Higher scores on the JLO, $r = 0.47$, $p = 0.01$, faster TMT-B completion, $r = -0.57$, $p < 0.01$, and greater efficiency on the Match to Sample task, $r = 0.61$, $p < 0.01$, were significantly correlated with a greater proportion of accurate responses in Block 3.

(4) Relational Items: Higher scores on the JLO, $r = 0.64$, $p < 0.01$, quicker TMT-B completion, $r = -0.46$, $p = 0.01$, higher raw scores on the RCFT copy, $r = 0.59$, $p < 0.01$, and the RCFT delay, $r = 0.67$, $p < 0.01$, and better signal detection values on the Go/No-Go task, $r = 0.40$, $p = 0.03$, were significantly correlated with fewer errors on the relational items. Shape items: Higher scores on the JLO, $r = 0.42$, $p = 0.02$, and higher raw scores on the RCFT copy, $r = 0.44$, $p = 0.02$, and the RCFT delay, $r = 0.60$, $p < 0.01$, was significantly correlated with fewer errors on the shape items. Spoilers were not significantly correlated with any neuropsychological or cognitive scores.

Linear Regression. Whole Task: Performance on the Relational Learning Task was significantly correlated with scores on the JLO, TMT-B, and efficiency on the ANAM Match to Sample task. When these scores were included in the regression model, in addition to including group (HC or SLE), the model significantly predicted performance on the Relational Learning Task, $F(4, 24) = .14$, $p < 0.01$. Group was a significant predictor, $p = 0.04$, whereas the JLO, $p = 0.10$, TMT-B, $p = 0.83$, and ANAM Match to Sample task, $p = 0.07$, were not. The model accounted for 54% of the variance.

When Ab status was included in the regression, the model significantly predicted performance, $F(5, 23) = 5.47$, $p < 0.01$. However, none of the tasks nor Ab status emerged as significant predictors ($ps > 0.05$). The model accounted for 54% of the variance.

Block 3: Performance on Block 3 of the Relational Learning Task was significantly correlated with scores on the JLO, TMT-B, and efficiency on the ANAM Match to Sample task.
When these scores were included in the regression model, in addition to including group (HC or SLE), the model significantly predicted performance on Block 3 of the Relational Learning task, $F(4, 24) = 6.73, p < 0.01$. However, none of the variables emerged as significant predictors, $p > 0.05$. This model accounted for 53% of the variance.

When Ab status was included in the regression, the model significantly predicted performance on Block 3 of the Relational Learning Task, $F(5, 23) = 5.41, p < 0.01$. Although efficiency on the Match to Sample task was close, it did not significantly predict performance, $p = 0.06$, whereas the JLO, $p = 0.38$, TMT-B, $p = 0.14$, DNRAb- status, $p = 0.20$, and DNRAb+ status, $p = 0.56$, did not predict performance on Block 3. The model accounted for 54% of the variance.

Recognition Stimuli: While spoiler recognition items were not significantly correlated with neuropsychological measures, both relational and spatial items were. Errors on the relational recognition items were significantly correlated with scores on the JLO, TMT-B, RCFT copy, RCFT delay, and signal detection values on the Go/No-Go task. When these scores were included in the regression model, in addition to including group (HC or SLE), the model significantly predicted errors on the relational items of the Relational Learning task, $F(6, 22) = 7.79, p < 0.01$. Signal detection on the Go/No-Go task, $p = 0.01$, emerged as a significant predictor, whereas the JLO, $p = 0.52$, TMT-B, $p = 0.13$, RCFT copy, $p = 0.47$, and group, $p = 0.94$, did not. Scores on the RCFT delay trended towards significant predictors, $p = 0.06$. The model accounted for 68% of the variance.

When Ab status was included in the regression, the model significantly predicted errors on the relational recognition items, $F(7, 21) = 7.08, p < 0.01$. Both the RCFT delay, $p = 0.03$, and signal detection on the Go/No-Go task, $p = 0.01$, emerged as significant predictors,
whereas the JLO, $p = 0.95$, TMT-B, $p = 0.23$, RCFT copy, $p = 0.39$, and DNRAb- status, $p = 0.52$, and DNRAb+ status, $p = 0.58$, did not. The model accounted for 70% of the variance.

Errors on the shape recognition items were significantly correlated with scores on the JLO, RCFT copy, and RCFT delay. When these scores were included in the regression model, in addition to including group (HC or SLE), the model significantly predicted errors on the shape items, $F(4, 24) = 3.43, p = 0.02$. The RCFT delay, $p = 0.04$, was a significant predictor, whereas the JLO, $p = 0.90$, the RCFT copy, $p = 0.85$, and group, $p = 0.86$, were not. The model accounted for 36% of the variance.

When Ab status was included in the regression, the model significantly predicted errors on the shape recognition items, $F(5, 23) = 2.95, p = 0.03$. The RCFT delay, $p = 0.02$, was a significant predictor, whereas the JLO, $p = 0.75$, RCFT copy, $p = 0.84$, and DNRAb- status, $p = 0.50$, and DNRAb+ status, $p = 0.77$, did not. The model accounted for 39% of the variance.

*Relational Learning Task Regression Summary*: Group (HC or SLE), but not Ab status, was a significant predictor of performance on the Relational Learning task, whereas performance on the JLO, TMT-B, and ANAM Match to Sample were not. None of the items emerged as significant predictors of performance on Block 3 of the task. Signal detection measured on the Go/No-Go task was a significant predictor of errors on the relational items. However, when Ab status was included both RCFT delay scores and signal detection on the Go/No-Go task were significant predictors of errors on the relational items. The RCFT delay scores were significant predictors of errors on the shape items, and remained the only significant variable even when Ab status was included in the analyses.
CHAPTER IV

Discussion

SLE patients frequently experience cognitive impairment; however, research on the etiology of this symptom has been limited, unsatisfactory, and, at times, even contradictory. There are many reasons and factors that have contributed to this variability in cognitive deficits observed in SLE patients. Some of these factors include the inflammatory autoimmune response, disease activity, psychosocial and affective/psychological, and biological and environmental factors. Although these different factors have been explored in previous research, results have been inconclusive and generally report that there are many nonspecific factors that result in cognitive dysfunction.

To address the insufficient research in this area, researchers have begun to examine whether greater precision in predicting specific kinds of cognitive deficits can be achieved by assessment of Abs that are present. One Ab of interest that may influence learning and memory is the DNRAb as it targets NMDARs, causing cell apoptosis, and NMDARs are often located in brain regions critical for learning and memory. In particular, murine models have demonstrated that damage caused by DNRAbs results in apoptosis in the hippocampus, which has shown impaired spatial memory in mice (Chang et al., 2015; Faust et al., 2010; Kowal et al., 2006; Mackay et al., 2011). DNRAbs are a subtype of anti-DNA antibodies that cross-react with DNA and the NR2 subunit of NMDA receptors, which are typically found in the hippocampus. Furthermore, recently, research has emerged that supports the relationship between cognitive deficits (particularly in visuospatial, learning, and memory domains) and the presence of NMDAR Abs (Lapteva et al., 2006, Omdal et al., 2005), while controlling for factors (e.g., age and disease duration) often associated with cognitive decline (Mackay et al., 2015). Given this
body of research, murine studies support the relationship among the presence of the DNRAbs in a SLE model, hippocampal damage, and spatial memory impairments.

One study by Chang and colleagues (2015) specifically examined spatial memory deficits in an SLE population and murine models of SLE. They found that high levels of DNRAbs were associated with spatial memory impairment, such that mice injected with a virus mimicking SLE demonstrated greater inability to navigate a spatial environment and that their impairment was directly correlated with hippocampal damage. Interestingly, the place cells within the hippocampus of the “SLE mice” were less specific when encoding/navigating an environment compared to control mice. Moreover, the researchers found that within a human SLE population, elevated level DNRAbs led to similar impairment on a spatial memory task; however, the sample size was quite limited. One goal of the present study was to replicate these earlier findings and to extend cognitive impairment to relational memory.

Another purpose of this project was to explore whether a diagnosis of SLE, and specifically whether high levels of DNRAbs, were associated with cognitive deficits in spatial and relational learning and memory. The broader aim was to better predict cognitive deficits in SLE patients, which has implications for treatment and psychological well-being. Based on the background literature, it was predicted that SLE groups would have poorer performance across neuropsychological and behavioral tasks as compared to HCs. It was further anticipated that the DNRAb+ SLE participants would have greater spatial and relational memory deficits as compared to their SLE DNRAb- counterparts.

In general, the predicted findings only partially supported by the current data. More specifically, although group (HC or SLE, or DNRAb status) was initially associated with performance differences across the behavioral Spatial Memory and Relational Learning tasks, the expected finding that the DNRAb- group would perform more similarly to the HC group did
not emerge. On the Spatial Memory Task, while the HC group was significantly more accurate than the DNRAb+ group, the DNRAb- group’s performance did not significantly differ from either the HC or DNRAb+ groups. It appeared that on this spatial task, the DNRAb status was related to poorer performance. However, the Relational Learning task did not demonstrate similar findings. Although the relational learning task found that HCs were more accurate than the SLE group, Ab status was not related to performance. In contrast, the DNRAb groups performed similarly to one another.

The failure to obtain results that significantly differentiated the DNRAb groups may be due to group differences or due to performance differences on a number of the neuropsychological measures. There were significant differences between the HC and SLE groups. For instance, the HC group was younger, had a higher education, and lower levels of depression and state anxiety than the SLE group. In addition, the SLE group was 2.8 times more likely to have a family history of autoimmune disease and 8.56 times more likely to report having a history of cognitive dysfunction. When Ab status was used to divide the SLE group into DNRAb- and DNRAb+ subgroups, the results remained relatively similar. The HC group had a higher level of education than DNRAb+, lower levels of depression than both DNRAb- and DNRAb+ groups, and lower trait anxiety than DNRAb-. These differences remained constant even when using criteria that are more stringent and eliminating participants who were more than 55 years old and had more than 21 years of education. Furthermore, it is possible these differences could have impacted performances on cognitive testing.

Aim 1: Group differences in Spatial Memory between HCs and SLE patients.

The initial results supported prior research by Chang and colleagues (2015). Most critically, when assessing the easy matrix, the DNRAb+ group performed worse on the Spatial Memory Task compared to the other groups, replicating their findings. Specific to the current
project, it was observed that individuals had better memory for the easy items, compared to the more difficult items, and were more accurate for the nonspatial items compared to the spatial items. Overall, the HC group outperformed the SLE group, with the worst performance observed in the DNRAb+ subgroup. In further support of the Chang study, the difficult items were considered especially challenging for the participants, and all participants performed equally well on the nonspatial memory items.

However, other factors may have influenced the participants' performance on the Spatial Memory task. When examining background factors that were correlated and predicted performance on the Spatial Memory task, there were some notable findings. For instance, age and education predicted performance on both nonspatial and spatial items of the Spatial Memory task. Speed on the TMT-B task accounted for performance differences for spatial memory items only. Affective and neuropsychological measures were not associated with or predicted performance on the Spatial Memory task. Surprisingly, the RCFT copy was related to nonspatial performance. However, neither the copy nor delay were related to nor predicted spatial memory performance. This finding was unexpected given the spatial nature of the RCFT task. Yet, the RCFT task relies on multiple cognitive functions, including planning and organization. Thus, it is possible that these other functions more appropriately characterize this task. Future studies may examine how these intricately connected cognitive functions differentially contribute to performances on cognitive tasks in SLE populations.

Aim 2: Group Differences in Relational Learning between HCs and SLE patients.

Interestingly, the Relational Learning Task did not provide as compelling results as the Spatial Memory Task. The HC group, overall, was more accurate than the SLE group, and performance improved with each subsequent block. Upon further review of the results, however, the performance between the two DNRAb subgroups was not significantly different. As for the
types of errors participants made, overall the entire sample made fewer errors on spoiler items than on the shape and relational items. Importantly, Ab status did not predict error rates for any of the foil types, suggesting that all SLE individuals made errors in a similar manner.

Performance on the relational learning task was also assessed in relation to demographic factors, affective measures, and neuropsychological assessments. Individuals with higher education performed better on the Relational Learning task, as a whole, but education did not predict performance on Block 3, suggesting that education influenced initial learning but not overall learning. When the ANAM Sleep Scale, a measure of current fatigue levels, was included in analyses, it was a significant predictor of accuracy on the Relational Learning task for HCs versus SLE patients. Current levels of fatigue can influence cognitive performance, particular in SLE patients, who are susceptible to fatigue (Kozora et al., 2006). It is important to evaluate how fatigue would affect patient performance on a variety of cognitive tasks. However, a majority of the HCs did not receive the ANAM Sleep Scale, due to a computer error, and a subsequent analysis was conducted without this scale. The remaining affective and neuropsychological measures did not significantly predict performance on the task as a whole or on Block 3. Specific to recognition stimuli, better performance on the Go/No-Go task and RCFT delay predicted fewer errors on the relational recognition items, and only better performance on the RCFT delay predicted fewer errors on shape recognition stimuli. Thus, the ability to differentiate between target and non-target stimuli (Go/No-Go task) and better memory of the RCFT may be related to better identification of target stimuli and possibly to the ability to prevent distraction on the Relational Learning Task.

**Neurobiological Implications**

Taken together, the results of the present study appeared to find deficits in spatial memory or relational learning in individuals with SLE, however, we did not find robust findings
differentiating SLE patients on the basis of their Ab status. The DNRAb- group did not significantly differ from the DNRAb+ groups on either of the computerized measures. This is contradictory to our expected findings, especially considering the developing body of research that associates (1) DNRAb levels with hippocampal atrophy, and (2) resulting spatial memory impairment in mice. It is possible that the current study did not support previous animal models because learning and memory are complex processes that rely on multiple systems. Moreover, other factors, such as education or age may have had a greater influence on performance than SLE status. Animal research often does not have to worry about these vital and important factors that are uniquely associated with human research.

As a reminder, DNRAbs are known to bind to both DNA and preferentially to the GLU2 subunits of NMDA receptors. NDMA receptors are found throughout the brain; however, GluN2A and GluN2B are most prevalent in the CA1 unit of the hippocampus and amygdala (Aranow, Diamond, & Mackay, 2010; Danysz & Parsons, 1998; Omdal et al., 2005). Further, the CA1 unit of the hippocampus has been associated with the facilitation of spatial memory (Lee & Kesner, 2002). Yet, the hippocampus is not the only structure involved in spatial memory. Many areas of the brain contribute to spatial processing and attention, including parietal lobe, right prefrontal cortex, and other subcortical structures. It is quite possible that these other areas may aid to compensate for spatial deficits in the hippocampus and more importantly other demographic and intellectual factors may also facilitate compensatory processes that may mask hippocampal-dependent spatial memory deficits.

The hippocampus works as part of a greater network of neuroanatomical structures in order to process spatial and relational learning and memory. The dorsal pathway of the hippocampus is known to be related to spatial memory and to project to other areas of the brain, including reciprocal connections from the DG, medial and lateral mammillary nuclei, and the
anterior thalamic complex (Fanselow & Dong, 2010; Moscovitch et al., 2005). Therefore, it is possible that the entire network of hippocampal dependent spatial memory was not affected by the DNRAb presence and could possibly compensate for some of the disruption if there was hippocampal damage. The extensive network involved in the formation, maintenance, and retrieval of spatial memory, extend far beyond the hippocampus and may account for some of the correlations between some of the cognitive measures that were observed in this study. Further, the poorer performance on the neuropsychological measures across the groups appears to indicate that the SLE participants experienced subtle, but diffuse cognitive deficits. Specifically, SLE participants may have more diffuse cognitive deficits than HCs, and these deficits may increase the difficulty to identify hippocampal-dependent spatial memory deficits. For instance, deficits in attention or working memory may impair spatial memory performance to a greater degree than any hippocampal-dependent spatial memory impairment, as such deficits in attention and working memory may impair the ability to properly encode spatial information.

It is curious that the findings did not support the previous literature. Given the fact that the DNRAb+ participants did not significantly differ from DNRAb- on the two computerized behavioral tasks, additional hypotheses were generated to explain the findings of the study. Speculatively, it could be possible that the blood serum levels detected in the SLE did not result in a breach of the BBB and apoptosis of hippocampal NDMAR. This could be addressed in future papers, as PET imaging was also obtained on these subjects (Cohort B only) to determine whether the hippocampus or other neuroanatomical structures were implicated in the disease pathology of the current sample. Although this imaging was not available at the time of the current study, it will be available for future analyses. Thus, it might be imperative when assessing blood serum to also include imaging analysis to determine extent of hippocampal atrophy, DNRAb presence, and cognitive deficits to determine if serum is sufficient or if assessment from CNS fluid is more appropriate and predictive of cognitive deficits.
Treatment Implications

Intact processing of spatial and relational learning and memory are essential functions. It is by these processes that living beings are able to understand the visual world around them and to use visual cues to navigate through their environment. Dysfunction in spatial cognition, particularly in learning and memory, will pose adverse limitations on the way that individuals can interact with others and the space around them. It could affect an individual’s ability to complete simple daily tasks, such as perceive and remember objects or their locations, and more complex activities, such as run an errand and recall how to return home. For instance, as part of the present study, Cohort B participants were asked to complete a navigation task (not discussed within this project), and the SLE patients had extremely difficulty navigating a spatial environment presented on a laptop. Thus, in the most extreme circumstances, an individual with impaired visuospatial learning and memory will need to rely on others to care for them to assist in or complete tasks that rely heavily on spatial memory, or they will need to find support elsewhere, either through a form of cognitive training or relying on navigational technology (e.g., Google Maps).

The current study found only limited cognitive impairments in the SLE participants. It is consistent with previous SLE literature that continues to yield inconclusive, and at times, contradictory neurocognitive findings. Additional studies that aim to replicate, and expand upon, the findings by Chang and colleagues (2015) will be needed in order to support the presence of deficits in spatial and relational learning and memory. Moreover, future research should look to directly link hippocampal atrophy and spatial/relational learning deficits in real time (fMRI or PET studies) and to more accurately assess Abs presence in the CNS in order to provide a clearer and more complete picture linking specific Abs to specific cognitive deficits. If such links are found to be more conclusive, then researchers and treating clinicians of SLE patients should
seek preventative treatments and/or compensatory strategies to cope with the burden of a visuospatial learning and memory impairment.

While the SLE (both DNRAb- and DNRAb+) groups did not significantly differ on their performance on the Spatial Memory or Relational Learning tasks, they did appear to differ on measures of cognitive functioning relating to processing speed and executive function. These findings do support the existing literature, which indicates that these domains are impaired in SLE patients (El-Shafey et al., 2012; Emori et al., 2005; Loukkola et al., 2003; Nowicka-Sauer et al., 2011; Vogel et al., 2011). In particular, cognitive switching was an area of difficulty among the SLE participants. This skill was impaired on a single neuropsychological measure, and future studies may provide a more nuanced approach to evaluating executive functioning deficits within an SLE population. The poorer performance on the Trailmaking Test requires the ability to switch between numbers and letters in a spatial environment. It is possible that the poorer performance on this task could be related to either set-switching or spatial abilities.

Therefore, an approach that eliminates the visuospatial burden (for instance, asking participants to orally switch between numbers and letters) may help to provide further insight to the aspects of the task that are difficult to participants. Future studies should look at alternative forms of switching tasks in order to evaluate whether the deficit is related to executive functions or spatial deficits. In particular, a more thorough understanding of cognitive deficits will help treating providers in serving patients with SLE.

Emotional functioning is an area of concern in SLE patients, which was also supported in the current study. The SLE groups did appear to have greater levels of depression and anxiety as compared to HCs. Curiously, the DNRAb- group was found to report greater levels of depression and anxiety as compared to HCs. Yet, the DNRAb+ group’s scores were somewhere in between the HC and DNRAb- groups and did not significantly differ among either
groups. Kozora and colleagues found that patients with NPSLE had higher levels of depression than non-NPSLE patients (2006). It is unclear what made the study samples different in their reports of depression. However, emotional functioning can certainly influence cognition (namely diffuse subcortical functions such as processing speed and attention) and subjective cognitive concerns (Covey et al., 2012; Denburg & Denburg, 1999; Hay et al., 1992; Kozora et al., 2006; 2007; Vogel et al., 2011). By identifying affective distress early in the course of the disease, providers could help patients seek early intervention and improve patient quality of life.

Incomplete data (on the ANAM Fatigue Scale) is considered one of the limitations of this project. Due to a computer error, one of the measures in this study that examined current levels of fatigue was not collected across all participants. SLE patients are known to be more susceptible to experience fatigue. Furthermore, fatigue is known to interact with symptoms such as pain, as well as both emotional and cognitive functioning. A body of literature is beginning to form that explores the use of nonpharmacological interventions to address concerns of fatigue in SLE. A variety of methods have been implemented, including cognitive/behavioral methods, dietary changes, exercise, phototherapy and homeopathic remedies (such as acupuncture). A meta-review of some of the available methods has yielded promising treatment results in reducing complaints of fatigue in SLE patients (del Pino-Sedeño et al., 2016). However, there were only a few available studies in the meta-analysis paper, which all implemented different techniques and measurements to conduct the nonpharmacological interventions. Nonpharmacological interventions are also available to treat symptoms of emotional distress, compensate for cognitive impairments, and address the widespread implications of chronic pain.

In addition to assessing cognitive deficits associated with specific Abs in SLE patients, future studies should aim to implement nonpharmacological interventions and evaluate their efficacy. Addressing these symptoms using nonpharmacological techniques would provide cost-
effective treatments without the side effects typically associated with medications. Since SLE participants already experience a variety of physical symptoms, the reduction of medication adverse effects may be a beneficial alternative to the current available pharmacological agents.

Limitations of the Present Study

The present study had several limitations, including sample selection, sample size, Ab measurement, and battery of tests. With regards to the selection of the sample, there were two separate cohorts who were recruited with somewhat different inclusion and exclusion criteria. While Cohort B’s criteria were more stringent than Cohort A’s criteria, both required strict criteria in order to limit the potential confounds associated with SLE disease pathology. Importantly, a history of focal neuroanatomical insult was an exclusionary criterion. However, this could limit potential findings if DNRAbs caused focal insult and subsequent cognitive impairment. Thus, Cohort B may have been less impaired cognitively and with less brain insult than Cohort A, reducing the possibility to find cognitive deficits with such a restrictive range.

The sample size was another limitation of this study. To increase the power for the Memory Task, Cohorts A and B were combined. However, only Cohort B completed the Relational Learning Task. A power analysis conducted prior to subject recruitment of Cohort B found that a sample size of 12 HCs and 24 SLE patients would be sufficient to detect a significant difference between groups at the alpha = 0.01 level. However, after three years of recruitment, the group sizes had not been reached. Rather, the HC group had only nine subjects with analyzable data, which is below the generally accepted standard of n = 10 used for statistical analyses. And the entire Cohort B sample only included 29 total subjects with analyzable data. Further, given the large number of variables included in the models, an even larger sample size would need to be included to increase the power. However, these sample
sizes were deemed adequate for the current study given the low prevalence rate of SLE in the general population.

The method of measuring the presence of DNRAbs may be a limitation. Antibody presence was measured using blood serum, rather than measuring CSF. CSF is a more reliable method of assessing whether DNRAbs are affecting the CNS. Further, Ab presence in CSF is consistently related to cognitive impairment in SLE participants, whereas serum Ab has not been as reliable (Fragoso-Loyo, et al., 2008; Kowal et al., 2006; Mackay et al., 2015). While assessing CSF Abs levels would provide a more effective measure in associating cognitive impairment with Ab level, this level was not selected due to the cost and risk to the participants and because previous literature suggested that there is an association between serum levels, NP involvement, and cognitive deficits (Kowal et al., 2006; Mackay et al., 2015).

The selection and order of the cognitive tests may serve as a limitation. Cohort B received only two of the recommended measures from the ACR recommended battery (Table 3), the TMT A & B and the RCFT (Kozora et al., 2004). The remaining measures were not selected due to the study’s focus on spatial and relational learning and memory. Instead, a measure of visuoperception was included in order to rule out basic deficits in visual acuity. The ANAM battery was included, and only tasks that appeared to be related to attention, processing speed, visuospatial functioning, and executive functioning were selected. The Spatial Memory and Relational Learning tasks were developed to provide objective assessments regarding different aspects of visuospatial learning and memory that are not readily available on current paper-and-pencil neuropsychological measures. It is possible that these particular measures were not sensitive enough to detect the subtle cognitive decline that is often reported in SLE populations. The neuropsychological, behavioral, and ANAM battery did not include an estimate of premorbid functioning or intellectual functioning, nor did it include measures of language
functioning. While these measures would certainly be important to assess and to provide a comprehensive evaluation, they were not included in order to develop a time-limited battery that focused specifically on the study’s aims.

The method of scoring tasks may pose as a limitation in this study. Raw scores for the JLO, TMT tasks, and RCFT were used in the analyses. This method is consistent with the methodologies implemented in former studies (El-Shafey et al., 2012; Kozora, 2008). However, the demographic differences between the groups on age and years of education, future studies may seek to use standardized scores to minimize the influence these factors have on scores.

In addition to the type of measures selected for this battery, as well as their method of scoring, another limitation to this study is the order of the tasks. Cohort B received the Spatial Memory Task during the delay for the RCFT. There were some concerns regarding the Spatial Memory Task interfering with the delayed recall on the RCFT. Typically, during an ideal testing situation, it is preferable to have verbal tasks during the delay of a visuospatial memory task. Due to the time constraints of the testing battery, the Spatial Memory Task was used during the delay period for the RCFT task. Thus, the results for the RCFT must be interpreted cautiously. However, the images presented in both tasks were different modalities (one was a nonsensical/complex figure, while the other was line drawings of common objects). Furthermore, the tasks used different administrations: the complex figure was a paper and pencil task whereas the Spatial Memory Task was computerized. It is important to note that during the delayed recall for the RCFT, none of the participants (from both HC and SLE groups) drew any of the line drawings from the computer screen or reported confusion between the two tasks. Furthermore, since all groups experienced the same distraction, if group differences did arise in the RCFT, it would suggest a deficit related to the group differences.
Another limitation of this study was the fact that imaging was not available to directly link Ab status, hippocampal (and other brain area) damage, and cognitive deficits. Specific to Cohort B, these participants were recruited for a longitudinal study that included PET, MRI, and DTI measures of brain functioning, structural damage, and network connections, respectively. The ability to analyze imaging would allow the identification of brain regions affected by NMDAR disruption and if that disruption was predictive of spatial and relational memory deficits than assessing Ab status via serum. Furthermore, the identification of these areas would allow additional analyses on how cognition is related to regional and network dysfunction.

**Future Directions**

The current study served as a baseline regarding the evaluation of spatial and relational learning and memory deficits. While there is only one known study that examines spatial memory in SLE patients who are DNRAb+ (Chang et al., 2015), the literature is greatly limited regarding relational learning and memory. The participants in Cohort B of this project were part of a larger, longitudinal imaging study that will continue for a period of 3 annual evaluations (baseline, approximately 1 year later, and approximately 1 more year after that). The longitudinal study will allow researchers to evaluate how the presence of DNRAbs vary over time and whether there are associated changes in cognitive status that are related to structural and functional changes in the brain, particularly the hippocampus. Furthermore, recruitment will continue in order to increase the Cohort B sample sizes to increase power and to protect against participant attrition. This study will also be strengthened with the inclusion of the neuroimaging techniques that have been gathered but not yet analyzed.

As more studies begin to evaluate the relationship between Abs and cognitive functioning, a wider variety of neuropsychological measures should be implemented in order to assess for subtle cognitive decline. Future studies could further analyze the influence of DNRAb
levels on cognitive dysfunction by employing a greater variety of affective and spatial measures. This study only analyzed spatial memory based on the murine SLE model of DNRAb atrophy. However, the human hippocampus is also intricately involved in verbal and episodic memory, which should also be evaluated in this select population (Banich & Compton, 2011; Blumenfeld, 2002; Burgess et al., 2002). In addition, the current study found deficits in executive function and processing speed for the SLE patients. Although the literature does appear to support this, the extent and breadth of these deficits have not been thoroughly investigated.

**Conclusion**

Taken together, the current study was developed to identify the presence of spatial memory and relational learning deficits in participants with SLE. It additionally examined the contribution of demographic factors, (i.e., age, education, disease duration), affective functioning (i.e., depression, anxiety, fatigue, and happiness), and cognitive functioning (i.e., attention, processing speed, basic visuoperception, and executive functioning) on hippocampal-dependent visuospatial memory functions. The results supported the presence of spatial or relational learning and memory deficits in SLE patients overall, however it did not support murine models of spatial memory deficits in DNRAb+ mice. However, there were group differences on a measure of executive functioning, particularly on a cognitive set-switching task, which were consistent with the previous literature.
### Tables and Figures

#### Table 1.
**1997 Update of the 1982 American College of Rheumatology Revised Criteria for Classification of Systemic Lupus Erythematosus**

<table>
<thead>
<tr>
<th>Criterion</th>
<th>Definition</th>
</tr>
</thead>
<tbody>
<tr>
<td>Malar Rash</td>
<td>Fixed erythema, flat or raised, over the malar eminences, tending to spare the nasolabial folds</td>
</tr>
<tr>
<td>Discoid Rash</td>
<td>Erythematous raised patches with adherent keratotic scaling and follicular plugging; atrophic scarring may occur in older lesions</td>
</tr>
<tr>
<td>Photosensitivity</td>
<td>Skin rash as a result of unusual reaction to sunlight, by patient history or physician observation</td>
</tr>
<tr>
<td>Oral Ulcers</td>
<td>Oral or nasopharyngeal ulceration, usually painless, observed by physician</td>
</tr>
<tr>
<td>Nonerosive Arthritis</td>
<td>Involving 2 or more peripheral joints, characterized by tenderness, swelling, or effusion</td>
</tr>
<tr>
<td>Pleuritis or Pericarditis</td>
<td>Pleuritis--convincing history of pleuritic pain or rubbing heard by a physician or evidence of pleural effusion</td>
</tr>
<tr>
<td></td>
<td>Pericarditis--documented by electrocardiogram or rub or evidence of pericardial effusion</td>
</tr>
<tr>
<td>Renal Disease</td>
<td>Persistent proteinuria &gt; 0.5 grams per day or &gt; than 3+ if quantitation not performed</td>
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<tr>
<td></td>
<td>OR</td>
</tr>
<tr>
<td></td>
<td>Cellular casts--may be red cell, hemoglobin, granular, tubular, or mixed</td>
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<tr>
<td>Neurologic Disorder</td>
<td>Seizures--in the absence of offending drugs or known metabolic derangements; e.g., uremia, ketoacidosis, or electrolyte imbalance</td>
</tr>
<tr>
<td></td>
<td>OR</td>
</tr>
<tr>
<td></td>
<td>Psychosis--in the absence of offending drugs or known metabolic derangements, e.g., uremia, ketoacidosis, or electrolyte imbalance</td>
</tr>
<tr>
<td>Hematologic Disorder</td>
<td>Hemolytic anemia--with reticulocytosis</td>
</tr>
<tr>
<td></td>
<td>OR</td>
</tr>
<tr>
<td></td>
<td>Leukopenia--&lt; 4,000/mm$^3$ on ≥ 2 occasions</td>
</tr>
<tr>
<td></td>
<td>OR</td>
</tr>
<tr>
<td></td>
<td>Lyphopenia--&lt; 1,500/mm$^3$ on ≥ 2 occasions</td>
</tr>
<tr>
<td></td>
<td>OR</td>
</tr>
<tr>
<td></td>
<td>Thrombocytopenia--&lt;100,000/mm$^3$ in the absence of offending drugs</td>
</tr>
<tr>
<td>Immunologic Disorder</td>
<td>Anti-DNA: antibody to native DNA in abnormal titer</td>
</tr>
<tr>
<td></td>
<td>OR</td>
</tr>
<tr>
<td></td>
<td>Anti-Sm: presence of antibody to Sm nuclear antigen</td>
</tr>
<tr>
<td></td>
<td>OR</td>
</tr>
<tr>
<td></td>
<td>Positive finding of antiphospholipid antibodies on:</td>
</tr>
<tr>
<td></td>
<td>An abnormal serum level of IgG or IgM anticardiolipin antibodies,</td>
</tr>
</tbody>
</table>
A positive test result for lupus anticoagulant using a standard method, or
A false-positive test result for at least 6 months confirmed by Treponema pallidum immobilization or fluorescent treponemal antibody absorption test

| Positive Antinuclear Antibody | An abnormal titer of antinuclear antibody by immunofluorescence or an equivalent assay at any point in time and in the absence of drugs |

<table>
<thead>
<tr>
<th>Central Nervous System</th>
<th>Peripheral Nervous System</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aseptic meningitis</td>
<td>Autonomic disorder</td>
</tr>
<tr>
<td>Cerebrovascular disease</td>
<td>Mononeuropathy</td>
</tr>
<tr>
<td>Cognitive dysfunction</td>
<td>Cranial neuropathy</td>
</tr>
<tr>
<td>Headache</td>
<td>Plexopathy</td>
</tr>
<tr>
<td>Movement disorder (Chorea)</td>
<td>Polyneuropathy</td>
</tr>
<tr>
<td>Seizures</td>
<td>Acute inflammatory demyelinating polyradiculoneuropathy (Guillain-Barré syndrome)</td>
</tr>
<tr>
<td>Acute confusional state</td>
<td></td>
</tr>
<tr>
<td>Anxiety disorder</td>
<td>Myasthenia gravis</td>
</tr>
<tr>
<td>Mood disorder</td>
<td></td>
</tr>
<tr>
<td>Psychosis</td>
<td></td>
</tr>
<tr>
<td>Demyelinating syndrome</td>
<td></td>
</tr>
<tr>
<td>Myelopathy (transverse myelitis)</td>
<td></td>
</tr>
</tbody>
</table>

Note: Adapted from American College of Rheumatology Ad Hoc Committee on Neuropsychiatric Lupus Nomenclature (1999). The American College of Rheumatology nomenclature and cause definitions for neuropsychiatric lupus syndromes. Arthritis and Rheumatism, 42:4, 599-608.
Table 3.  
**ACR Proposed One-Hour Neuropsychological Battery for SLE**

<table>
<thead>
<tr>
<th>Cognitive Tests:</th>
<th>Additional Tests:</th>
</tr>
</thead>
<tbody>
<tr>
<td>North American Adult Reading Test (to estimate IQ)</td>
<td>Depressive symptoms:</td>
</tr>
<tr>
<td>Digit Symbol Substitution Test</td>
<td>Center for Epidemiological Studies-Depression Scale (CES-D)</td>
</tr>
<tr>
<td>Trail Making Test (Parts A &amp; B)</td>
<td>Fatigue and pain:</td>
</tr>
<tr>
<td>WAIS III Letter-Number Sequencing</td>
<td>Pain Visual Analog Scales, Fatigue Severity Scale</td>
</tr>
<tr>
<td>Stroop Color and Word Test</td>
<td>Self-reported cognitive function:</td>
</tr>
<tr>
<td>California Verbal Learning Test</td>
<td>(e.g.) Cognitive Failures Questionnaire (CFQ)</td>
</tr>
<tr>
<td>Rey-Osterrieth Complex Figure Test (with delayed recall)</td>
<td>Multiple Assessment Questionnaire (MAQ)</td>
</tr>
<tr>
<td>Controlled Oral Word Association Test (FAS)</td>
<td>Patient Assessment of Own Functioning (PAOFI)</td>
</tr>
<tr>
<td>Animal Naming</td>
<td>Impact on daily living:</td>
</tr>
<tr>
<td>Finger Tapping</td>
<td>(e.g.) 36-item Short-Form Health Survey Questionnaire (SF-36)</td>
</tr>
</tbody>
</table>

### Table 4.
Comparison of cognitive performance of (1) NP-SLE patients, (2) SLE patients, and (3) HC across studies.

<table>
<thead>
<tr>
<th></th>
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<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>MMSE</td>
<td>-</td>
<td>2 &lt; 3</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>1 &lt; 3</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>MOCA</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>NS</td>
<td>NS</td>
</tr>
</tbody>
</table>

### Intelligence

| WAIS-R Verbal IQ | - | - | - | - | - | NS* | - | - | - | - | - |
| WAIS-R Performance IQ | - | - | - | - | - | 1 < 3 | - | - | - | - | - |

### Attention/Processing Speed

| WAIS-R Digit Span (DS) | NS | - | NS | - | - | - | - | - | - | NS |
| WAIS-R DS Forward | NS | - | - | NS | - | - | - | - | NS | - |
| WAIS-R DS Backward | NS | - | - | NS | - | - | - | - | NS | - |
| Letter-Number Sequencing** | - | - | - | - | NS | NS | 2 < 3 | - | - | - |
| Paced Auditory Addition Test | - | - | - | - | 1 < 3 | NS | - | - | - | - |
| Spatial Span Forward** | NS | - | - | NS | - | - | - | - | NS | - |
| Spatial Span Backward** | - | - | - | NS | - | - | - | - | - | - |
| Stroop Color-Word Test: Word Naming | 1 < 3 | - | - | - | 2 < 3 | - | - | 1 < 2, 3 | - |
| Stroop Color-Word Test: Color Naming | 1 < 2, 3 | - | - | - | 2 < 3 | - | - | - | - |
| Trail Making Test – Part A | 1 < 3 | NS | 1, 2 < 3 | - | 2 < 3 | 1, 2 < 3 | - | NS | - | NS |
| Digit Symbol Substitution Test** | 1 < 3 | - | NS | 2 < 3 | 2 < 3 | 1, 2 < 3 | NS | - | NS | - |

### Executive Functioning

| Controlled Oral Word Association Test | - | - | - | - | NS | 1 < 3 | NS | - | - | NS |
| Design Fluency | 1 < 2, 3 | - | - | NS | - | - | - | - | - | - |
| Ruff Figural Fluency Test | - | - | - | - | NS | - | - | - | - | - |
| Trail Making Test – Part B | 1 < 3 | 2 < 3 | 1 < 3 | - | 2 < 3 | 1, 2 < 3 | NS | - | 1 < 2, 3 | NS |
| Stroop Color-Word Test: Interference | - | - | NS | - | NS | 1 < 3 | NS | - | 1 < 2, 3 | - |
| Category Test | - | - | NS | - | - | 1 < 3 | NS | - | - | - |
| Wisconsin Card Sorting Test (WCST) | - | - | NS | - | - | - | - | - | NS | - |
| WCST: Perseverative Errors | - | - | NS | - | - | - | - | - | NS | - |
| Raven’s Coloured Progressive Matrices | - | - | NS | - | - | - | - | - | NS | - |
| WAIS-R Similarities | - | - | NS | - | NS | NS | - | NS | - | - |
| Clock Drawing Test | - | - | - | - | - | - | - | - | 1 < 3 | - |

### Motor Functioning

<p>| Finger Tapping Test Dominant Hand | - | - | - | - | NS | 1, 2 &lt; 3 | NS | - | - | - |
| Finger Tapping Test Non-Dominant Hand | - | - | - | - | NS | 2 &lt; 3 | NS | - | - | - |</p>
<table>
<thead>
<tr>
<th>Visuospatial Processing</th>
</tr>
</thead>
<tbody>
<tr>
<td>Facial Recognition Test</td>
</tr>
<tr>
<td>Block Design**</td>
</tr>
<tr>
<td>WAIS-R Object Assembly</td>
</tr>
<tr>
<td>WAIS Picture Completion</td>
</tr>
<tr>
<td>RCFT Copy</td>
</tr>
<tr>
<td>WCFT Recognition**</td>
</tr>
<tr>
<td>RCFT Delayed Recall**</td>
</tr>
<tr>
<td>RCFT Immediate Recall**</td>
</tr>
<tr>
<td>Language</td>
</tr>
<tr>
<td>WAIS-R Vocabulary</td>
</tr>
<tr>
<td>WAIS-R Comprehension</td>
</tr>
<tr>
<td>Information**</td>
</tr>
<tr>
<td>BDAE Complex Ideational Material</td>
</tr>
<tr>
<td>Peabody Individual Achievement Test – Reading</td>
</tr>
<tr>
<td>Recognition Test</td>
</tr>
<tr>
<td>Boston Naming Test</td>
</tr>
<tr>
<td>Category Fluency</td>
</tr>
<tr>
<td>Memory</td>
</tr>
<tr>
<td>Verbal List Learning**</td>
</tr>
<tr>
<td>Verbal Immediate List Recall**</td>
</tr>
<tr>
<td>Verbal Immediate Cued List Recall**</td>
</tr>
<tr>
<td>Verbal Delayed List Recall**</td>
</tr>
<tr>
<td>Verbal Delayed Cued List Recall**</td>
</tr>
<tr>
<td>Verbal List Recognition**</td>
</tr>
<tr>
<td>WMS Verbal Paired Associates</td>
</tr>
<tr>
<td>Logical Memory Immediate Recall**</td>
</tr>
<tr>
<td>Logical Memory Delayed Recall**</td>
</tr>
<tr>
<td>Logical Memory Recognition**</td>
</tr>
<tr>
<td>Visual Reproduction Immediate Recall**</td>
</tr>
<tr>
<td>Visual Reproduction Delayed Recall**</td>
</tr>
<tr>
<td>WMS-III Visual Reproduction Recognition</td>
</tr>
<tr>
<td>RCFT Immediate Recall</td>
</tr>
<tr>
<td>RCFT Delayed Recall</td>
</tr>
<tr>
<td>RCFT Recognition</td>
</tr>
<tr>
<td>Benton Visual Retention Test</td>
</tr>
</tbody>
</table>

* Note: NS = Not significant.

** Letter-Number Sequencing = WAIS-III (Kozora et al., 2004; Kozora et al., 2008), WMS-III (Glanz et al., 2005). Digit Symbol Substitution Test = WAIS (Denburg et al., 1987a), WAIS-R (Emori et al., 2005; Glanz et al., 2005; Kozora et al., 2004; Kozora et al., 2008; Loukkola et al., 2003). Block Design = WAIS (Denburg et al., 1987a), WAIS-R (Emori et al., 2005; Glanz et al., 1997; Glanz et al., 2005; Kozora et al., 2004; Kozora et al., 2008; Loukkola et al., 2003). Spatial Span = Corsi Block-Tapping Test (Denburg et al., 1987a; Monastero et al., 2001), WMS-III (Glanz et al., 2005). Information = WAIS (Denburg et al., 1987a), WAIS-R (Glanz et al., 1997). Verbal Learning, Verbal Immediate Recall, Verbal Immediate Cued Recall, Verbal Delayed Recall, Verbal Delayed Cued Recall, Verbal Recognition = RAVLT (Denburg et al., 1987a; Emori et al., 2005; Monastero, 2001), CVLT (Glanz et al., 2005; Kozora et al., 2004; Kozora et al., 2008; Loukkola et al., 2003), CVLT-II (Kozora et al., 2011). Passages Immediate Recall, Delayed Recall = WMS (Denburg et al., 1987a), Logical Memory Immediate Recall, Logical Memory Delayed
Recall, Logical Memory Recognition = WMS-R (Glanz et al., 1997; Loukkola et al., 2003), WMS-III (Glanz et al., 2005). Visual Reproduction Immediate & Delayed Recalls = WMS (Denburg et al., 1987a; Glanz et al., 1997;), WMS-III (Glanz et al., 2005).

Table 5. 
**Eligibility Requirements for Cohort A and Cohort B**

<table>
<thead>
<tr>
<th>SLE Inclusion Criteria</th>
<th>Cohort A</th>
<th>Cohort B</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Must be 18 years of age or older.</td>
<td>2. Must fulfill the current American College of Rheumatology revised criteria for the diagnosis of SLE.</td>
<td>1. Must be ≥18 and ≤55 years of age.</td>
</tr>
<tr>
<td>2. Must fulfill the current American College of Rheumatology revised criteria for the diagnosis of SLE.</td>
<td>3. Must be willing and able to sign informed consent.</td>
<td>2. Must fulfill the current American College of Rheumatology (ACR) revised criteria for the diagnosis of SLE.</td>
</tr>
<tr>
<td>3. Must be willing and able to sign informed consent.</td>
<td>4. Must have stable disease activity and medication doses for 4 weeks prior to screening.</td>
<td>3. Must be willing and able to sign informed consent.</td>
</tr>
<tr>
<td>4. Must have stable disease activity and medication doses for 4 weeks prior to screening.</td>
<td></td>
<td>4. Must have stable disease activity and medication doses for 8 weeks prior to screening.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>SLE Exclusion Criteria</th>
<th>Cohort A</th>
<th>Cohort B</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. History of neurological diseases including head injury resulting in a loss of consciousness, strokes (secondary to hypertension, atherosclerosis, diabetes), seizures, toxic exposure, any difficulties at birth, mental retardation.</td>
<td>2. History of documented transient ischemic attacks within six months of screening.</td>
<td>1. History of neurological diseases including head injury resulting in a loss of consciousness, strokes (secondary to hypertension, atherosclerosis, diabetes), seizures, toxic exposure, any difficulties at birth, mental retardation.</td>
</tr>
<tr>
<td>2. History of documented transient ischemic attacks within six months of screening.</td>
<td>3. Limited fluency with English that in the opinion of the investigator would limit the subject's performance on neuropsychological testing.</td>
<td>2. History of documented transient ischemic attacks within six months of screening.</td>
</tr>
<tr>
<td>3. Limited fluency with English that in the opinion of the investigator would limit the subject's performance on neuropsychological testing.</td>
<td>4. History of illicit drug use (cannabis, heroin) that can result in altered cognition.</td>
<td>3. Currently taking anti-convulsant medication.</td>
</tr>
<tr>
<td>4. History of illicit drug use (cannabis, heroin) that can result in altered cognition.</td>
<td>5. Increased disease activity within 4 weeks defined by an increase in SLEDAl by 3 points or more exclusive of points from serologies.</td>
<td>4. Limited fluency with English that in the opinion of the investigator would limit the subject's performance on the ACR battery of cognitive tests or the N-back task chosen for the working memory task during the PET scan.</td>
</tr>
<tr>
<td>5. Increased disease activity within 4 weeks defined by an increase in SLEDAl by 3 points or more exclusive of points from serologies.</td>
<td>6. Any increase in steroid dose or addition of disease modifying agents within 4 weeks.</td>
<td>5. History of illicit drug use (cannabis, heroin) that can result in altered cognition.</td>
</tr>
<tr>
<td>6. Any increase in steroid dose or addition of disease modifying agents within 4 weeks.</td>
<td>7. History of an anxiety disorder, depression or other psychiatric illness that requires medication.</td>
<td>6. Increased disease activity within 8 weeks defined by an increase in SLEDAl by 3 points or more, exclusive of points from serologies.</td>
</tr>
<tr>
<td>7. History of an anxiety disorder, depression or other psychiatric illness that requires medication.</td>
<td>8. Exceeding the weight limit on the MRI scanner.</td>
<td>7. Any increase in steroid dose or addition of disease modifying agents within 8 weeks.</td>
</tr>
<tr>
<td>8. Exceeding the weight limit on the MRI scanner.</td>
<td>9. Suffering from claustrophobia.</td>
<td>8. Exceeding the weight limit on the MRI scanner.</td>
</tr>
<tr>
<td>9. Suffering from claustrophobia.</td>
<td>10. Have any of the following: cardiac pacemakers, auto defibrillators, neural stimulators, aneurysm clips, metallic prostheses, cochlear implants, any implanted devices (pumps, infusion devices, stents), permanent eye make-up, IUD’s, shrapnel injuries.</td>
<td>9. Suffering from claustrophobia.</td>
</tr>
<tr>
<td>10. Have any of the following: cardiac pacemakers, auto defibrillators, neural stimulators, aneurysm clips, metallic prostheses, cochlear implants, any implanted devices (pumps, infusion devices, stents), permanent eye make-up, IUD’s, shrapnel injuries.</td>
<td>11. Current use of anxiolytic, antidepressant or antipsychotic medications.</td>
<td>10. Have any of the following: cardiac pacemakers, auto defibrillators, neural stimulators, aneurysm clips, metallic prostheses, cochlear implants, any implanted devices (pumps, infusion devices, stents), permanent eye make-up, IUD’s, shrapnel injuries.</td>
</tr>
<tr>
<td>11. Current use of anxiolytic, antidepressant or antipsychotic medications.</td>
<td>12. Pregnant and/or lactating women</td>
<td>11. Current use of anxiolytic, antidepressant or antipsychotic medications.</td>
</tr>
<tr>
<td>12. Pregnant and/or lactating women</td>
<td>13. A glomerular filtration rate less than ≤60 mL/min or any evidence of active renal disease from any cause that would put the subject at risk for increased toxicity from gadolinium contrast for the MRI study.</td>
<td>12. Pregnant and/or lactating women</td>
</tr>
<tr>
<td>13. A glomerular filtration rate less than ≤60 mL/min or any evidence of active renal disease from any cause that would put the subject at risk for increased toxicity from gadolinium contrast for the MRI study.</td>
<td>14. The presence of uncontrolled or severe hypertension, diabetes mellitus or liver disease that would increase the risk of increased toxicity from gadolinium contrast.</td>
<td>13. A glomerular filtration rate less than ≤60 mL/min or any evidence of active renal disease from any cause that would put the subject at risk for increased toxicity from gadolinium contrast for the MRI study.</td>
</tr>
<tr>
<td>14. The presence of uncontrolled or severe hypertension, diabetes mellitus or liver disease that would increase the risk of increased toxicity from gadolinium contrast.</td>
<td></td>
<td>14. The presence of uncontrolled or severe hypertension, diabetes mellitus or liver disease that would increase the risk of increased toxicity from gadolinium contrast.</td>
</tr>
<tr>
<td>HC Inclusion Criteria</td>
<td>HC Exclusion Criteria</td>
<td></td>
</tr>
<tr>
<td>--------------------------------------------------------------------------------------</td>
<td>----------------------------------------------------------------------------------------</td>
<td></td>
</tr>
<tr>
<td>1. Adults that are ≥18 and ≤55 years of age</td>
<td>1. History of neurological diseases including head injury resulting in a loss of consciousness, strokes (secondary to hypertension, atherosclerosis, diabetes), seizures, toxic exposure, any difficulties at birth, mental retardation.</td>
<td></td>
</tr>
<tr>
<td>2. No first degree relatives with SLE or other autoimmune diseases such as rheumatoid arthritis, scleroderma, Sjogren's Syndrome, Multiple Sclerosis</td>
<td>2. History of documented transient ischemic attacks within six months of screening.</td>
<td></td>
</tr>
<tr>
<td>3. Must be willing and able to sign informed consent</td>
<td>3. Currently taking anti-convulsant medication.</td>
<td></td>
</tr>
<tr>
<td>1. History of autoimmune disease</td>
<td>4. Limited fluency with English that in the opinion of the investigator would limit the subject's performance on neuropsychological testing.</td>
<td></td>
</tr>
<tr>
<td>2. First degree relative of a patient with autoimmune disease</td>
<td>5. History of illicit drug use (cocaine, cannabis, heroin) that can result in altered cognition.</td>
<td></td>
</tr>
<tr>
<td>3. History of neurological diseases including head injury resulting in a loss of consciousness, strokes (secondary to hypertension, atherosclerosis, diabetes), seizures, toxic exposure, any difficulties at birth, mental retardation.</td>
<td>6. Exceeding the weight limit on the MRI scanner.</td>
<td></td>
</tr>
<tr>
<td>4. History of documented transient ischemic attacks within six months of screening.</td>
<td>7. Suffering from claustrophobia.</td>
<td></td>
</tr>
<tr>
<td>5. Limited fluency with English that in the opinion of the investigator would limit the subject's performance on neuropsychological testing.</td>
<td>8. Have any of the following: cardiac pacemakers, auto defibrillators, neural stimulators, aneurysm clips, metallic prostheses, cochlear implants, any implanted devices (pumps, infusion devices, stents), permanent eye make-up, IUD's, shrapnel injuries.</td>
<td></td>
</tr>
<tr>
<td>6. History of illicit drug use (cocaine, cannabis, heroin) that can result in altered cognition.</td>
<td>9. Current use of anxiolytic, antidepressant or antipsychotic medications.</td>
<td></td>
</tr>
<tr>
<td>1. Adults that are ≥18 and ≤55 years of age</td>
<td>10. Pregnant and/or lactating women</td>
<td></td>
</tr>
<tr>
<td>2. No first degree relatives with SLE or other autoimmune diseases such as rheumatoid arthritis, scleroderma, Sjogren's Syndrome, Multiple Sclerosis</td>
<td>3. Must be willing and able to sign informed consent</td>
<td></td>
</tr>
<tr>
<td>3. Must be willing and able to sign informed consent</td>
<td>4. Limited fluency with English that in the opinion of the investigator would limit the subject's performance on neuropsychological testing.</td>
<td></td>
</tr>
<tr>
<td>5. History of neurological diseases including head injury resulting in a loss of consciousness, strokes (secondary to hypertension, atherosclerosis, diabetes), seizures, toxic exposure, any difficulties at birth, mental retardation.</td>
<td>6. History of documented transient ischemic attacks within six months of screening.</td>
<td></td>
</tr>
<tr>
<td>7. Suffering from claustrophobia.</td>
<td>8. Have any of the following: cardiac pacemakers, auto defibrillators, neural stimulators, aneurysm clips, metallic prostheses, cochlear implants, any implanted devices (pumps, infusion devices, stents), permanent eye make-up, IUD's, shrapnel injuries.</td>
<td></td>
</tr>
<tr>
<td>8. Current use of anxiolytic, antidepressant or antipsychotic medications.</td>
<td>9. Exceeding the weight limit on the MRI scanner.</td>
<td></td>
</tr>
<tr>
<td>10. Pregnant and/or lactating women</td>
<td>10. Suffering from claustrophobia.</td>
<td></td>
</tr>
</tbody>
</table>
Table 6. 
**Descriptive Demographics of HC and SLE participants for Cohorts A and B**

<table>
<thead>
<tr>
<th></th>
<th>HC (N = 42)</th>
<th>SLE (N = 60)</th>
<th>t</th>
<th>p</th>
<th>Group Differences</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Mean (SD)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age</td>
<td>35.86(12.09)</td>
<td>41.13(10.65)</td>
<td>-2.329</td>
<td>.02*</td>
<td>HC &lt; SLE</td>
</tr>
<tr>
<td>Education (years)</td>
<td>15.05(2.34)</td>
<td>13.66(2.17)</td>
<td>3.04</td>
<td>&lt;.01*</td>
<td>SLE &lt; HC</td>
</tr>
<tr>
<td>BDI</td>
<td>2.86(3.25)</td>
<td>7.53(6.07)</td>
<td>-4.54</td>
<td>&lt;.01*</td>
<td>HC &lt; SLE</td>
</tr>
<tr>
<td>STA-Y</td>
<td>28.95(8.56)</td>
<td>31.27(10.23)</td>
<td>-1.17</td>
<td>.25</td>
<td></td>
</tr>
<tr>
<td>TAI-Y</td>
<td>30.07(6.96)</td>
<td>36.22(9.21)</td>
<td>-3.58</td>
<td>&lt;.01*</td>
<td>HC &lt; SLE</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>N(%)</th>
<th>N(%)</th>
<th>$\chi^2$</th>
<th>p</th>
<th>Group Differences</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gender, females</td>
<td>42(100.00)</td>
<td>58(96.70)</td>
<td>1.43</td>
<td>.51</td>
</tr>
<tr>
<td>Handedness, right^</td>
<td>9(100.00)</td>
<td>17(81.00)</td>
<td>1.98</td>
<td>.29</td>
</tr>
<tr>
<td>Race:</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Caucasian</td>
<td>8(19.04)</td>
<td>7(11.67)</td>
<td>3.28</td>
<td>.53</td>
</tr>
<tr>
<td>Hispanic</td>
<td>6(14.28)</td>
<td>11(18.33)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>African American</td>
<td>23(54.76)</td>
<td>39(65.00)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Asian</td>
<td>2(4.76)</td>
<td>1(1.67)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Other/Unknown</td>
<td>3(7.14)</td>
<td>2(3.33)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Family Autoimmune Hx, yes</td>
<td>9(21.40)</td>
<td>26(43.30)</td>
<td>5.26</td>
<td>.03*</td>
</tr>
<tr>
<td>Hx of Cognitive Dysfunction, yes</td>
<td>6(18.18)</td>
<td>38(65.52)</td>
<td>18.87</td>
<td>&lt;.01*</td>
</tr>
</tbody>
</table>

Note. * = signifies a statistically significant effect at $p < 0.05$ threshold.  
^ = Handedness data was only collected on the 30 Cohort B participants.
Table 7.
**Descriptive Demographics of HC, DNRAb-, and DNRAb+ groups**

<table>
<thead>
<tr>
<th></th>
<th>HC (N = 42)</th>
<th>DNRAb- (N = 34)</th>
<th>DNRAb+ (N = 21)</th>
<th>F/t</th>
<th>p</th>
<th>Group Differences</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Mean (SD)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td><strong>Age</strong></td>
<td>35.86(12.09)</td>
<td>41.97(10.09)</td>
<td>41.48(10.52)</td>
<td>3.24</td>
<td>.04</td>
<td></td>
</tr>
<tr>
<td>Education (years)</td>
<td>15.05(2.35)</td>
<td>13.91(2.29)</td>
<td>13.29(2.13)</td>
<td>4.77</td>
<td>.01*</td>
<td>HC &lt; DNRAb+</td>
</tr>
<tr>
<td>Disease Duration (years)</td>
<td>-</td>
<td>11.82(8.23)</td>
<td>13.67(9.71)</td>
<td>0.57</td>
<td>.45</td>
<td></td>
</tr>
<tr>
<td>BDI</td>
<td>2.86(3.25)</td>
<td>8.32(5.77)</td>
<td>5.95(6.57)</td>
<td>11.15</td>
<td>&lt;.01*</td>
<td>HC &lt; DNRAb-</td>
</tr>
<tr>
<td>STA-Y</td>
<td>28.95(8.56)</td>
<td>30.71(9.78)</td>
<td>31.95(11.39)</td>
<td>0.71</td>
<td>.50</td>
<td></td>
</tr>
<tr>
<td>TAI-Y</td>
<td>30.07(6.96)</td>
<td>37.78(9.22)</td>
<td>32.60(8.05)</td>
<td>8.37</td>
<td>&lt;.01*</td>
<td>HC &lt; DNRAb-</td>
</tr>
<tr>
<td><strong>N(%)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gender, females</td>
<td>42(100.00)</td>
<td>32(94.12)</td>
<td>21(100.00)</td>
<td>3.78</td>
<td>.15</td>
<td></td>
</tr>
<tr>
<td>Handedness, right^</td>
<td>9(100.00)</td>
<td>9(81.82)</td>
<td>8(80.00)</td>
<td>1.99</td>
<td>.37</td>
<td></td>
</tr>
<tr>
<td>Race:</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Caucasian</td>
<td>8(19.05)</td>
<td>4(11.76)</td>
<td>3(14.29)</td>
<td>13.30</td>
<td>.10</td>
<td></td>
</tr>
<tr>
<td>Hispanic</td>
<td>6(14.29)</td>
<td>2(5.88)</td>
<td>8(38.10)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>African American</td>
<td>23(54.76)</td>
<td>25(73.53)</td>
<td>10(42.86)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Asian</td>
<td>2(4.76)</td>
<td>1(2.94)</td>
<td>0(0.00)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Other/Unknown</td>
<td>3(7.14)</td>
<td>2(5.88)</td>
<td>0(0.00)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Family Autoimmune Hx, yes</td>
<td>9(21.43)</td>
<td>17(50.00)</td>
<td>8(38.10)</td>
<td>6.85</td>
<td>.03*</td>
<td>HC &lt; DNRAb-</td>
</tr>
<tr>
<td>Hx of Cognitive Dysfunction, yes</td>
<td>6(18.18)</td>
<td>21(61.76)</td>
<td>15(71.43)</td>
<td>18.96</td>
<td>&lt;.01*</td>
<td>HC &lt; DNRAb-, +</td>
</tr>
</tbody>
</table>

Note. * = signifies a statistically significant effect at \( p < 0.05 \) threshold. Group comparisons for Family Autoimmune History were not significant. ^ = Handedness data was only collected on the 30 Cohort B participants.
Table 8. 
Neuropsychological Performances as a Function of Group

<table>
<thead>
<tr>
<th></th>
<th>HC (N = 34)</th>
<th>SLE (N = 60)</th>
<th>t</th>
<th>p</th>
<th>Group Differences</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Judgment of Line Orientation (JLO)</strong>^</td>
<td>19.33 (6.02)</td>
<td>16.81 (4.58)</td>
<td>1.26</td>
<td>0.22</td>
<td>-</td>
</tr>
<tr>
<td>JLO z-score</td>
<td>-0.61 (1.06)</td>
<td>-0.118 (0.98)</td>
<td>1.41</td>
<td>0.17</td>
<td>-</td>
</tr>
<tr>
<td><strong>Trails A (sec)</strong></td>
<td>30.24 (11.00)</td>
<td>38.89 (14.36)</td>
<td>-3.01</td>
<td>&lt;0.01*</td>
<td>HC &lt; SLE</td>
</tr>
<tr>
<td>Trails A z-score</td>
<td>-0.49 (1.35)</td>
<td>-1.13 (1.78)</td>
<td>1.79</td>
<td>0.08</td>
<td>-</td>
</tr>
<tr>
<td><strong>Trails B (sec)</strong></td>
<td>65.29 (28.78)</td>
<td>125.31 (82.71)</td>
<td>-4.07</td>
<td>&lt;0.01*</td>
<td>HC &lt; SLE</td>
</tr>
<tr>
<td>Trails B z-score</td>
<td>-0.79 (2.12)</td>
<td>-4.68 (6.82)</td>
<td>3.23</td>
<td>&lt;0.01*</td>
<td>SLE &lt; HC</td>
</tr>
<tr>
<td><strong>Rey Copy</strong></td>
<td>32.32 (3.99)</td>
<td>28.00 (5.96)</td>
<td>3.74</td>
<td>&lt;0.01*</td>
<td>SLE &lt; HC</td>
</tr>
<tr>
<td>Rey Copy z-score</td>
<td>-1.61 (2.51)</td>
<td>-4.19 (4.70)</td>
<td>2.95</td>
<td>&lt;0.01*</td>
<td>SLE &lt; HC</td>
</tr>
<tr>
<td><strong>Rey Delay</strong></td>
<td>16.79 (5.68)</td>
<td>14.91 (5.66)</td>
<td>1.52</td>
<td>0.13</td>
<td>-</td>
</tr>
<tr>
<td>Rey Delay z-score</td>
<td>-1.45 (1.30)</td>
<td>-1.41 (1.27)</td>
<td>-0.15</td>
<td>0.88</td>
<td>-</td>
</tr>
</tbody>
</table>

Note: ^JLO task N’s: HC = 9, DNRAb- =11, DNRAb+ = 10
<table>
<thead>
<tr>
<th>Neuropsychological Performances as a Function of Group</th>
</tr>
</thead>
<tbody>
<tr>
<td>HC (N = 34)</td>
</tr>
<tr>
<td>--------------</td>
</tr>
<tr>
<td><strong>M (SD)</strong></td>
</tr>
<tr>
<td>Judgment of Line Orientation (JLO)^</td>
</tr>
<tr>
<td>JLO z-score</td>
</tr>
<tr>
<td>Trails A (sec)</td>
</tr>
<tr>
<td>Trails A z-score</td>
</tr>
<tr>
<td>Trails B (sec)</td>
</tr>
<tr>
<td>Trails B z-score</td>
</tr>
<tr>
<td>Rey Copy</td>
</tr>
<tr>
<td>Rey Copy z-score</td>
</tr>
<tr>
<td>Rey Delay</td>
</tr>
<tr>
<td>Rey Delay z-score</td>
</tr>
</tbody>
</table>

Note: ^JLO task N's: HC = 9, DNRAb- = 11, DNRAb+ = 10
Table 10. *Proportion of Correct Responses on the Spatial Memory Task as a Function of Array Difficulty, Memory Type, and Group*

<table>
<thead>
<tr>
<th></th>
<th>HC (N = 42)</th>
<th>SLE (N = 60)</th>
<th>DNRAb- (N = 34)</th>
<th>DNRAb+ (N = 21)</th>
</tr>
</thead>
<tbody>
<tr>
<td>M (SD)</td>
<td>M (SD)</td>
<td>M (SD)</td>
<td>M (SD)</td>
<td></td>
</tr>
<tr>
<td>2x2 Nonspatial</td>
<td>0.94 (0.10)</td>
<td>0.93 (0.10)</td>
<td>0.93 (0.10)</td>
<td>0.94 (0.10)</td>
</tr>
<tr>
<td>3x2 Nonspatial</td>
<td>0.88 (0.11)</td>
<td>0.84 (0.12)</td>
<td>0.86 (0.08)</td>
<td>0.79 (0.11)</td>
</tr>
<tr>
<td>2x2: Spatial</td>
<td>0.84 (0.11)</td>
<td>0.76 (0.13)</td>
<td>0.79 (0.12)</td>
<td>0.72 (0.11)</td>
</tr>
<tr>
<td>3x2: Spatial</td>
<td>0.76 (0.13)</td>
<td>0.67 (0.13)</td>
<td>0.67 (0.14)</td>
<td>0.68 (0.09)</td>
</tr>
<tr>
<td></td>
<td>HC (N = 9)</td>
<td>SLE (N = 20)</td>
<td>DNRAb- (N = 10)</td>
<td>DNRAb+ (N = 10)</td>
</tr>
<tr>
<td>--------------------------</td>
<td>------------</td>
<td>--------------</td>
<td>-----------------</td>
<td>-----------------</td>
</tr>
<tr>
<td>Whole Task Total Correct</td>
<td>0.79 (0.09)</td>
<td>0.66 (0.08)</td>
<td>0.66 (0.07)</td>
<td>0.67 (0.10)</td>
</tr>
<tr>
<td>Block 1</td>
<td>0.71 (0.10)</td>
<td>0.60 (0.08)</td>
<td>0.60 (0.07)</td>
<td>0.58 (0.09)</td>
</tr>
<tr>
<td>Block 2</td>
<td>0.79 (0.12)</td>
<td>0.67 (0.12)</td>
<td>0.67 (0.11)</td>
<td>0.68 (0.13)</td>
</tr>
<tr>
<td>Block 3</td>
<td>0.86 (0.10)</td>
<td>0.73 (0.12)</td>
<td>0.71 (0.11)</td>
<td>0.75 (0.14)</td>
</tr>
<tr>
<td>Spoiler Items</td>
<td>0.99 (0.03)</td>
<td>0.98 (0.05)</td>
<td>0.98 (0.04)</td>
<td>0.97 (0.06)</td>
</tr>
<tr>
<td>Target Items</td>
<td>0.79 (0.11)</td>
<td>0.61 (0.23)</td>
<td>0.62 (0.22)</td>
<td>0.59 (0.24)</td>
</tr>
<tr>
<td>Shape Items</td>
<td>0.69 (0.18)</td>
<td>0.63 (0.23)</td>
<td>0.61 (0.25)</td>
<td>0.65 (0.21)</td>
</tr>
<tr>
<td>Location Items</td>
<td>0.69 (0.27)</td>
<td>0.56 (0.26)</td>
<td>0.50 (0.30)</td>
<td>0.62 (0.22)</td>
</tr>
<tr>
<td>Learning over blocks</td>
<td>3.67 (2.24)</td>
<td>3.65 (3.17)</td>
<td>3.00 (3.02)</td>
<td>4.30 (3.34)</td>
</tr>
</tbody>
</table>

Note: Location Items - indicates that the location of one of the shapes changed. Shape Items - indicates that one of the shapes changed. Spoiler items – one of the items is a novel shape. Learning computed by the [(Maximum of Trial 2 or 3 Raw Total Correct) – Trial 1]
Table 12.  
*Correlations between demographic variables and proportion of accurate responses on the Spatial Memory Task*

<table>
<thead>
<tr>
<th>Age</th>
<th>Education (years)</th>
<th>Disease Duration (years)</th>
<th>Memory Task - 2x2 Nonspatial Accuracy Proportion</th>
<th>Memory Task - 3x2 Nonspatial Accuracy Proportion</th>
<th>Memory Task - 2x2 Spatial Accuracy Proportion</th>
<th>Memory Task - 3x2 Spatial Accuracy Proportion</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pearson Correlation</td>
<td>1.00</td>
<td>-.10</td>
<td>.34**</td>
<td>-.19</td>
<td>-.19</td>
<td>-.27**</td>
</tr>
<tr>
<td>Sig. (2-tailed)</td>
<td></td>
<td></td>
<td>.32</td>
<td>.01</td>
<td>.06</td>
<td>.05</td>
</tr>
<tr>
<td>N</td>
<td>102</td>
<td>100</td>
<td>60</td>
<td>102</td>
<td>102</td>
<td>102</td>
</tr>
<tr>
<td>Education (years)</td>
<td>Pearson Correlation</td>
<td>-.10</td>
<td>1.00</td>
<td>-.12</td>
<td>.26**</td>
<td>.20</td>
</tr>
<tr>
<td>Sig. (2-tailed)</td>
<td>.32</td>
<td>.35</td>
<td>.01</td>
<td>.05</td>
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<td>&lt;.01</td>
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<tr>
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<td>100</td>
<td>59</td>
<td>100</td>
<td>100</td>
<td>100</td>
</tr>
<tr>
<td>Disease Duration (years)</td>
<td>Pearson Correlation</td>
<td>.34**</td>
<td>-.12</td>
<td>1.00</td>
<td>.04</td>
<td>-.30*</td>
</tr>
<tr>
<td>Sig. (2-tailed)</td>
<td>.01</td>
<td>.35</td>
<td>.75</td>
<td>.02</td>
<td>.01</td>
<td>.34</td>
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<td>59</td>
<td>60</td>
<td>60</td>
<td>60</td>
<td>60</td>
</tr>
<tr>
<td>Memory Task - 2x2 Nonspatial Accuracy Proportion</td>
<td>Pearson Correlation</td>
<td>-.19</td>
<td>.26**</td>
<td>-.04</td>
<td>1.00</td>
<td>.08</td>
</tr>
<tr>
<td>Sig. (2-tailed)</td>
<td>.06</td>
<td>.01</td>
<td>.75</td>
<td>.42</td>
<td>&lt;.01</td>
<td>&lt;.01</td>
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<td>100</td>
<td>60</td>
<td>104</td>
<td>104</td>
<td>104</td>
</tr>
<tr>
<td>Memory Task - 3x2 Nonspatial Accuracy Proportion</td>
<td>Pearson Correlation</td>
<td>-.19</td>
<td>.20</td>
<td>-.30*</td>
<td>.08</td>
<td>1.00</td>
</tr>
<tr>
<td>Sig. (2-tailed)</td>
<td>.05</td>
<td>.05</td>
<td>.02</td>
<td>.42</td>
<td>.01</td>
<td>&lt;.01</td>
</tr>
<tr>
<td>N</td>
<td>102</td>
<td>100</td>
<td>60</td>
<td>104</td>
<td>104</td>
<td>104</td>
</tr>
<tr>
<td>Memory Task - 2x2 Spatial Accuracy Proportion</td>
<td>Pearson Correlation</td>
<td>-.27**</td>
<td>.38**</td>
<td>-.34*</td>
<td>.36**</td>
<td>.24*</td>
</tr>
<tr>
<td>Sig. (2-tailed)</td>
<td>.01</td>
<td>&lt;.01</td>
<td>.01</td>
<td>&lt;.01</td>
<td>.01</td>
<td>&lt;.01</td>
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<tr>
<td>N</td>
<td>102</td>
<td>100</td>
<td>60</td>
<td>104</td>
<td>104</td>
<td>104</td>
</tr>
<tr>
<td>Memory Task - 3x2 Spatial Accuracy Proportion</td>
<td>Pearson Correlation</td>
<td>-.36**</td>
<td>.39**</td>
<td>-.13</td>
<td>.35**</td>
<td>.33**</td>
</tr>
<tr>
<td>Sig. (2-tailed)</td>
<td>&lt;.01</td>
<td>&lt;.01</td>
<td>.34</td>
<td>&lt;.01</td>
<td>&lt;.01</td>
<td>&lt;.01</td>
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<tr>
<td>N</td>
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<td>100</td>
<td>60</td>
<td>104</td>
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</tbody>
</table>

**. Correlation is significant at the 0.01 level (2-tailed).
*. Correlation is significant at the 0.05 level (2-tailed).
Table 13.
Correlations between affective inventories and proportion of accurate responses on the Spatial Memory Task

<table>
<thead>
<tr>
<th></th>
<th>Memory Task - 2x2 Nonspatial Accuracy Proportion</th>
<th>Memory Task - 3x2 Nonspatial Accuracy Proportion</th>
<th>Memory Task - 2x2 Spatial Accuracy Proportion</th>
<th>Memory Task - 3x2 Spatial Accuracy Proportion</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>BDI</strong></td>
<td><strong>Pearson Correlation</strong></td>
<td><strong>Sig. (2-tailed)</strong></td>
<td><strong>N</strong></td>
<td><strong>N</strong></td>
</tr>
<tr>
<td></td>
<td>1.00</td>
<td>.55**</td>
<td>.72**</td>
<td>-.17</td>
</tr>
<tr>
<td></td>
<td><strong>&lt;.01</strong></td>
<td><strong>&lt;.01</strong></td>
<td>.36</td>
<td>-.21*</td>
</tr>
<tr>
<td></td>
<td>101</td>
<td>95</td>
<td>96</td>
<td>101</td>
</tr>
<tr>
<td><strong>STA-Y</strong></td>
<td><strong>Pearson Correlation</strong></td>
<td><strong>Sig. (2-tailed)</strong></td>
<td><strong>N</strong></td>
<td><strong>N</strong></td>
</tr>
<tr>
<td></td>
<td>.55**</td>
<td>.100</td>
<td>.58**</td>
<td>-.21*</td>
</tr>
<tr>
<td></td>
<td><strong>&lt;.01</strong></td>
<td><strong>&lt;.01</strong></td>
<td>.81</td>
<td>-.13</td>
</tr>
<tr>
<td></td>
<td>95</td>
<td>95</td>
<td>91</td>
<td>95</td>
</tr>
<tr>
<td><strong>TAI-Y</strong></td>
<td><strong>Pearson Correlation</strong></td>
<td><strong>Sig. (2-tailed)</strong></td>
<td><strong>N</strong></td>
<td><strong>N</strong></td>
</tr>
<tr>
<td></td>
<td>.72**</td>
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**. Correlation is significant at the 0.01 level (2-tailed).

*. Correlation is significant at the 0.05 level (2-tailed).
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*. Correlation is significant at the 0.05 level (2-tailed).

**. Correlation is significant at the 0.01 level (2-tailed).
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**. Correlation is significant at the 0.01 level (2-tailed).
* Correlation is significant at the 0.05 level (2-tailed).
### Table 16.

**Correlations between neuropsychological tests and proportion of accurate responses on the Spatial Memory Task**

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**Notes:**
- *Correlation is significant at the 0.05 level (2-tailed).
- **Correlation is significant at the 0.01 level (2-tailed).**

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Table 17. Correlations between ANAM cognitive scales and proportion of accurate responses on the Spatial Memory Test: Visuospatial Tasks

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**. Correlation is significant at the 0.01 level (2-tailed).
*. Correlation is significant at the 0.05 level (2-tailed).
## Table 18.
Correlations between ANAM cognitive scales and proportion of accurate responses on the Spatial Memory Test: Simple RT and Running Memory CPT

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**. Correlation is significant at the 0.01 level (2-tailed).
*. Correlation is significant at the 0.05 level (2-tailed).
Table 19. Correlations between ANAM cognitive scales and proportion of accurate responses on the Spatial Memory Test: Go/No-Go

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<th>Go/No-Go (ANAM) - Hits</th>
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* Correlation is significant at the 0.05 level (2-tailed).
** Correlation is significant at the 0.01 level (2-tailed).
Table 20.
Correlations between demographic variables and Block 1 of the Relational Learning Task

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<th>Relational Learning - Block 1 Shape Accuracy Proportion</th>
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**. Correlation is significant at the 0.01 level (2-tailed).
*. Correlation is significant at the 0.05 level (2-tailed).
### Table 21.

**Correlations between demographic variables and Block 2 of the Relational Learning Task**

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**. Correlation is significant at the 0.05 level (2-tailed).

**. Correlation is significant at the 0.01 level (2-tailed).
### Table 22.
Correlations between demographic variables and Block 3 of the Relational Learning Task

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<sup>**</sup>. Correlation is significant at the 0.01 level (2-tailed).
<sup>*</sup>. Correlation is significant at the 0.05 level (2-tailed).
### Table 23.
**Correlations between demographic variables and the whole Relational Learning Task**

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<th>Education (Years)</th>
<th>Disease Duration (Years)</th>
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<th>Relational Task - Wrong Shape Accuracy Proportion</th>
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**. Correlation is significant at the 0.05 level (2-tailed).**

**Correlation is significant at the 0.01 level (2-tailed).**

^Learning = (Max Trial 2 or 3 - Trial 1)
Table 24.
Correlations between affective inventories and Block 1 of the Relational Learning Task

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** Correlation is significant at the 0.01 level (2-tailed).
Table 25.  
Correlations between affective inventories and Block 2 of the Relational Learning Task

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** Correlation is significant at the 0.01 level (2-tailed).
* Correlation is significant at the 0.05 level (2-tailed).
Table 26.  
Correlations between affective inventories and Block 3 of the Relational Learning Task

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**. Correlation is significant at the 0.01 level (2-tailed).  
*. Correlation is significant at the 0.05 level (2-tailed).
### Table 27.

**Correlations between affective inventories and the whole Relational Learning Task**

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**. Correlation is significant at the 0.01 level (2-tailed).
*. Correlation is significant at the 0.05 level (2-tailed).
### Table 28.

**Correlations between ANAM sleep scales and Block 1 of the Relational Learning Task**

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∗Correlation is significant at the 0.05 level (2-tailed).

∗∗Correlation is significant at the 0.01 level (2-tailed).
Table 29. Correlations between ANAM sleep scales and Block 2 of the Relational Learning Task

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* Correlation is significant at the 0.05 level (2-tailed).
** Correlation is significant at the 0.01 level (2-tailed).
Table 30.
Correlations between ANAM sleep scales and Block 3 of the Relational Learning Task

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* Correlation is significant at the 0.05 level (2-tailed).
** Correlation is significant at the 0.01 level (2-tailed).
### Table 31.

**Correlations between ANAM sleep scales and the whole Relational Learning Task**

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* Correlation is significant at the 0.05 level (2-tailed).
** Correlation is significant at the 0.01 level (2-tailed).
## Table 32.

**Correlations between ANAM mood scales and Block 1 of the Relational Learning Task**

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<th>Mood Scale (ANAM) - Anger</th>
<th>Mood Scale (ANAM) - Anxiety</th>
<th>Mood Scale (ANAM) - Depression</th>
<th>Mood Scale (ANAM) - Happiness</th>
<th>Mood Scale (ANAM) - Vigor</th>
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<th>Relational Learning - Block 1 Relation Accuracy Proportion</th>
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**Note:** Correlation is significant at the 0.01 level (2-tailed).
### Table 33.
Correlations between ANAM mood scales and Block 2 of the Relational Learning Task

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<th>Mood Scale (ANAM) - Depression</th>
<th>Mood Scale (ANAM) - Happiness</th>
<th>Mood Scale (ANAM) - Vigor</th>
<th>Relational Learning - Block 2 Target Accuracy Proportion</th>
<th>Relational Learning - Block 2 Relation Accuracy Proportion</th>
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<td><strong>.81</strong> (p &lt; .01)</td>
<td><strong>.98</strong> (p &lt; .01)</td>
<td><strong>.92</strong> (p &lt; .01)</td>
<td><strong>.92</strong> (p &lt; .01)</td>
<td><strong>.67</strong> (p &lt; .01)</td>
<td><strong>.88</strong> (p &lt; .01)</td>
<td><strong>.92</strong> (p &lt; .01)</td>
<td><strong>.92</strong> (p &lt; .01)</td>
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**. Correlation is significant at the 0.01 level (2-tailed).
* Correlation is significant at the 0.05 level (2-tailed).
Table 34. Correlations between ANAM mood scales and Block 3 of the Relational Learning Task

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<th>Mood Scale (ANAM) - Happiness</th>
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<th>Relational Learning - Block 3 Relation Accuracy Proportion</th>
<th>Relational Learning - Block 3 Shape Accuracy Proportion</th>
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**. Correlation is significant at the 0.01 level (2-tailed).
*. Correlation is significant at the 0.05 level (2-tailed).
Table 35. **Correlations between ANAM mood scales and the whole Relational Learning Task**

<table>
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<tr>
<th></th>
<th>Mood Scale (ANAM) - Anger</th>
<th>Mood Scale (ANAM) - Anxiety</th>
<th>Mood Scale (ANAM) - Depression</th>
<th>Mood Scale (ANAM) - Happiness</th>
<th>Mood Scale (ANAM) - Vigor</th>
<th>Relational Task - Wrong Location Accuracy Proportion</th>
<th>Relational Task - Wrong Shape Accuracy Proportion</th>
<th>Relational Task - Spoiler Accuracy Proportion</th>
<th>Relational Task - Target Accuracy Proportion</th>
<th>Relational Task - Learning Accuracy Proportion</th>
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<td>Pearson Correlation: .63*</td>
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<td>Pearson Correlation: .09</td>
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<td>.22</td>
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<td>Pearson Correlation: -.04</td>
<td>Sig. (2-tailed): &lt;.01</td>
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<td>.03</td>
<td>-.08</td>
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<td>Pearson Correlation: -.11</td>
<td>Sig. (2-tailed): &lt;.01</td>
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<td>-.26</td>
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</table>

**. Correlation is significant at the 0.01 level (2-tailed).
*. Correlation is significant at the 0.05 level (2-tailed).
Table 36.  
**Correlations between neuropsychological tests and Block 1 of the Relational Learning Task**

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<th>RCFT Delay</th>
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<td>&lt;.01</td>
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*Correlation is significant at the 0.05 level (2-tailed).
**Correlation is significant at the 0.01 level (2-tailed).
Table 37.
Correlations between neuropsychological tests and Block 2 of the Relational Learning Task

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* Correlation is significant at the 0.05 level (2-tailed).
** Correlation is significant at the 0.01 level (2-tailed).
### Table 38.

**Correlations between neuropsychological tests and Block 3 of the Relational Learning Task**

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<th>Relational Learning - Block 3 Relation Accuracy Proportion</th>
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*Correlation is significant at the 0.05 level (2-tailed).**Correlation is significant at the 0.01 level (2-tailed).
Table 39.
Correlations between neuropsychological tests and the whole Relational Learning Task

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*. Correlation is significant at the 0.05 level (2-tailed).
**. Correlation is significant at the 0.01 level (2-tailed).
Table 40.
Correlations between ANAM cognitive scales and proportion of accurate responses on Block 1 of the Relational Learning Task: Visuospatial Tasks

<table>
<thead>
<tr>
<th>Matching Grids (ANAM)</th>
<th>Correlation</th>
<th>Sig. (2-tailed)</th>
<th>N</th>
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<td>.24</td>
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<td>.11</td>
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<tr>
<td>Match to Sample (ANAM) - Accuracy</td>
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<td>.35</td>
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<tr>
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<td>.10</td>
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<td>Relational Learning - Block 1 Target</td>
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<td>Relational Learning - Block 1 Shape</td>
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<td>Relational Learning - Block 1 Spoi</td>
<td>Pearson Correlation</td>
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<td>.25</td>
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| **Correlation is significant at the 0.01 level (2-tailed).**
| *Correlation is significant at the 0.05 level (2-tailed).**
Table 41. 
Correlations between ANAM cognitive scales and proportion of accurate responses on Block 2 of the Relational Learning Task: Visuospatial Tasks

|                      | Matching Grids (ANAM) - Mean RT | Matching Grids (ANAM) - Accuracy | Matching Grids (ANAM) - Throughput | Match to Sample (ANAM) - Mean RT | Match to Sample (ANAM) - Accuracy | Match to Sample (ANAM) - Throughput | Relational Learning - Block 2 Target Accuracy Proportion | Relational Learning - Block 2 Relation Accuracy Proportion | Relational Learning - Block 2 Shape Accuracy Proportion | Relational Learning - Block 2 Spoiler Accuracy Proportion | Relational Task - Block 2 Total Accuracy Proportion |
|----------------------|---------------------------------|---------------------------------|-----------------------------------|----------------------------------|----------------------------------|-----------------------------------|-------------------------------------|-------------------------------------------------|-------------------------------------------------|-------------------------------------------------|-------------------------------------------------|-------------------------------------------------|
| Matching Grids (ANAM) - Mean RT | Pearson Correlation | .100 | -.02 | .85* | .78* | -.17 | -.57* | .07 | -.35 | -.30 | -.06 | -.14 |
| Sig. (2-tailed)     |                                 | .91 | <.01 | <.01 | .37 | <.01 | .72 | .06 | .12 | .76 | .46 |
| N                   |                                 | 30 | 30 | 30 | 30 | 30 | 30 | 29 | 29 | 29 | 29 | 29 |
| Matching Grids (ANAM) - Accuracy | Pearson Correlation | -.02 | 1.00 | .24 | .11 | .35 | .10 | -.13 | .12 | .24 | -.15 | <.01 |
| Sig. (2-tailed)     |                                 | .91 | .21 | .55 | .06 | .61 | .51 | .54 | .21 | .43 | .98 |
| N                   |                                 | 32 | 30 | 30 | 30 | 30 | 30 | 29 | 29 | 29 | 29 | 29 |
| Matching Grids (ANAM) - Throughput | Pearson Correlation | -.85* | .24 | 1.00 | -.69* | .28 | .69* | .08 | .24 | .18 | .08 | .22 |
| Sig. (2-tailed)     |                                 | <.01 | .21 | <.01 | .14 | <.01 | .68 | .22 | .35 | .66 | .24 |
| N                   |                                 | 30 | 30 | 30 | 30 | 30 | 30 | 29 | 29 | 29 | 29 | 29 |
| Match to Sample (ANAM) - Mean RT | Pearson Correlation | .78* | .11 | -.69* | 1.00 | -.16 | -.78* | -.20 | -.23 | -.01 | .05 | -.26 |
| Sig. (2-tailed)     |                                 | <.01 | .55 | <.01 | .39 | <.01 | .29 | .23 | .96 | .81 | .18 |
| N                   |                                 | 32 | 30 | 30 | 30 | 30 | 30 | 29 | 29 | 29 | 29 | 29 |
| Match to Sample (ANAM) - Accuracy | Pearson Correlation | -.17 | .35 | .28 | -.16 | 1.00 | -.60 | .29 | .50 | .01 | -.20 | .48* |
| Sig. (2-tailed)     |                                 | .37 | .05 | .14 | .39 | <.01 | .13 | .01 | .94 | .30 | .01 |
| N                   |                                 | 30 | 30 | 30 | 30 | 30 | 30 | 29 | 29 | 29 | 29 | 29 |
| Match to Sample (ANAM) - Throughput | Pearson Correlation | -.57* | .10 | .69* | -.78* | .60* | 1.00 | .36 | .34 | -.01 | .24 | .45* |
| Sig. (2-tailed)     |                                 | <.01 | .61 | <.01 | <.01 | <.01 | .06 | .07 | .97 | .22 | .01 |
| N                   |                                 | 30 | 30 | 30 | 30 | 30 | 30 | 29 | 29 | 29 | 29 | 29 |
| Relational Learning - Block 2 Target Accuracy Proportion | Pearson Correlation | .07 | -.13 | .08 | -.20 | .29 | .36 | 1.00 | -.02 | -.43 | .01 | .79* |
| Sig. (2-tailed)     |                                 | .72 | .51 | .68 | .29 | .13 | .06 | .97 | .82 | <.01 | .96 |
| N                   |                                 | 29 | 29 | 29 | 29 | 29 | 29 | 29 | 29 | 29 | 29 | 29 |
| Relational Learning - Block 2 Relation Accuracy Proportion | Pearson Correlation | -.35 | .12 | .24 | -.23 | .50* | .34 | -.02 | 1.00 | .41 | .04 | .53 |
| Sig. (2-tailed)     |                                 | .06 | .54 | .22 | .23 | .01 | .07 | .92 | .03 | .84 | <.01 |
| N                   |                                 | 29 | 29 | 29 | 29 | 29 | 29 | 29 | 29 | 29 | 29 | 29 |
| Relational Learning - Block 2 Shape Accuracy Proportion | Pearson Correlation | -.30 | .24 | .18 | -.01 | .01 | -.01 | -.43 | .41 | 1.00 | .05 | .08 |
| Sig. (2-tailed)     |                                 | .12 | .21 | .35 | .96 | .94 | .97 | .02 | .03 | .81 | .66 |
| N                   |                                 | 29 | 29 | 29 | 29 | 29 | 29 | 29 | 29 | 29 | 29 | 29 |
| Relational Learning - Block 2 Spoiler Accuracy Proportion | Pearson Correlation | -.06 | -.15 | .08 | -.05 | -.20 | -.24 | .01 | .04 | .05 | 1.00 | .13 |
| Sig. (2-tailed)     |                                 | .76 | .43 | .66 | .81 | .30 | .22 | .96 | .84 | .81 | .52 |
| N                   |                                 | 29 | 29 | 29 | 29 | 29 | 29 | 29 | 29 | 29 | 29 | 29 |
| Relational Task - Block 2 Total Accuracy Proportion | Pearson Correlation | -.14 | <.01 | .22 | -.26 | .48* | .45* | .79* | .53* | .08 | .13 | 1.00 |
| Sig. (2-tailed)     |                                 | .46 | .98 | .24 | .18 | .01 | .01 | <.01 | <.01 | .66 | .52 |
| N                   |                                 | 29 | 29 | 29 | 29 | 29 | 29 | 29 | 29 | 29 | 29 | 29 |

**. Correlation is significant at the 0.01 level (2-tailed).
* Correlation is significant at the 0.05 level (2-tailed).
## Table 42.
Correlations between ANAM cognitive scales and proportion of accurate responses on Block 3 of the Relational Learning Task: Visuospatial Tasks

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<tr>
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<th>Matching Grids (ANAM) - Accuracy</th>
<th>Match to Sample (ANAM) - Throughput</th>
<th>Match to Sample (ANAM) - Accuracy</th>
<th>Relational Learning - Block 3 Target Accuracy Proportion</th>
<th>Relational Learning - Block 3 Relation Accuracy Proportion</th>
<th>Relational Learning - Block 3 Shape Accuracy Proportion</th>
<th>Relational Learning - Block 3 Spoiler Accuracy Proportion</th>
<th>Relational Task - Block 3 Total Accuracy Proportion</th>
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<td>-.85**</td>
<td>.78*</td>
<td>-.17</td>
<td>-.57**</td>
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**. Correlation is significant at the 0.01 level (2-tailed).
*Correlation is significant at the 0.05 level (2-tailed).
Table 43.
Correlations between ANAM cognitive scales and proportion of accurate responses on the whole Relational Learning Task: Visuospatial Tasks

<table>
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<tr>
<th></th>
<th>Matching Grids (ANAM) - Mean RT</th>
<th>Matching Grids (ANAM) - Accuracy</th>
<th>Match to Sample (ANAM) - Throughput</th>
<th>Match to Sample (ANAM) - Accuracy</th>
<th>Relational Task - Wrong Shape Accuracy Proportion</th>
<th>Relational Task - Wrong ShapeSpoiler Accuracy Proportion</th>
<th>Relational Task - Whole Task - Learning Accuracy Proportion</th>
<th>Relational Task - Whole Task - Accuracy Proportion</th>
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<td>.78&lt;sup&gt;**&lt;/sup&gt;</td>
<td>-.17</td>
<td>-.57&lt;sup&gt;**&lt;/sup&gt;</td>
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<sup>**</sup> Correlation is significant at the 0.01 level (2-tailed).

<sup>*</sup> Correlation is significant at the 0.05 level (2-tailed).
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**. Correlation is significant at the 0.01 level (2-tailed).
* . Correlation is significant at the 0.05 level (2-tailed).
Table 45.
Correlations between ANAM cognitive scales and proportion of accurate responses on Block 2 of the Relational Learning Task: Simple RT and Running Memory CPT

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<td>.02</td>
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<td>Sig. (2-tailed)</td>
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<td>.99</td>
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<td>&lt;.01</td>
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<sup>**</sup> Correlation is significant at the 0.01 level (2-tailed).
<sup>*</sup> Correlation is significant at the 0.05 level (2-tailed).
Table 46.  
Correlations between ANAM cognitive scales and proportion of accurate responses on Block 3 of the Relational Learning Task: Simple RT and Running Memory CPT

<table>
<thead>
<tr>
<th></th>
<th>Simple RT (ANAM) - Mean RT</th>
<th>Simple RT (ANAM) - Throughput</th>
<th>Running Memory CPT (ANAM) - Mean RT</th>
<th>Running Memory CPT (ANAM) - Throughput</th>
<th>Relational Learning - Block 3 Target Proportion</th>
<th>Relational Learning - Block 3 Relation Proportion</th>
<th>Relational Learning - Block 3 Shape Proportion</th>
<th>Relational Learning - Block 3 Spoiler Proportion</th>
<th>Relational Task - Block 3 Total Accuracy Proportion</th>
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<td>Pearson Correlation .47</td>
<td>Sig. (2-tailed) .05</td>
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<tr>
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<td>Pearson Correlation -.97**</td>
<td>Sig. (2-tailed) &lt;.01</td>
<td>Pearson Correlation .24</td>
<td>Sig. (2-tailed) .09</td>
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<td>Sig. (2-tailed) .36</td>
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<td>.25</td>
<td>.03</td>
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<td>Sig. (2-tailed) &lt;.01</td>
<td>Pearson Correlation .78</td>
<td>Sig. (2-tailed) .03</td>
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<td>.73**</td>
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<td>N 29</td>
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</table>

**. Correlation is significant at the 0.01 level (2-tailed).
*. Correlation is significant at the 0.05 level (2-tailed).
Table 47.
Correlations between ANAM cognitive scales and proportion of accurate responses on the whole Relational Learning Task: Simple RT and Running Memory CPT

<table>
<thead>
<tr>
<th></th>
<th>Simple RT (ANAM) - Mean RT</th>
<th>Simple RT (ANAM) - Throughput</th>
<th>Running Memory CPT (ANAM) - Mean RT</th>
<th>Running Memory CPT (ANAM) - Accuracy</th>
<th>Running Memory CPT (ANAM) - Throughput</th>
<th>Relational Task - Wrong Location Accuracy Proportion</th>
<th>Relational Task - Wrong Shape Accuracy Proportion</th>
<th>Relational Task - Spoiler Accuracy Proportion</th>
<th>Relational Task - Target Accuracy Proportion</th>
<th>Relational Task - Learning</th>
<th>Relational Task - Whole Task Accuracy Proportion</th>
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<tbody>
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<td>Pearson Correlation 1.00</td>
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<td>-.06</td>
<td>-.03</td>
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<td>-.28</td>
<td>-.12</td>
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<td>-.09</td>
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* Correlation is significant at the 0.01 level (2-tailed).
* Correlation is significant at the 0.05 level (2-tailed).
### Table 48.
Correlations between ANAM cognitive scales and proportion of accurate responses on Block 1 of the Relational Learning Task: Go/No-Go

<table>
<thead>
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<th></th>
<th>Go/No-Go (ANAM) - Mean RT</th>
<th>Go/No-Go (ANAM) - Accuracy</th>
<th>Go/No-Go (ANAM) - Hits</th>
<th>Go/No-Go (ANAM) - Omissions</th>
<th>Go/No-Go (ANAM) - Commissions</th>
<th>Go/No-Go (ANAM) - D'</th>
<th>Relational Learning - Block 1 Target Accuracy Proportion</th>
<th>Relational Learning - Block 1 Relation Accuracy Proportion</th>
<th>Relational Learning - Block 1 Shape Accuracy Proportion</th>
<th>Relational Learning - Block 1 Spoiler Accuracy Proportion</th>
<th>Relational Learning - Block 1 Total Accuracy Proportion</th>
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<tr>
<td><strong>Go/No-Go (ANAM) - Mean RT</strong></td>
<td>Pearson Correlation</td>
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<td><strong>Go/No-Go (ANAM) - Commissions</strong></td>
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<td>0.02</td>
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<td>0.23</td>
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*Correlation is significant at the 0.05 level (2-tailed).
**Correlation is significant at the 0.01 level (2-tailed).
Table 49. Correlations between ANAM cognitive scales and proportion of accurate responses on Block 2 of the Relational Learning Task: Go/No-Go

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<thead>
<tr>
<th></th>
<th>Go/No-Go (ANAM) - Mean RT</th>
<th>Go/No-Go (ANAM) - Accuracy</th>
<th>Go/No-Go (ANAM) - Hits</th>
<th>Go/No-Go (ANAM) - Omissions</th>
<th>Go/No-Go (ANAM) - Commission s</th>
<th>Go/No-Go (ANAM) - D’</th>
<th>Relational Learning - Block 2 Target</th>
<th>Relational Learning - Block 2 Relation</th>
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<td>-.12</td>
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<td>.79*</td>
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</table>

* Correlation is significant at the 0.05 level (2-tailed).
** Correlation is significant at the 0.01 level (2-tailed).
Table 50.  
Correlations between ANAM cognitive scales and proportion of accurate responses on Block 3 of the Relational Learning Task: Go/No-Go

| Go/No-Go (ANAM) - Mean RT | Go/No-Go (ANAM) - Accuracy | Go/No-Go (ANAM) - Hits | Go/No-Go (ANAM) - Omissions | Go/No-Go (ANAM) - Commissions | Go/No-Go (ANAM) - D' | Relational Learning - Block 3 Target Accuracy Proportion | Relational Learning - Block 3 Relation Accuracy Proportion | Relational Learning - Block 3 Shape Accuracy Proportion | Relational Learning - Block 3 Spoiler Accuracy Proportion | Relational Task - Block 3 Total Accuracy Proportion |
|---------------------------|-----------------------------|------------------------|-----------------------------|-------------------------------|--------------------------|----------------------------------------------------------------|-----------------------------------------------------|-----------------------------------------------------|-----------------------------------------------------|-----------------------------------------------------|-----------------------------------------------------|
| Go/No-Go (ANAM) - Mean RT | Pearson Correlation | 1.00 | -.02 | -.44 | .49 | -.44 | -.29 | -.07 | -.44 | -.36 | -.36 | -.41 |
| Sig. (2-tailed) | N | 30 | 30 | 30 | 30 | 30 | 30 | 29 | 29 | 29 | 29 | 29 |
| Go/No-Go (ANAM) - Accuracy | Pearson Correlation | -.02 | 1.00 | .75 | -.68 | -.61 | .80 | -.02 | -.32 | -.32 | -.18 | -.29 |
| Sig. (2-tailed) | N | 30 | 30 | 30 | 30 | 30 | 30 | 29 | 29 | 29 | 29 | 29 |
| Go/No-Go (ANAM) - Hits | Pearson Correlation | -.44 | .75 | 1.00 | -.97 | -.01 | .69 | .17 | -.15 | -.05 | -.05 | .06 |
| Sig. (2-tailed) | N | 30 | 30 | 30 | 30 | 30 | 30 | 29 | 29 | 29 | 29 | 29 |
| Go/No-Go (ANAM) - Omissions | Pearson Correlation | .49 | -.68 | -.97 | 1.00 | -.05 | -.68 | -.18 | -.12 | .03 | .02 | -.09 |
| Sig. (2-tailed) | N | 30 | 30 | 30 | 30 | 30 | 30 | 29 | 29 | 29 | 29 | 29 |
| Go/No-Go (ANAM) - Commissions | Pearson Correlation | -.44 | -.67 | -.01 | -.05 | 1.00 | -.44 | .23 | .30 | .41 | .21 | .50 |
| Sig. (2-tailed) | N | 30 | 30 | 30 | 30 | 30 | 30 | 29 | 29 | 29 | 29 | 29 |
| Go/No-Go (ANAM) - D' | Pearson Correlation | -.29 | .80 | .69 | -.68 | -.44 | 1.00 | .03 | -.29 | -.19 | -.06 | -.18 |
| Sig. (2-tailed) | N | 29 | 29 | 29 | 29 | 29 | 29 | 29 | 29 | 29 | 29 | 29 |
| Relational Learning - Block 3 Target Accuracy Proportion | Pearson Correlation | -.07 | .02 | .17 | -.18 | .23 | .03 | 1.00 | -.23 | -.22 | -.04 | .73 |
| Sig. (2-tailed) | N | 29 | 29 | 29 | 29 | 29 | 29 | 29 | 29 | 29 | 29 | 29 |
| Relational Learning - Block 3 Relation Accuracy Proportion | Pearson Correlation | -.44 | -.32 | -.15 | -.12 | .30 | -.29 | -.23 | 1.00 | .52 | .26 | .43 |
| Sig. (2-tailed) | N | 29 | 29 | 29 | 29 | 29 | 29 | 29 | 29 | 29 | 29 | 29 |
| Relational Learning - Block 3 Shape Accuracy Proportion | Pearson Correlation | -.36 | -.32 | -.05 | .03 | .41 | -.19 | -.22 | .52 | 1.00 | -.06 | -.37 |
| Sig. (2-tailed) | N | 29 | 29 | 29 | 29 | 29 | 29 | 29 | 29 | 29 | 29 | 29 |
| Relational Learning - Block 3 Spoiler Accuracy Proportion | Pearson Correlation | -.36 | -.18 | -.05 | .02 | .21 | -.06 | -.04 | .26 | -.06 | 1.00 | .13 |
| Sig. (2-tailed) | N | 29 | 29 | 29 | 29 | 29 | 29 | 29 | 29 | 29 | 29 | 29 |
| Relational Task - Block 3 Total Accuracy Proportion | Pearson Correlation | -.41 | -.29 | -.06 | -.09 | .50 | -.18 | .73 | .43 | .37 | .13 | 1.00 |
| Sig. (2-tailed) | N | 29 | 29 | 29 | 29 | 29 | 29 | 29 | 29 | 29 | 29 | 29 |

* Correlation is significant at the 0.05 level (2-tailed).
** Correlation is significant at the 0.01 level (2-tailed).
Table 51.
Correlations between ANAM cognitive scales and proportion of accurate responses on the whole Relational Learning Task: Go/No-Go

<table>
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<tr>
<th>Correlation</th>
<th>Go/No-Go (ANAM) - Mean RT</th>
<th>Go/No-Go (ANAM) - Accuracy</th>
<th>Go/No-Go (ANAM) - Hits</th>
<th>Go/No-Go (ANAM) - Omissions</th>
<th>Go/No-Go (ANAM) - Commissions</th>
<th>Go/No-Go (ANAM) - D'</th>
<th>Relational Task - Wrong</th>
<th>Relational Task - Shape</th>
<th>Relational Task -Spoiler</th>
<th>Relational Task - Target</th>
<th>Relational Task - Whole</th>
<th>Relational Task - Accuracy</th>
<th>Proportion</th>
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<td>0.37</td>
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* Correlation is significant at the 0.05 level (2-tailed).

** Correlation is significant at the 0.01 level (2-tailed).
Figure 1. The Spatial Memory Task: The mean proportion of correct responses as a function of Group (HC or SLE), Memory Type (spatial or nonspatial), and Item Difficulty (easy or hard).
Figure 2. The Spatial Memory Task: The mean proportion of correct responses as a function of group (HC or SLE) and array difficulty (easy or hard) for all items (regardless of whether they related to spatial or nonspatial questions). This graph demonstrates the significant Group by Item Difficulty two-way interaction of the 2 x 2 mixed ANOVA.

*Note: Bars represent standard error.
Figure 3. The Spatial Memory Task: The mean proportion of correct responses as a function of Group (HC or SLE) and Memory Type (spatial or nonspatial) for only the easy items. This graph demonstrates the Group by Memory Type interaction of the 2 x 2 mixed ANOVA.

*Note: Bars represent standard error.
Figure 4. The Spatial Memory Task: The mean proportion of correct responses as a function of Group (HC, DNRAb-, or DNRAb+) and Item Difficulty (easy or hard). This graph demonstrates the Group by Item Difficulty interaction of the 3 x 2 x 2 mixed ANOVA.

*Note: Bars represent standard error.
Figure 5. The Spatial Memory Task: The mean proportion of correct responses as a function of Group (HC, DNRAb-, or DNRAb+) and Memory Type, separated by Item Difficulty (easy or hard). This graph demonstrates the significant 3-way Group by Memory Type by Item Difficulty interaction of the 3 x 2 x 2 mixed ANOVA.

*Note: Bars represent standard error.
Figure 6. The Relational Learning Task: The mean proportion of correct responses as a function of Group (HC or SLE) and Block.

*Note: Bars represent standard error.*
Figure 7. The Relational Learning Task: The mean proportion of correct responses as a function of Group (HC, DNRAb-, DNRAb+) and Block.

*Note: Bars represent standard error.
Figure 8. The Relational Learning Task: The mean proportion correct on error items as a function of Group (HC or SLE), Block, and Recognition Stimuli Type.

*Note: Bars represent standard error.*
Figure 9. The Relational Learning Task: The mean proportion correct on error items as a function of Group (HC, DNRAb-, DNRAb+), Block, and Recognition Stimuli Type.

*Note: Bars represent standard error.
Appendix A

Below are the results regarding the correlations among the demographic, affective, and neuropsychological variables.

Relations among background factors to each other: Age and disease duration were moderately correlated, $r = 0.34, p < 0.01$. Age and education, and education and disease duration were not significantly correlated with one another.

Relations among affective factors to each other: Depression on the BDI was significantly and strongly correlated with scores on the state anxiety self-report (STA-Y), $r = 0.55, p < 0.01$; trait anxiety self-report (TAI-Y), $r = 0.72, p < 0.01$. For both of these scales, higher scores on the BDI were associated with higher scores both anxiety inventories. Scores on the STA-Y were significantly correlated with scores on the TAI-Y, $r = 0.58, p < 0.01$, such that higher STA-Y scores were associated with higher TAI-Y scores. Higher scores on the sleepiness scale were significantly correlated with higher scores on the fatigue scale, $r = -0.51, p = 0.01$; lower scores on the happiness scale, $r = -0.42, p = 0.04$; and lower scores on the vigor scale, $r = -0.65, p < 0.01$. Higher scores on the anger scale were significantly correlated with higher scores on the ANAM anxiety scale, $r = 0.61, p < 0.01$; higher scores on the ANAM depression scale, $r = 0.67, p < 0.01$; and higher scores on the restlessness scale, $r = 0.66, p < 0.01$. High scores on the ANAM anxiety scale were associated with higher scores on the ANAM depression scale, $r = 0.81, p < 0.01$, higher scores on the fatigue scale, $r = 0.46, p = 0.01$, and higher scores on the restlessness scale, $r = 0.77, p < 0.01$. Higher scores on the ANAM depression scale was associated with higher scores on the fatigue scale, $r = 0.40, p = 0.03$. Higher scores on the fatigue scale was associated with higher scores on the restlessness scale, $r = 0.46, p = 0.01$. Higher scores on the happiness scale was significantly correlated with higher scores on the vigor scale, $r = 0.88, p < 0.01$.

Relations among cognitive and neuropsychological tasks to each other: A higher number of correct answers on the JLO was significantly correlated with a fewer number of seconds to complete the TMT-A task, $r = -0.39, p = 0.04$; a higher raw score on the RCFT copy task, $r = 0.59, p < 0.01$; a higher raw score on the RCFT delay, $r = 0.65, p < 0.01$; greater accuracy on the Matching Grids task, $r = 0.38, p = 0.04$; greater efficiency on the Matching Grids task, $r = 0.38, p = 0.04$; greater accuracy on the Match to Sample task, $r = 0.42, p = 0.02$; greater efficiency on the Match to Sample task, $r = 0.44, p = 0.01$; efficiency on the Running Memory CPT, $r = 0.37, p = 0.04$; lower accuracy on the Go/No-Go task, $r = 0.37, p = 0.04$; and higher number of commission errors on the Go/No-Go task, $r = 0.50, p < 0.05$.

The longer amount of time taken to complete the TMT-A task (in seconds) was significantly correlated with a higher number of seconds to complete the TMT-B, $r = 0.55, p < 0.01$; lower raw score on the RCFT copy, $r = -0.41, p < 0.01$; lower efficiency on the Matching Grids task, $r = -0.54, p < 0.01$; lower efficiency on the Match to Sample task, $r = -0.43, p = 0.02$; and lower efficiency on the Running Memory CPT, $r = -0.60, p < 0.01$.

A longer amount of time complete the TMT-B task (in seconds) was significantly correlated with a lower raw score on the RCFT copy, $r = -0.54, p < 0.01$; lower raw score RCFT delay, $r = 0.36, p < 0.01$; lower efficiency on the Matching Grids tasks, $r = -0.41, p = 0.03$; lower efficiency on the Match to Sample task, $r = -0.45, p = 0.01$; and lower efficiency on the Running Memory CPT, $r = -0.43, p = 0.02$.

Higher raw scores on the RCFT copy task were significantly correlated with higher raw scores on the RCFT delay, $r = 0.46, p < 0.01$, and efficiency on the Match to Sample task, $r = 0.43, p = 0.02$. Higher scores on the RCFT delay were significantly correlated with greater...
efficiency on the Match to Sample task, $r = 0.46, p = 0.01$; and greater efficiency on the Running Memory CPT, $r = 0.37, p = 0.05$. Greater efficiency on the Simple RT task was significantly correlated with greater efficiency on the Running Memory CPT, $r = 0.39, p = 0.03$. Greater efficiency on the Matching Grids task was significantly correlated with greater efficiency on the Match to Sample task, $r = 0.69, p < 0.01$, and greater efficiency on the Running Memory CPT, $r = 0.40, p = 0.03$. Greater efficiency on the Match to Sample task was significantly correlated with greater efficiency on the Running Memory CPT, $r = 0.38, p = 0.40$. Greater efficiency on the Running Memory CPT was significantly correlated with greater $D'\prime$, $r = 0.46, p = 0.01$. 


Julian, L. J. (2011). Measures of anxiety: State-Trait Anxiety Inventory (STAI), Beck Anxiety Inventory (BAI), and Hospital Anxiety and Depression Scale-Anxiety (HADS-A). *Arthritis Care & Research, 63*(S11), S467-S472.


