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## Functional capacity in schizophrenia: Relationship among effort, reinforcement learning and self-beliefs

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FUNCTIONAL CAPACITY IN SCHIZOPHRENIA: RELATIONSHIP AMONG  
EFFORT, REINFORCEMENT LEARNING, AND SELF-BELIEFS

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May 2015

## ABSTRACT

### **Functional capacity in schizophrenia: Relationship among effort, reinforcement learning and self-beliefs**

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Individuals with schizophrenia exhibit a wide range of complex neurocognitive, psychiatric and behavioral impairments. Recent research suggests that this complex array of symptoms can be at least partially accounted for by dysfunction in more basic mechanisms, such as the ability to learn from positive and negative reinforcement, or the ability to exert adequate effort when completing simple tasks. Evidence also suggests that deficits in these basic mechanisms may contribute to more complex symptoms, such as functional impairment. Also, the relationship between neurocognitive deficits and functional impairment has been found to be mediated by defeatist performance beliefs. However, studies have not examined the relationships among these various constructs so it is not clear how impairment in more basic processes relate to the development or maintenance of complex psychological and behavioral disturbances. The current study addresses these matters by examining effort, reinforcement learning, defeatist performance beliefs and functional capacity in individuals with schizophrenia and controls using a path analysis. After examining a number of competing models, the best fitting model was one in which defeatist performance beliefs were predicting effort, reinforcement learning, and functional capacity while effort and reinforcement learning were also predicting functional capacity. This model depicted the opposite relationship

among the variables than was expected. The current study suggests that defeatist performance beliefs have a more predictive and potentially causal influence on complex neurocognitive abilities related to learning and reward, as well as functional capacity. Results of this study support the use of therapeutic strategies aimed at reducing cognitive distortions, e.g. defeatist performance beliefs, and that these therapeutic strategies may be impacting neurocognitive abilities and functional outcome more directly than previously believed.

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## CHAPTER 1

### INTRODUCTION

Schizophrenia is a debilitating disorder that entails cognitive impairments and often leads to functional impairment (MacDonald & Schulz, 2009). Schizophrenia has high heritability rates (Cardno, Rijsdijk, Sham, Murray, & McGuffin, 2002; Hughes et al., 2005; MacDonald & Schulz, 2009), genetic factors have been found to be associated with the presence of the disorder (MacDonald & Schulz, 2009), and pharmacological medications are a first line of treatment for the most debilitating symptoms of the disorder (MacDonald & Schulz, 2009; Rector & Beck, 2001). Thus, it is no surprise that leading frameworks of the disease are biological. It is also well understood that having genetic risk factors alone will not result in the disorder (Beck, 2004; MacDonald & Schulz, 2009). In addition to genetic risk factors, environmental risk factors constitute an additive effect and must also be present in order to “push” someone into psychosis. Beck (2004) proposed a cognitive model of both positive and negative symptoms of schizophrenia that could serve as one explanation for additional factors necessary to develop or perpetuate the disorder. He states that many of the symptoms of schizophrenia are more extreme versions of thoughts and experiences that many people without the disorder will experience. What differentiates these normal experiences and the experiences present in schizophrenia is the level of distress caused, the intensity, and the certainty of the beliefs.

Studies examining the treatment of schizophrenia have provided evidence supporting the beneficial effects of cognitive behavioral therapy (CBT) on positive and negative symptoms of schizophrenia as well as functional outcome (Gould, Mueser,

Bolton, Mays, & Goff, 2001). Some symptoms of schizophrenia that are addressed by CBT are cognitive distortions based on defeatist beliefs regarding oneself, ones capabilities, and ones environment. Furthermore, defeatist performance beliefs have been found to mediate the relationship between neurocognitive impairment and functional outcome (Grant & Beck, 2008). Specifically, Grant and Beck (2008) examined the performance of traditional measures of neurocognitive function in schizophrenia, such as attention and executive functioning, in relation to defeatist performance beliefs and functional outcome and found the mediating effect of defeatist performance beliefs. Multiple neurocognitive functions have been found to be impaired in schizophrenia, including the basic neurocognitive abilities of reinforcement learning (Prentice, Gold, & Buchanan, 2008; Strauss et al., 2011; Waltz, Frank, Robinson, & Gold, 2007) and effort (Avery, Startup, & Calabria, 2009; Lafargue & Franck, 2009).

Reinforcement learning is the ability to learn from positive and negative outcomes in the environment (Schultz, 2002). Reinforcement learning is associated with midbrain dopaminergic areas and areas within the prefrontal cortex (Barch & Dowd, 2010). Effort is a cost-benefit analysis between energy expended and benefit gained (Merriam-Webster, 2012). Rewards/reinforcements are one potential source of benefits gained for the cost of effort. Thus, brain areas associated with the evaluation of reward would be suggested to contribute to effort. Research has in fact suggested that midbrain dopaminergic areas and prefrontal areas are involved in effort decisions (Botvinick, Huffstetler, & McGuire, 2009; Croxson, Walton, O'Reilly, Behrens, & Rushworth, 2009). It seems then that similar dysfunctional brain areas in schizophrenia may impact

both reinforcement learning and effort, an idea supported by the impairment in both of these abilities in schizophrenia.

The causal relationship between reinforcement learning, effort, defeatist performance beliefs, and functional capacity has not yet been examined, despite the fact that they have been found to be associated with one another. The following study examined the causal relationship between these variables. It is hypothesized that the more basic neurocognitive abilities, reinforcement learning and effort, will have an additive and causal effect on the more complex behavior of defeatist performance beliefs, which will then exerts a causal and additive effect on functional capacity.

In order to implement effective treatments that target these functional abilities and outcomes, one must understand what components of a disorder are contributing to the problems. Identifying specific components of a disorder that contribute to poor outcome allows treatments to be tailored to changing those aspects. The results of this study could provide direction for both pharmacological and cognitive behavioral treatments for schizophrenia.

## CHAPTER 2

### LITERATURE REVIEW

In the following sections, each of the areas involved in the current study are reviewed with regard to recent and relevant literature. Specifically, effort and suggested brain regions associated with the evaluation and allocation of effort are reviewed. Reinforcement learning and brain regions associated with this task are also reviewed. Defeatist performance beliefs in general, in schizophrenia, and some of the potential sources for these beliefs are reviewed. Finally, functional outcome and capacity are reviewed.

#### **Reinforcement Learning**

Reinforcement learning has been conceptualized as a result of the difference or error between what an animal or human predicts will occur and the actual outcome (Dayan & Daw, 2008; Hollerman & Schultz, 1998). Hollerman & Schultz (1998) state that learning ensues if the outcome or reward is better than what was expected or predicted by the animal. Learning tapers off as the outcome and prediction equal one another. Extinction will occur if the outcome or reward is worse than what was expected or predicted by the animal. This theory of reinforcement-based learning has been termed temporal difference error learning, with the error being the difference between outcome and prediction. Midbrain dopamine activity has been implicated in the coding of error signals, with increases and decreases in dopamine activity coding for positive and negative temporal difference errors, respectively. Specifically, Hollerman & Schultz (1998) studied the activity of dopamine neurons in the pars compacta and ventral tegmental area in monkeys in response to reward. They found that dopamine neurons in

these areas responded to reward during initial learning trials but rarely responded to reward in later trials following familiar pictures that had previously been rewarded. During learning trials, dopamine neurons in these areas would respond to reward initially and taper off responding as the association was learned (i.e., the prediction matched the outcome). Additionally, they found that dopamine activity was significantly depressed when an incorrect choice was made and no reward was delivered. If reward delivery was delayed, significant depressions in dopamine activity were found at the time the reward was expected but were significantly activated when the reward was then administered. These transient increases and decreases in dopamine are thought to facilitate reinforcement learning via two separate pathways originating in the basal ganglia (Waltz et al., 2007). The direct “Go” pathway is excited by D1 receptors and facilitates responding to reinforced, rewarding stimuli by enhancing activity and plasticity. The indirect “NoGo” pathway is inhibited by D2 receptors and suppresses responding to negatively reinforced or punished stimuli by increasing inhibition and further decreasing activity in the Go pathway. These pathways are connected to the prefrontal cortex and aid in the acquisition and update of reinforcement learning.

Midbrain dopamine areas are not the only regions thought to be responsible for reinforcement-based learning. In addition, areas of the prefrontal cortex have been implicated (Barch & Dowd, 2010; Waltz et al., 2007). The orbitofrontal cortex is one area of the prefrontal cortex thought to be involved in reinforcement learning. Specifically, it is thought to be involved in the working memory of reward, such as the ability to maintain, update, and integrate the expected values of a reward (Barch & Dowd, 2010). Such working memory aspects of reinforcement learning suggest that the

orbitofrontal cortex is responsible for immediate reward evaluations (Waltz et al., 2007). Additionally, the working memory of reward component of the orbitofrontal cortex is thought to be connected to the midbrain dopamine areas. The orbitofrontal cortex updates the basal ganglia providing a top-down evaluation of reward (Gold et al., 2012). The orbitofrontal cortex is also thought to be updated by the transient increases and decreases of dopamine activity in response to reward prediction error in midbrain dopamine areas providing bottom-up processing of reward, specifically from the basal ganglia (Waltz et al., 2007).

Animal and human studies of dopamine cell activity have sparked interest in studying temporal difference error learning in humans with deficits in dopamine activity, including Parkinson's disease (Frank, Seeberger, & Reilly, 2004) and schizophrenia (Prentice et al., 2008; Waltz et al., 2007).

**Reinforcement-Learning Deficits in Schizophrenia.** Dopamine's role in schizophrenia was implicated following the realization that antipsychotic medications acted on dopamine systems, a theory confirmed by imaging studies (Kapur, Mizrahi, & Li, 2005). Given that individuals with schizophrenia display both learning deficits and abnormal dopamine function (Kapur et al., 2005), reinforcement learning has been examined in these individuals (Prentice et al., 2008; Waltz et al., 2007).

Waltz and colleagues (2007) examined reinforcement learning in individuals with schizophrenia using a probabilistic selection task that had previously been used to examine reinforcement learning deficits in Parkinson's disease (Frank et al., 2004). This type of measure is commonly used in both human and animal studies of reinforcement learning and can differentiate the use of positive and negative feedback. The specific



probabilistic selection task used in this study requires participants to learn the most frequently positively reinforced stimulus within three stimulus pairs of variable reinforcement schedules (i.e., 80:20, 70:30, 60:40). Once learned, the original stimuli are presented in novel pairings and the selection of the most frequently rewarded stimulus and avoidance of the least frequently rewarded stimulus is examined. They found a general impairment in individuals with schizophrenia's acquisition of probabilistic contingencies, although they demonstrated eventual learning of the easiest contingencies (80:20). In addition to overall task performance, they also examined the use of positive and negative feedback and found that patients were significantly less able to maintain a correct response after being positively reinforced and significantly less able to shift a response after receiving negative feedback. These results suggest that individuals with schizophrenia are impaired on their use of rapid trial-by-trial feedback to guide behavior, but after many presentations gradual learning is intact. The authors suggest that this pattern of performance can be explained by dopamine hypofunction in the prefrontal cortex, negatively effecting rapid reward evaluations, but a less impaired dopamine dysregulation in the basal ganglia, supporting gradual and eventual reinforcement-learning. They also found that control subjects were significantly better than individuals with schizophrenia at choosing the most frequently positively reinforced stimuli, suggesting an impairment in Go learning in individuals with schizophrenia. In contrast, no difference was found between control and schizophrenia groups on their ability to avoid the least frequently rewarded (i.e. most often punished) stimuli, suggesting an intact NoGo pathway. Patients were medicated with antipsychotics at the time of testing. Antipsychotic medications are D2 antagonists, which have been suggested to enhance

NoGo learning (Strauss et al., 2011). Notably, measures of cognitive functioning were not associated with any variable of reinforcement learning and when included as a covariate did not change the results. This could suggest that the more basic neurocognitive process of reinforcement learning exerts a unique contribution to higher order/more complex cognitions and behaviors, such as defeatist performance beliefs and functional capacity.

Additionally, Prentice, Gold & Buchanan (2008) evaluated individuals with schizophrenia on the ability to utilize negative and positive feedback using the Wisconsin Card Sorting Test (WCST). The WCST is traditionally used as a task of executive functioning, but has been used in both animal and human studies evaluating reinforcement learning (Montague, Hyman, & Cohen, 2004; Schultz, 2002). Initial WCST trials can be conceptualized from a temporal difference error standpoint, with responses resulting in outcomes better than expected being associated with increases in dopamine activity and responses resulting in outcomes worse than expected being associated with decreases in dopamine activity. Prentice and colleagues (2008) found that individuals with schizophrenia had greater deficits than controls in using rapid trial-by-trial feedback to guide behavior. This impairment was attributable to patients' impaired ability to shift responses following negative feedback. Patients demonstrated no significant impairment in their ability to maintain a positively reinforced response. A general reinforcement learning impairment is consistent with Waltz et al., (2007), however, disparate findings were found between the two studies on patient's use of rapid trial-by-trial use of positive and negative feedback. As mentioned, Waltz et al, (2007) found an impaired ability to shift a response following negative feedback as well as an

impaired ability to maintain a response following positive feedback in individuals with schizophrenia, while Prentice et al., (2008) found individuals with schizophrenia to be impaired only on their use of negative feedback demonstrated by an impaired ability to shift a response following negative feedback. Both studies evaluated reinforcement learning in individuals with schizophrenia and did not evaluate performance based on specific symptoms of schizophrenia.

Strauss and colleagues (2011) examined reinforcement learning using a probabilistic selections task in individuals with schizophrenia. They expanded on the Waltz et al., (2007) study in several ways. First, they divided the schizophrenia group into those with high negative symptoms and low negative symptoms. Additionally, reward probabilities and magnitudes varied as a function of response time, rather than having constant reward probabilities. They found that individuals with schizophrenia were impaired in their ability to increase response time in order to maximize reward but showed no impairment on their ability to slow down in response to negative prediction errors. These findings are consistent with Waltz et al. (2007) impaired Go learning but intact NoGo learning in individuals with schizophrenia. Additionally, Go learning deficits were found to be more severe in patients with high negative symptoms.

Participant's willingness to explore novel options when uncertain about the value of reward was also examined. They found that anhedonia was significantly related to novel exploration such that individuals with higher anhedonia explored less. Dopamine dysregulation in the prefrontal cortex is thought to contribute to impairments in exploration during uncertainty as well as negative symptoms (Carter, 2007). Also of

note, general cognitive functioning did not relate to or alter the performance on these reinforcement learning measures.

Gold and colleagues (2012) argue that it is unclear whether previous findings in reward-learning impairments in schizophrenia are due to dysfunction in the prediction error system (bottom-up processing mediated by the basal ganglia) or the valuation of reward (top-down processing mediated by the orbitofrontal cortex). In order to evaluate this distinction they used a probabilistic selection task in which monetary gains, losses, or neither a gain nor loss were probabilistically presented. The task was set up in such a way that with some stimuli pairings a no gain or loss response would result in positive prediction errors (paired with monetary loss), while on other pairings no gain or loss would result in negative prediction errors (paired with monetary gain). Stimuli associated with monetary gain and those associated with no gain or loss but positive prediction errors were paired, as well as stimuli associated with monetary loss and those associated with no gain or loss but negative prediction errors. Because they were associated with the same prediction error, they would be equally likely to be selected when paired together if the value of the reward was not taken into consideration (i.e. choice related to basal ganglia function rather than orbitofrontal cortex). Consistent with previous research, they found that individuals with high negative symptoms had the most substantial deficits in reinforcement learning. Patients were also found to have no deficit in their ability to avoid losses. Additionally, they found that individuals with schizophrenia with high negative symptoms did not show a preference for stimuli that were associated with a higher reward over those with no monetary gain that were associated with positive prediction errors. These results suggest that the dysfunction in

the orbitofrontal cortex resulting in impaired valuations or reward is the primary mechanism for previous reward-learning impairments found in individuals with schizophrenia and high negative symptoms.

Barch and Dowd (2010) examined motivation with regard to goal representations in individuals with schizophrenia. They proposed that individuals with schizophrenia exhibit impairment in the evaluation of reward, related to reward prediction and mediated in the midbrain dopaminergic areas. The impaired evaluation of reward results in an impairment in future goal representation and lack of motivation. Studies have not yet examined the relationship between reinforcement learning and effort. As discussed by Barch and Dowd, these actions share similar midbrain and prefrontal pathways, suggesting their performance may be related.

### **Effort**

Effort is a conscious exertion of power or energy (Merriam-Webster, 2012). Typically effort is thought of in terms of physical exertion but can also be applied to cognitive exertion. When considering effort in the realm of neuroeconomics, the allocation of effort is an adaptive mechanism (Kool, McGuire, Rosen, & Botvinick, 2010). Deciding how much effort to exert requires a cost-benefit analysis, often completed below conscious awareness. The cost is the amount of exertion or energy required and the benefit is the gain achieved as a result of the effort. The law of less work is a behavioral and economic theory which states that an action chosen will and should be the one which requires the least amount of effort or work (Kool et al., 2010). When given the choice between two actions, both resulting in the same reward, people tend to choose the action that requires the least amount of effort. The law of less work

applies to physical as well as cognitive actions. Kool and colleagues (2010) tested the law of less work with regard to cognitive demand in healthy individuals. They used a series of demand selection tasks, requiring participants to repeatedly choose between two actions, each requiring different levels of cognitive demand. It was not explicitly stated which option was low demand and which was high demand or that this difference existed between the options. Across six experiments with different demand selection tasks, but all involving a low demand and a high demand option, participants chose the low cognitive demand task significantly more often than the high cognitive demand task. Besides cognitive demand, the tasks did not differ in relation to amount of time the task took or incentive value. They were also able to establish that the choice for less cognitive demand was not in order to minimize the number of errors. Also, participants were unable to explain any difference between the two options or recognize that they had preferred one task over another. A seventh task was included that was similar to the previous six tasks in relation to low and high cognitive demand options, but an additional incentive value/reward was added to the tasks. Participants were given \$0.01 or \$0.10 for completing the tasks, regardless of whether they chose the low demand or high demand task. Again, participants were not aware of the difference in cognitive demand between the tasks. With the addition of reward, the difference between selection of the low and high cognitive demand tasks reduced, suggesting that cognitive demand is less aversive when a benefit is available to offset the cost of cognitive demand. They found no difference between high or low cognitive demand selection between the participants in the \$0.01 and the \$0.10 incentive trials group, suggesting that even a very small reward offsets the aversiveness of cognitive demand.

Effort and reward evaluation have both been found to be associated with midbrain dopaminergic activity (Barch & Dowd, 2010; Botvinick et al., 2009; Botvinick & Rosen, 2009; Croxson et al., 2009). In a theory known as effort discounting, the same midbrain dopaminergic areas implicated in reward evaluation have been implicated in effort. Effort discounting suggests that the same reward in two situations would be more valuable in the situation that required the least amount of effort to obtain it. Effort discounting also considers the choices made between less energy and less reward versus more energy and more reward. Thus, effort discounting is often measured using choice responses between a low effort, low reward and high effort, high reward option. Studies have demonstrated that dopamine antagonists decrease the amount of effort and wait time a rat is willing to endure for a reward, demonstrated by rats preferring a lesser reward and effort (Floresco, Tse, & Ghods-Sharifi, 2008). Similarly, studies have demonstrated that dopamine agonists increase the amount of effort and wait time a rat is willing to endure for a larger reward, demonstrated by rats preferring a larger reward even when requiring more effort (Floresco et al., 2008). There is a similar theory, called delay discounting, which takes into account the amount of time an organism is willing to wait for a reward. In studies of delay discounting, tasks involve a larger reward following a longer delay or a smaller reward following a shorter delay. Many of the same brain areas are implicated in effort and delay discounting. It is possible that the results in prior studies of effort discounting are a by-product of delay discounting, because typically the more effortful task takes longer, thus increasing the delay for the reward as compared to the simpler, less effortful task. Floresco and colleagues (2008) examined this possibility in rats. They manipulated effort and reward in a typical manner to examine effort, but they included a

condition that equalized the delay for reward between less and more effortful tasks. They found that dopamine antagonists in rats still reduce the choice of high effort, high reward, even though the wait for the small reward is the same length. Additionally, for the equal wait times they found that dopamine agonists in rats still increased the high effort, high reward choice. They also examined the effect of NMDA antagonists on delay and effort discounting. They found that NMDA effected effort choices when the delay between the high and low effort tasks were different, but the effect disappeared when equal delays were incorporated. These results suggest that brain areas associated with dopamine are more involved in effort discounting, while NMDA associated areas are involved in delay discounting.

It is worthwhile to evaluate effort discounting in the absence of choice between effort and reward, specifically, neural responses to varying levels of reward and effort/cognitive demand. This would also aid in the distinction between effort and delay discounting. Botvinick, Huffstetler and McGuire (2009) evaluated effort and reward evaluation in the nucleus accumbens (NAcc) and other brain areas implicated in both reward and effort processing or only reward but not effort processing using fMRI. High and low cognitive demand tasks were paired with high and low reward values. Participants were not told the differences between the tasks in effort levels and were notified of reward following the completion of the task. They found an effort discounting reaction in the NAcc, as anticipated. More specifically, NAcc response was stronger for high reward as compared to low reward and reward activation was reduced when the task just completed required a higher level of effort, discounting the reward response and supporting the neural cost of effort expenditure. They also found the dorsal anterior



cingulate cortex (ACC), an area implicated in both reward and effort processing, to be activated during effort processing but not reward processing. The orbitofrontal cortex and the medial prefrontal cortex, areas both implicated in reward processing but not effort, were both activated in response to reward evaluation, but were not effected by level of effort required. Croxson and colleagues (2009) also examined reward and effort evaluation in humans using an fMRI. They had participants complete a task at 4 different effort levels, and each level of effort was paired with 2 levels of reward. Participants again made no choice between tasks, but simply completed the one presented following a cue indicting which level of effort and reward task they were about to complete. They found ACC activation in response to both reward and effort evaluation. They also found activation in the ventral striatum and midbrain in response to both reward and effort evaluation, although they did not specify what aspect of the midbrain had been evaluated, simply that it was calculated as a region of interest based on prior research examining reward. In contrast to Botvinick et al. (2009) and other research implicating the orbitofrontal cortex in reward, they found no activation in the orbitofrontal cortex for either reward or effort processing. The orbitofrontal cortex is implicated in the working memory of reward (reviewed above). It is possible that Croxson and colleagues did not find an effect in the orbitofrontal cortex because the task did not require participants to make a choice in effort or reward or to evaluate the level of reward, but simply respond to varying levels of both after being told what level of reward they would be performing for.

**Effort in Schizophrenia.** While it is adaptive to expend less effort for the same reward, it is theorized that some individual's are willing to expend more effort in general (Eisenberger, 1992). Eisenberger stated that some individual's exhibit 'learned

industriousness' and are more willing to expend effort because they have been internally or environmentally reinforced for their effort, making effort less aversive. It seems plausible then, that there would in turn be 'learned passivity'. In other words, individuals may have been either punished for expending effort, or reinforced for not expending effort, by both internal and external means. These individuals would exhibit a willingness to take a lesser reward in order to expend less effort, or an unwillingness to expend effort regardless of reward. Considering that individuals with schizophrenia often do not believe they are capable or worthy of rewards, they may opt not to expend effort because they believe that they are incapable of expending more effort or unworthy of the reward that more effort may bestow. This view is consistent with the 'why try' effect discussed below with regard to internalized stigma and defeatist performance beliefs (Corrigan, Larson, & Rüsç, 2009). Individuals with schizophrenia then accept that they have lesser rewards in life and less fulfilling lives because of their illness, perpetuating the lack of effort and functional impairments.

Research has been conducted in individuals with schizophrenia evaluating whether they put forth less effort than healthy individuals and whether the level of effort expended is related to neurocognitive performance or specific symptoms of schizophrenia (Barch & Dowd, 2010; Gorissen, Sanz, & Schmand, 2005). Gorissen and colleagues (2005) examined effort and neurocognitive performance in individuals with schizophrenia. Their performance was compared with a group of neurological patients with confirmed structural brain damage due to head injury and healthy controls. A larger percentage of the schizophrenia group put forth insufficient effort, as determined by a common neuropsychology recognition memory task designed to determine insufficient

effort/malingering, than either the neurologic or healthy control groups. The schizophrenia group was then divided into two groups based on effort, an insufficient effort group, comprised of those individuals that failed the effort measure, and a normal effort group, comprised of those that passed the effort measure. The insufficient effort group performed significantly worse on the neurocognitive measures than the normal effort group. Negative symptoms were found to be associated with insufficient effort. One limitation of this study was that the effort measure used can only be evaluated on a pass/fail basis and interpretations cannot be made on a continuum of effort. Furthermore, they found a much higher rate of low effort than other studies that have used the same effort measure in individuals with schizophrenia (Avery et al., 2009).

Based on the fact that brain areas associated with effort have been found to be dysfunctional in individuals with schizophrenia, cognitive impairment may be partially responsible for less effort expenditure. Also, defeatist performance beliefs have been theorized to be associated with a lower willingness to expend effort (the ‘why try’ effect), although it has not been formally tested.

### **Defeatist-Performance Beliefs**

Individuals with schizophrenia experience stigma within their environments. This cultural stigma against those with mental illnesses, including schizophrenia, is present and known prior to the individual’s symptom presentation and subsequent diagnosis. Once the diagnosis occurs, the cultural stigma often becomes internalized. Holding the belief that individuals with mental illness are weak or incapable, and then becoming a part of that group can result in then applying that stigma or stereotype to oneself.

Internalizing stigma has been found to be associated with low self-esteem, low self-efficacy, dysfunctional attitudes, and poor functional outcome.

Beck's (2004) cognitive model of schizophrenia outlines how cognitive distortions and biases result in psychotic symptoms. A component of these biases are dysfunctional attitudes and beliefs about oneself and the environment.

**Stigma.** Individuals with mental illnesses, including schizophrenia, experience a large amount of stigma from others because of their illness (Angermeyer, Beck, Dietrich, & Holzinger, 2004; Dickerson, Sommerville, Origoni, Ringel, & Parente, 2002; Link, Struening, Neese-Todd, Asmussen, & Phelan, 2001; Wright, Gronfein, & Owens, 2000). In fact, schizophrenia has been considered one of the most stigmatizing mental illnesses (Angermeyer & Schulze, 2001). A study conducted in Germany found that the most common perceptions by the public of schizophrenia were that of incompetence, unpredictability, and dangerousness (Angermeyer & Matschinger, 2004). Stigma that is both perceived/anticipated and stigma that is actually experienced has been found to negatively impact self-esteem, social relationships, employment, quality of life, and result in increased demoralization and depressiveness (Angermeyer et al., 2004; Corrigan et al., 2009; Link et al., 2001; Lysaker, Tsai, Yanos, & Roe, 2008). Self-esteem is a person's appraisal or attitude of him/herself, which is often contributed to by perceived group membership (Lysaker et al., 2008). Orth, Robins, and Widaman (2012) conducted an analysis of self-esteem on life outcomes across the lifespan. Using growth curve analyses they concluded that self-esteem is not merely a by-product or result of success or failure but that it is better conceptualized as a cause of life outcomes, such as psychological symptoms, relationships, and job satisfaction. One aspect of self-esteem that has been

found to be impacted from stigmatization is self-deprecation (Wright et al., 2000). Wright and colleagues (2000) evaluated self-esteem in a group of individuals diagnosed with various mental illnesses that had recently been deinstitutionalized due to a hospital closure and followed them longitudinally for 2 years. Participants in this study had been institutionalized at the same hospital for an average of 8 years before being discharged due to the hospital's closure. They found that both perceived and experienced stigmatization led to self-deprecation, which led to feelings of loss of mastery and control over the environment.

**Internalized Stigma.** Internalized stigma, or self-stigma, is one means that stigmatization leads to low self-esteem and poorer outcomes (Cavelti, Kvrjic, Beck, Rüsçh, & Vauth, 2012; Park, Bennett, Couture, & Blanchard, 2012; Vauth, Kleim, Wirtz, & Corrigan, 2007). Self-stigma involves being aware of the cultural stigma that is present against a particular group, believing or agreeing with it, and, if it is a group that one belongs to, internalizing or applying it to oneself (Cavelti et al., 2012; Corrigan et al., 2009). Corrigan and colleagues (2009) described an effect of self-stigma they labeled the “why try” effect. Self-esteem and self-efficacy are thought to be mediators between self-stigmatization and life goal attainments, such as employment and quality of life. Self-efficacy is an individual's belief that he/she can successfully act on situations (Corrigan et al., 2009). Similar to low self-esteem, low self-efficacy has been found to be associated with unemployment, poor functional outcome and lower quality of life (Corrigan et al., 2009; Vauth et al., 2007). Individuals with mental illness that experience self-stigma which result in low self-efficacy and self-esteem may think that they are unworthy or unable to succeed in basic life functions, such as living independently. In

other words, they may adopt an attitude that is consistent with the notion “why try”. Lysaker and colleagues (2008) found that individuals with schizophrenia that internalize stigma view themselves as less competent, which is a component of self-esteem that is related to self-efficacy. Similarly, Vauth and colleagues (2007) examined the relationship between self-stigma, self-efficacy, depression, and quality of life in individuals with schizophrenia. Using structural equation modeling, they found that self-stigmatization led to decreased self-efficacy and empowerment, which led to increased depression and decreased quality of life.

**Dysfunctional Attitudes.** Applying stigma to oneself involves negative or dysfunctional self-statements and self-schemas (Park et al., 2012). One commonly researched type of dysfunctional beliefs is defeatist performance beliefs. Defeatist performance beliefs are overly negative and generalized beliefs about one’s ability to perform tasks and the likelihood of succeeding (Beck & Grant, 2008; Couture, Blanchard, & Bennett, 2011; Grant & Beck, 2008; Park et al., 2012). Individuals with schizophrenia report higher levels of defeatist performance beliefs than controls (Horan et al., 2010) and similar levels of dysfunctional attitudes as individuals with depression (Zimmerman, Coryell, Corenthal, & Wilson, 1986). Park and colleagues (2012) found a significant correlation between defeatist performance beliefs and internalized stigma in individuals with schizophrenia.

**Cognitive Model of Psychosis.** Beck (2004) proposed a cognitive model of schizophrenia to understand and explain both positive and negative symptoms of schizophrenia. Beck explained that all symptoms of schizophrenia, such as hallucinations, delusions, disorganization, and negative symptoms are experienced along

a continuum throughout the population. The symptoms experienced by individuals with schizophrenia are on the severe end of this continuum. On the more severe end of the spectrum, these symptoms become more consuming, debilitating, and more firmly held. The delusional content once becoming psychotic is usually held prior to psychosis. If an individual's delusion involves mind reading, he/she likely believed that was possible prior to his/her psychosis. Similarly, research has found that individual's delusions are culturally relevant (Suhail & Cochrane, 2002). A person would not have a delusion of something he/she had never heard of or been exposed to in some way.

Beck (2004) further explained that psychotic symptoms can be evaluated in terms of three biases in attention and misattribution of thinking. The first two biases, egocentric and externalizing biases, are related to the development of positive symptoms, such as hallucinations and delusions. The egocentric/self-centered bias results in individuals ascribing personal significance to a host of irrelevant environmental stimuli and events. This then creates a hyper-attentiveness to the environment, which perpetuates the personal attribution of irrelevant details within the environment. The externalizing bias involves over attributing one's distress to external causes. Distress could be physical or psychological, but the distress is attributed to external factors, such as God, government agencies, or unknown entities. The third bias is dysfunctional attitudes. It involves the first two biases and further creates distress, resulting in negative symptoms. Dysfunctional attitudes ultimately reinforce the cognitive, biological, and environmental impairments that are present because of the disorder. Beck recognizes that not all aspects of schizophrenia can be accounted for by cognitions. The model is an extension of existing biological models and provides an explanation for why some

individuals with biological risk factors or predispositions go on to develop the disorder. Dysfunctional attitudes are created and perpetuated by combinations of biological/neurocognitive deficits and cognitive biases/misattributions of the self (e.g. defeatist performance beliefs; internalized stigma) and environment. One source of dysfunctional attitudes is the real, such as stigma, or perceived negative judgments of others. As a result of fear of these judgments and negative beliefs about their abilities, individuals voluntarily disengage from social relationships and interactions. Disengagement and withdrawal from social interactions can include decreased communication, expressive gestures and motivation. Social disengagement is a form of safety mechanism and becomes automatic.

Some evidence exists to support this model. Low self-esteem has been found to be present in individuals with schizophrenia in their first episode of psychosis (Vracotas, Iyer, Joobar, & Malla, 2010). Also, low self-esteem at the time of the first psychotic episode was related to global functioning as measured by the Global Assessment of Functioning (GAF) at six-months follow-up (Vracotas et al., 2010). Low self-esteem has been found to be associated with paranoid delusions in individuals with schizophrenia and depression (Bentall et al., 2008). Bentall and colleagues (2008) suggested that the explanation for this connection is that constant low self-esteem, dysfunctional attitudes, and fear of negative appraisals leads to the expectation the others will act malevolently. Also, negative expectancy appraisals and defeatist performance beliefs have been found to be associated with negative symptoms of schizophrenia (Couture et al., 2011). In general, negative symptoms can be divided into at least two factors, which are diminished experience and diminished expressivity. Couture and colleagues (2011) found that



negative expectancy appraisals and defeatist performance beliefs were significantly associated with the negative symptoms of diminished experience rather than diminished expressivity. Furthermore, cognitive behavior therapy (CBT) has been found to improve negative and positive symptoms of schizophrenia by focusing on negative self-thought, which includes low self-esteem, defeatist performance beliefs, and fear of negative appraisals (Gould et al., 2001; Rector & Beck, 2001). Negative self-appraisals and self-blame for illness has also been found to be associated with greater risk of relapse, or exacerbations of psychotic symptoms, in individuals with schizophrenia (Gumley et al., 2006).

### **Functional Outcome and Capacity**

Functional ability is traditionally assessed in one of three ways: self-report, observation, and performance-based. Within the framework of functional ability, functional capacity and outcome have been separately identified and examined. Typically, functional outcome is assessed via self-report questionnaires or clinician rated forms following interviews. Functional outcome can be considered measures of real-world performance, or what a person is actually doing in his/her daily life, regardless of what they are capable of (Horan et al., 2010). Functional capacity is what a person is capable of doing given optimal circumstances (Horan et al., 2010). Functional capacity is typically assessed using performance-based measures. These measures require an individual to actually complete various tasks, such as making a shopping list or planning for a trip, and participants are rated on his/her accuracy. Functional capacity was chosen as the primary dependent variable in the current study, rather than functional outcome. The neurocognitive abilities of reinforcement learning and effort have been found to be

related to specific brain areas and dysfunction in these areas have been found to disrupt performance. Because individuals have disrupted brain regions associated with these tasks, functional capacity was thought to be effected more directly by these abilities than functional outcome.

Many factors, personal and environmental, contribute to the discrepancy between capacity and outcome. As previously discussed, poor self-esteem, self-efficacy, and defeatist performance beliefs have all been found to be associated with poor functional outcome. Neurocognitive impairment in general has also been found to be related to functional capacity and outcome (Beck & Grant, 2008; Grant & Beck, 2008). However, the effect of specific cognitive dysfunctions, specifically reinforcement learning and/or effort, on functional capacity has not been causally evaluated in any population, including schizophrenia.

### **Research Aims and Study Hypotheses**

The aim of the current study is to provide information regarding the causal factors contributing to functional impairment.

**Hypothesis 1.** First, I hypothesize that the schizophrenia group will perform more poorly on the reinforcement learning task, effort task, and functional capacity, as well as have more defeatist performance beliefs than the control group. Prior research has demonstrated that individuals with schizophrenia are less able to learn from reinforcement, put forth less effort to gain a reward, have impaired functional capacity, and have higher defeatist performance beliefs. All of these variables have not been examined together in one study.

Confirming these findings prior to evaluating a causal model between the variables will be important. If one variable were found to not distinguish between the groups, this would suggest that the variable is not a unique contributor of functional capacity in schizophrenia and would suggest its removal from the causal chain.

**Hypothesis 2.** Furthermore I hypothesize that the more basic functions of reinforcement learning and effort will predict defeatist performance beliefs, which will predict functional capacity. The causal model begins with more basic neural processes that have been tied to specific brain pathways and continues to more complex behavioral impairments, specifically defeatist performance beliefs and functional capacity. The primary model proposed predicts an additive effect of the more basic variables onto the more complex (i.e. reinforcement learning and effort → defeatist performance beliefs → functional capacity).

Additional models will be tested in order to determine if a different causal model is more appropriate for the given variables. The second model reverses the causal path of the independent variables, such that defeatist performance beliefs predict both reinforcement learning and effort and both reinforcement learning and effort predict functional capacity. A third model will be examined in which once causal path is added to model 2 between defeatist performance beliefs and functional capacity. Finally, a fourth model will be tested that resembles a standard regression equation, predicting no causal relationship among the independent variables, only examining the impact all independent variables have on the dependent variable functional capacity.

Each of the variables of interest in the current study have been examined singularly or in some combination in individuals with schizophrenia. However, they

have not been examined together and in a causative fashion. As previously mentioned, understanding the neural and behavior causes of functional impairment can provide direction for both pharmacological and behavioral interventions aimed at increasing quality of life and independent living skills.

## CHAPTER 3

### METHOD

#### **Participants**

Seventy-five individuals were included in the current study. Fifty individuals had a diagnosis of schizophrenia and 25 served as healthy controls. Participants ranged in age from 18 to 64 years. Individuals were included in the study if they spoke English as their first language, and did not have a history of traumatic brain injury or any other medical condition or neurological disease/damage which has the potential to adversely affect central nervous system functioning (e.g., liver disease, HIV). Additionally, participants were excluded from participation if they had hearing or visual deficits that would not allow them to complete the study procedures; had a history of alcohol or substance abuse or dependence within the past six months; or currently used prescription or over-the-counter medications that could produce significant cognitive effects, other than those medications prescribed to treat schizophrenia. An additional exclusionary criterion for controls included a family history of schizophrenia or bipolar disorder in any first-degree relative, as determined through a structured interview. Schizophrenia and bipolar disorder have high heritability rates and non-affected first-degree relatives of individuals with schizophrenia and bipolar disorder have been found to share some overlapping features, or endophenotypes, of the disorders (Frantom, Allen, & Cross, 2008; Hughes et al., 2005; MacDonald & Schulz, 2009).

## Measures

Six domains were measured in the current study and included: clinical symptomatology; defeatist performance beliefs; effort; reinforcement learning; functional capacity; and estimated intelligence and cognitive functions. Descriptions of the tests used to measure these domains are provided below. Client demographic and clinical information including medical and family history were obtained from the phone screening, demographic forms and medical records.

**Clinical Symptom Measures.** Patients had an existing diagnosis of schizophrenia as identified by a treating psychiatrist or psychologist. In addition, clinical diagnosis was confirmed using the electronic version of the Structured Clinical Interview for DSM-IV-TR (eSCID; First, Gibbon, Spitzer, & Williams, 1996). Controls had no existing psychiatric disorder or neurological condition, which was also confirmed using the eSCID. In order to measure current clinical symptomatology, all participants were administered the Calgary Depression Rating Scale (CDRS; Addington, Addington, & Schissel, 1990), the Brief Psychiatric Rating Scale (BPRS; Overall & Gorham, 1962), the Scale for the Assessment of Positive Symptoms (SAPS; Andreasen, 1984), and the Scale for the Assessment of Negative Symptoms (SANS; Andreasen, 1983). The CDRS assesses depressive symptoms associated with schizophrenia. The SAPS, SANS, and BPRS assess affective, anxiety, and positive and negative symptoms.

***Electronic Structured Clinical Interview for the DSM-IV-TR.*** The electronic Structured Clinical Interview for DSM-IV-TR (eSCID) is a semi-structured interview identical to the paper version of the Structured Clinical Interview for DSM-IV-TR (SCID; First et al., 2001). The SCID is designed to identify clinical symptoms and determine

Axis I psychiatric diagnoses. The eSCID is an electronic, computer-based version of the SCID that allowed for cost and resource savings by eliminating the need to copy the 200+ page document for each participant seen. The electronic format also allowed for ease of use by automatically jumping to appropriate sections based on participant responses during the interview, rather than requiring the examiner to flip through pages to the appropriate section.

The eSCID was used to verify a diagnosis of schizophrenia, rule out the presence of several other conditions that exhibit similar symptoms, as well as confirm the lack of Axis I disorder in the healthy control group.

***Calgary Depression Rating Scale.*** The Calgary Depression Rating Scale for Schizophrenia (CDRS; Addington et al., 1990) was designed to assess severity of depression in individuals with schizophrenia. The nine item rating scale is based upon the Hamilton Depression Rating Scale and the Present State Examination, and has been reliably shown to measure depression specific to individuals with schizophrenia, separate from positive, negative and extrapyramidal symptoms present in the disorder. Each item is rated on a scale from 0 to 3 (absent to severe), based upon the individual's subjective report given during a clinical interview. A total score was derived by summing the nine items.

***Brief Psychiatric Rating Scale.*** The Brief Psychiatric Rating Scale (BPRS; Overall & Gorham, 1962) is an 18-item clinician administered rating scale designed to assess affective symptoms as well as symptoms of anxiety and positive and negative psychotic symptoms. Each item is rated on a scale from 1 to 7 (absent to extremely severe). The rating of each item is based on the individual's subjective report over the

previous two weeks or behavioral observations made by the clinician during the time of the interview. A total score was derived by summing the 18 items. Additionally, four factors have been identified within the BPRS using factor analysis and were reported in the current study (Mueser, Curran, & McHugo, 1997). The four factors are: 1) thought disturbance, composed of items rating grandiosity, suspiciousness, hallucinatory behavior, and unusual thought content (items 8, 11, 12, and 15), resulting in a minimum score of 4 and a maximum score of 28; 2) anergia, composed of items rating emotional withdrawal, motor retardation, uncooperativeness, and blunted affect (items 3, 13, 14, and 16), resulting in a minimum score of 4 and a maximum of 28; 3) affect, composed of items rating somatic concern, anxiety, guilt feelings, depressive mood, and hostility (items 1, 2, 5, 9, and 10), resulting in a minimum score of 5 and a maximum of 35; and 4) disorganization, composed of items rating conceptual disorganization, tension, and mannerisms and posturing (items 4, 6, and 7), resulting in a minimum score of 3 and a maximum of 21. These factors have remained stable in a three-year longitudinal study of individuals with schizophrenia (Long & Brekke, 1999).

***Scale for the Assessment of Positive Symptoms.*** The Scale for the Assessment of Positive Symptoms (SAPS; Andreasen, 1984) is a 34-item clinician administered rating scale designed to assess positive psychotic symptoms. Positive symptoms include hallucinations, delusions, bizarre behavior, and formal thought disorder. Global ratings are also evaluated and are used to represent overall severity within each of these four domains. Items are rated on a scale from 0 to 5 (absent to severe). The rating of each item is based on the individual's subjective report over the previous two weeks, as well as on the behavioral observations of the clinician during the time of the interview. A total



score was derived by summing all 34 items. A score for each subscale was also derived by summing all items, including the global rating, within each subscale.

***Scale for the Assessment of Negative Symptoms.*** The Scale for the Assessment of Negative Symptoms (SANS; Andreasen, 1983) is a 30-item clinician administered rating scale designed to assess negative psychotic symptoms. Negative symptoms are organized in 5 core domains: affective flattening, alogia, avolition, anhedonia, and attentional impairment. Global ratings are also evaluated and are used to represent overall severity within each of these five domains. Items are rated on a scale from 0 to 5 (absent to severe). The rating of each item is based on the individual's subjective report over the previous week, as well as on the behavioral observations of the clinician during the time of the interview. A total score was derived by summing all 30 items. In addition, two subscale scores were calculated based on current research suggesting two general domains of negative symptoms, which are emotional expressivity and motivation/pleasure (Blanchard & Cohen, 2006; Kirkpatrick et al., 2011; Strauss et al., in press). Emotional Expressivity is the sum of items 1 – 15 (affective flattening and alogia). Motivation/pleasure is the sum of items 16 – 26 (avolition and anhedonia-asociality). Attention is not included in either subscale.

**Reinforcement Learning.** One measure of reinforcement learning was used in the current study in order to evaluate the ability to learn from positive feedback and to learn to avoid losses.

***Reinforcement learning task.*** The reinforcement learning task (RL; Strauss et al., 2012) is based off of a reinforcement learning task used by Pessiglione and colleagues (2006) and adapted for use in individuals with schizophrenia. It was administered via E-

Prime (Psychology Software Tools) and run on a desktop computer with a 17-in monitor. There are two phases to the task, an acquisition and a learning phase. During the acquisition phase, participants were presented with 4 pairs of landscape stimuli, 1 pair at a time. Two pairs have a potential gain if the correct item is selected and two pairs have a potential loss if the incorrect item is selected. For the gain pairs, if the correct item is selected an image of a nickel coupled with the feedback “Win!,” is presented, whereas if the incorrect item is selected, the feedback “Not a winner, Try again!” is presented. For the gain pairs, the correct response was reinforced on 90% of trials in one pair and on 80% of trials in the other pair. For the potential loss pairs, or loss avoidance pairs, selection of the correct response resulted in the feedback “Keep your money!,” whereas selection of the incorrect item resulted in the feedback “Lose!” If the correct response in the loss avoidance pairs was selected, participants avoided a loss 90% or 80% of the time. The acquisition phase consisted of 160 trials with all pair types presented in a randomized order and each pair being shown 40 times. The 160 trials are divided into 4 acquisition blocks of 40 trials each.

Following acquisition, the transfer test phase consisted of 64 trials. The original 4 training pairs were presented 4 times and 24 novel pairings were each presented twice. Novel pairings consisted of each trained item being presented with one another (e.g., an item that had been a 90% winner is paired with both items from the 80% gain pair, the 90% loss-avoidance pair, and the 80% loss-avoidance pair). No feedback was given during this phase. Variables that can be examined from this task include training and test phase accuracy scores, feedback valance (gain versus loss avoidance), and probability. Training phase accuracy was chosen as the independent variable for the current study.

**Effort.** One measure of effort was used in order to assess the level of effort one is willing to expend in order to gain rewards.

*The Effort Expenditure for Reward Task.* The Effort Expenditure for Reward Task (EEfRT; Treadway, Buckholtz, Schwartzman, Lambert, & Zald, 2009) is a multi-trial game in which participants are given an opportunity on each trial to choose between an easy and a hard task in order to obtain monetary rewards. The task was originally created in MATLAB, but a stand-alone version was provided by the tasks author, Michael Treadway, via Dropbox. The program was run on the same desktop computer with a 17-in monitor. For all trials of either difficulty, participants made repeated keyboard button presses within an allotted period of time. Each button press adds a lever inside of a bar viewed onscreen. Participants were eligible to win money for each trial if they successfully completed the trial (i.e., filled the bar within the prescribed time period). Successful completion of hard-task trials requires the participant to make 100 button presses within 21 seconds, using his/her non-dominant pinky finger, while successful completion of easy-task trials requires the participant to make 30 button presses within 7 seconds, using his/her dominant index finger. Participants were eligible to win \$1.00 on each easy-task trial they successfully completed and were eligible to win higher amounts varying between \$1.24 and \$4.30 on each hard-task trial they successfully completed. Each trial had a probability of providing monetary gain if successfully completed. Trials had three levels of probability: 88%, 50% and 12%. Probability levels applied to both the hard and easy tasks, and there was an equal proportion of each probability level across the experiment. Each level of probability

appears once in conjunction with each level of hard-task reward value. Trials were presented in the same order for every participant.

Participants had 5-seconds at the beginning of each trial to choose the easy or hard task. During this time, the probability level and monetary values were presented. If a participant did not make a choice within 5-seconds, the difficulty level was randomly assigned for that trial. After making a choice, a 1-second 'Ready' screen was presented followed by a white bar in the center of a black screen, at which time the participant began pressing the button corresponding to the difficulty level chosen. Following task completion or after the task time had elapsed, a 2-second feedback screen informing the participant that the task was successfully or unsuccessfully completed was presented. If the task was successfully completed, an additional 2-second feedback screen informing the participant whether or not money had been won for that trial was presented.

In addition to the reimbursement rate given for participating in the study, participants won the actual monetary value of two randomly selected trials that money is earned. The game lasted 20 minutes, regardless of easy or hard task selection throughout the game. Because of the time difference between easy and hard tasks, the total number of trials that participants played depended on the choices made. Making more hard-task choices reduced the total number of trials, which could reduce the number of high-value, high-probability trials that might appear towards the end of the playing time and this trade-off will be explained to participants. The goal of this trade-off is to ensure that always choosing the easy or always choosing the hard option could not lead to an 'optimal' strategy. Also, varying monetary reward levels, probability, loss of time for future trials, and brief decision periods makes formal calculation of an optimal response

selection difficult. Overall, the task reflected individual differences in willingness to expend effort for a given level of expected reward value.

Because the number of trials a participant completed during the task varies upon the choices made during the 20 minutes, the first 50 trials were used in data analysis. The mean proportion of hard-task choices was calculated for each participant and this served as a within-subjects variable in the ANOVA and the intervening variable in the path analysis.

**Defeatist Performance Beliefs.** One measure of defeatist performance beliefs was administered in order to examine one's beliefs about his/her ability to perform tasks.

*Defeatist Performance Beliefs from the Dysfunctional Attitudes Scale.* The Defeatist Performance Beliefs (DPB) subscale of the Dysfunctional Attitudes Scale (DAS; Weissman, 1978) is a 15-item self-report questionnaire containing statements about one's ability to perform tasks and the likelihood of success. Participants rate how strongly they agree with each item on a scale from 1 to 7 (agree totally to disagree totally). An example item is, "If you cannot do something well, there is little point in doing it at all". A total score is calculated by adding all 15-items.

**Functional Capacity.** Functional capacity was assessed using the UCSD performance based skills assessment, a performance-based measure of functional capacity.

*UCSD Performance-Based Skills Assessment.* The UCSD Performance-Based Skills Assessment (UPSA; Patterson, Goldman, McKibbin, Hughs, & Jeste, 2001) is a performance-based measure of everyday functioning. Participants are asked to complete a number of tasks to determine skills in five areas: planning recreational activities,

finance, communication, transportation, and household chores. Planning recreational activities requires participants to read two story scenarios and plan for a trip to those locations accordingly (e.g. a trip to the zoo and to the beach). The finance tasks require participants to count change and pay a bill by check. The communication tasks require participants to make mock telephone calls using various instructions, such as rescheduling a medical appointment or demonstrating the number they would dial in case of emergencies. The transportation tasks require participants to read bus schedules to determine the cost of a ride and which bus lines to travel. The household chores task requires participants to read a recipe for rice pudding, look for the ingredients in a mock pantry, and write a shopping list of the missing items they need to get from the store in order to complete the recipe. Each of the five subscales yield raw scores, which are transformed into a 0 to 20 scale by dividing the subscale raw score by the total points possible on that subscale and multiplying by 20. The five transformed subscale scores are summed, yielding a summary score ranging from 0 to 100.

The UPSA was developed for use with psychiatric patients (Patterson et al., 2001). Performance has been found to be more impaired in individuals with schizophrenia than controls (Patterson et al., 2001).

**Estimated Intelligence and other Cognitive Functions.** Three subtests from the Wechsler Adult Intelligence Scale, third edition (WAIS-III; Wechsler, 1997), Block Design, Vocabulary, and Matrix Reasoning, will be used to calculate an estimated current intelligence and estimated premorbid intelligence. The regression equation used to estimate current full scale IQ uses the Vocabulary and Block Design subtests [Ringe, Saine, Lacritz, Hynan, & Cullum, 2002;  $(VO \text{ Scaled Score} \times 2.727) + (BD \text{ Scaled Score}$

x 2.727) + 42.535]. The regression equation used to estimate premorbid intelligence uses the Vocabulary and Matrix Reasoning subtests [Schoenberg, Scott, Duff, & Adams, 2010; 45.997 + .652 (VO raw score) + 1.287 (MR raw score) + .157 (Age in years) + 1.034 (Education) + .652 (Ethnicity) – 1.015 (Gender)].

The Digit Symbol Coding and Digit Span subtests from the WAIS-III were also included to assess processing speed and working memory, respectively. Processing speed and/or working memory may negatively impact the primary outcome variables, so these subtests were included in order to evaluate the impact, if any, these cognitive functions are having on the primary outcome variables.

***WAIS-III Block Design Subtest.*** The Block Design subtest from the WAIS-III (Wechsler, 1997) requires an individual to assemble red and white blocks to match images of increasing complexity within a stimulus book and is a measure of perceptual reasoning. Total raw scores are converted to age-corrected scaled scores.

***WAIS-III Vocabulary Subtest.*** The Vocabulary subtest from the WAIS-III (Wechsler, 1997) requires an individual to define words of increasing difficulty and is a measure of vocabulary knowledge. Total raw scores are converted to age-corrected scaled scores for the current estimated full scale IQ and maintained as raw scores for the premorbid full scale IQ.

***WAIS-III Matrix Reasoning Subtest.*** The Matrix Reasoning subtest from the WAIS-III (Wechsler, 1997) requires an individual to solve visual puzzles of increasing complexity within a stimulus book and is a measure of perceptual reasoning. Total raw scores are maintained for the premorbid full scale IQ score.

***WAIS-III Digit Symbol Coding Subtest.*** The Digit Symbol Coding subtest from the WAIS-III (Wechsler, 1997) requires an individual to match numbers and corresponding symbols within two minutes. It is a measure of processing speed. Total raw scores are converted to scaled scores and correlated with the primary outcome variables.

***WAIS-III Digit Span Subtest.*** The Digit Span subtest from the WAIS-III (Wechsler, 1997) requires an individual to repeat a series of numbers of increasing length in the forward order for the first portion, followed by repetition of a series of number of increasing length in the reverse order. It is a measure of working memory. Total raw scores are converted to scaled scores and correlated with the primary outcome variables.

## **Procedure**

Participants were recruited from the University of Nevada, Las Vegas, community mental health centers, and the community at large. Participants were recruited through posted advertisements as well as presentations given to treating psychologists and psychiatrists in community mental health centers, which resulted in referrals to the study. Mojave Adult, Family, and Child Services, (Mojave Mental Health) an affiliate of the University of Nevada, Reno medical school as well as Southern Nevada Adult Mental Health allowed presentations and postings by our researchers at their facilities. Participants received monetary compensation at a rate of \$10/hour, prorated to \$5 for every half an hour. Compensation ranged from \$40-\$80 per participant. Study procedures were approved by the UNLV IRB for protection of human subjects. Facilities authorization was obtained from Jason Schwartz, Mojave Mental Health Director of Community Services, and approved by the UNLV IRB to allow testing at Mojave Mental



Health. Office space was provided and participants were tested on location at 4000 East Charleston Boulevard, Las Vegas, Nevada 89104.

Individuals interested in participating in the study initially called a private study line located in the Neuropsychology research laboratory on the UNLV campus. Individuals recruited at Mojave mental health completed the phone screening in person at Mojave mental health. Before answering any questions, participants were given a brief description of study procedures, including initial screening questions, and asked to provide verbal consent to be asked the initial screening questions. Once verbal consent was obtained, participants answered questions to determine eligibility for participating. Individuals that met initial selection criteria on screening were scheduled to complete additional testing procedures at the UNLV Neuropsychology research laboratory or in the case of individuals at Mojave mental health were scheduled to complete additional testing procedures on location. Before participants began study procedures, written informed consent was obtained and questions were encouraged.

Once informed consent was reviewed and obtained, participants completed diagnostic and screening procedures. As part of these procedures, basic demographic information was collected, followed by the eSCID to determine the presence or absence of Axis I psychiatric disorders, and a 15-minute interview assessed current symptomatology.

After diagnostic and screening procedures were completed and it was determined that the participant was eligible, the remaining battery of neurocognitive tests were administered in a fixed order. The interviews, questionnaires and neuropsychological tests used in this study were part of a larger battery of tests. All testing was conducted by

trained doctoral level graduate students in a quiet private room at either the UNLV Neuropsychology research laboratory or at Mojave Mental Health Center. Participants were provided breaks whenever requested or as deemed appropriate by the examiner in order to control for fatigue effects, alleviate anxiety, and maintain motivation.

### **Data Analysis**

**Data entry and screening.** Trained graduate students and research assistants scored all tests according to standardized procedures. Data was entered into a Microsoft Excel database. Scoring and data entry was double checked by visual inspection by trained individuals. Data was also evaluated for assumptions of parametric tests (described in detail in the next section).

**Preliminary analyses.** Basic demographic information was calculated before primary analyses were completed. One-way ANOVAs were conducted to examine differences between groups on these basic demographic variables.

In addition to basic demographic information, diagnostic and clinical variables in the schizophrenia group were calculated. These variables include, number of hospitalizations, and current symptomatology as obtained from the clinical symptom ratings scales (BPRS, CDRS, SAPS, and SANS). Finally, type of medication and the frequency of each drug class by patients were calculated. To ease comparability across antipsychotic generation and dosage, antipsychotic medication dosage was converted to a chlorpromazine equivalent dosage (Woods, 2003). The chlorpromazine equivalent dosage was calculated and reported as an additional clinical symptom descriptive.

### **Primary analyses.**

***Hypothesis 1.*** Univariate ANOVAs were used to investigate the differences between groups on the primary variables, i.e. reinforcement learning performance, effort, defeatist performance beliefs, and functional capacity. In these analyses, the test scores will serve as the dependent variable, while group membership is the independent variable. It was hypothesized that participants in the schizophrenia group would perform the worst on the behavioral measures (reinforcement learning, effort, and functional capacity) and would have more defeatist performance beliefs than healthy controls. These analyses would confirm prior research demonstrating these impairments in individuals with schizophrenia, although they have never all been collected in the same study.

***Hypothesis 2.*** In order to examine the second hypothesis, a path analysis was conducted. Path analysis allowed the examination of each dependent variable's unique prediction of the major outcome variable, functional capacity.

Path analysis is an extension of regression and allows for the comparison of multiple causal models. Regression analyses are performed for each of the relationships specified within a path model, and the weights predicted by the model are subsequently compared to the correlation matrix that was obtained from the actual data. Model fit indexes allow for comparisons between models in order to identify which of a number of competing models provides the best explanation of the observed data.

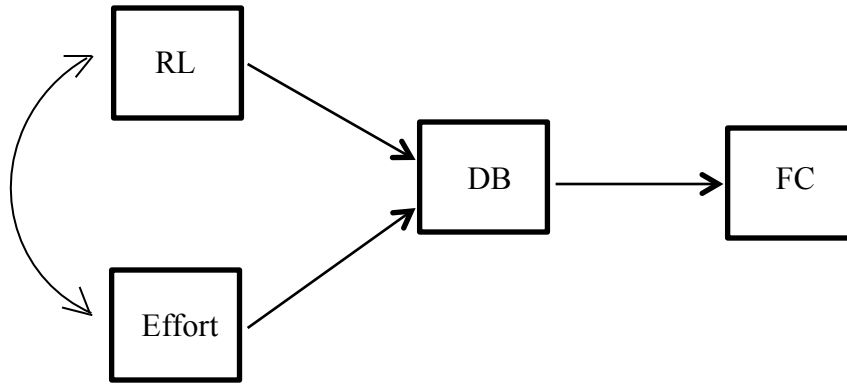
The adequacy of fit of proposed models is determined using a number of procedures. Path coefficients, which are standardized regression coefficients, can be evaluated to determine whether individual causal relationships in the hypothesized model are present in the actual data.

Figures 1-4 represents the path models evaluated. In these models, each variable is assessed using a single indicator, or total score from each relevant measure used to assess each variable. The models depict the relationships between the independent, intermediary and dependent variables. In the first model reinforcement learning and effort are the independent variables, defeatist performance beliefs are the intermediary variable, and functional capacity is the dependent variable. Models 2-4 maintains these variables but in varying arrangements.

In the models, causative relationships between the variable are indicated by single arrows from one variable to another, in order to indicate their hypothesized causative influence. The direction of each arrow indicates the direction of the hypothesized causal influence. The causative influences of the paths from one variable on another are determined by a standardized regression coefficient (beta). Exogenous/Independent variables in the model have no explicit causes as indicated by no arrows leading to them. The exception to this is when exogenous variables are correlated, which is indicated by a curved, bidirectional arrow. Endogenous variables do have arrows leading to them. Endogenous intervening variables have both incoming and outgoing arrows, and dependent endogenous variables have only incoming causal arrows.

Figure 1

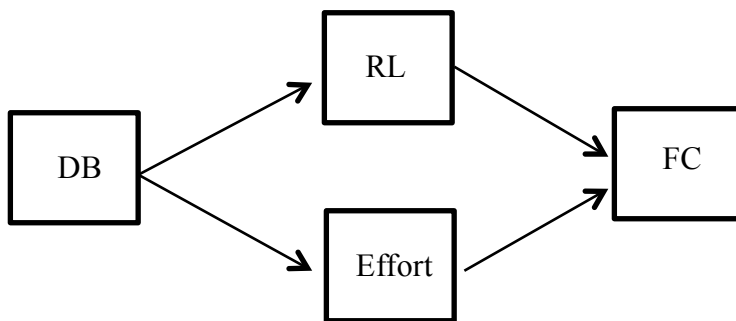
*Model 1. Model Reflecting Direct Contributions of Reinforcement Learning and Effort on Defeatist Performance Beliefs*



*Note.* RL = Reinforcement Learning. DB = Defeatist Performance Beliefs. FC = Functional Capacity.

Figure 2

*Model 2. Model Reflecting Direct Contributions of Defeatist Performance Beliefs on Reinforcement Learning and Effort*

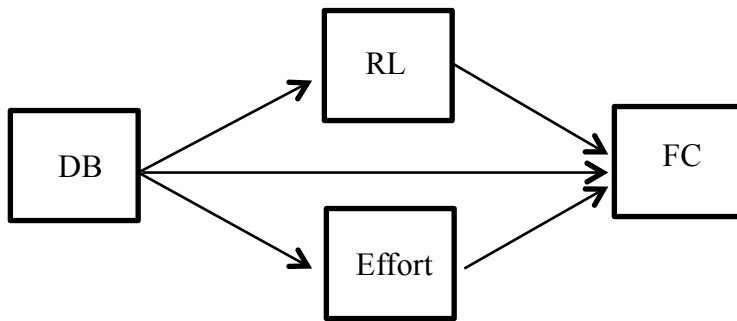


*Note.* RL = Reinforcement Learning. DB = Defeatist Performance Beliefs. FC = Functional Capacity.

Figure 3

*Model 3. Complete Model Reflecting Direct Contributions of Defeatist Performance Beliefs on Reinforcement Learning, Effort, and Functional Capacity*

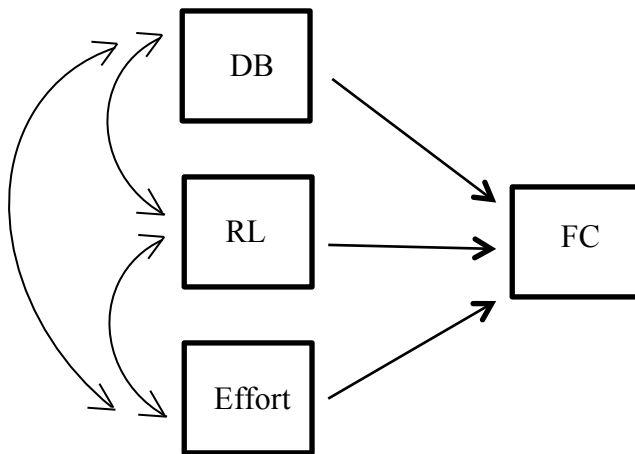
*Beliefs on Reinforcement Learning, Effort, and Functional Capacity*



*Note.* RL = Reinforcement Learning. DB = Defeatist Performance Beliefs. FC = Functional Capacity.

Figure 4

*Model 4. Standard Regression Model*



*Note.* RL = Reinforcement Learning. DB = Defeatist Performance Beliefs. FC = Functional Capacity.

## CHAPTER 4

### RESULTS

#### **Data Screening**

Initial screening and evaluation of the data took place in order to ensure accuracy of the data and assumptions of parametric tests were met.

**Accuracy of data file.** Frequency and range statistics were evaluated in order to ensure no duplicate cases existed and that all data fell within range. Data was also examined for missing cases, of which none were present.

**Outliers.** Scores that fall beyond 3.29 standard deviations from the mean are considered univariate outliers. Z-scores were calculated for each variable used in the path analysis in order to determine the presence of univariate outliers. For all variables, the largest z-score was an UPSA score of -3.26, indicating no univariate outliers.

Multivariate outliers are evaluated by a Mahalanobis distance of  $p < .001$  for the  $\chi^2$  value. Five variables are used in all path models, so the  $\chi^2$  value with a significance of  $p < .001$  is 20.52. Mahalanobis distance calculated using linear regression indicated distances ranging from 0.19 to 11.55, indicating no multivariate outliers.

**Normality.** Skewness and kurtosis statistics are reported in Table 1. Generally, skewness values within +/- 1 and kurtosis values within +/- 1.5 are considered to indicate normally distributed data. As can be seen from the table, the scores for all tests were generally within these cutoffs for normally distributed data, although the UPSA total score was -1.10 for skewness. Thus, no transformation was required to normalize the data.

Table 1

*Skewness and Kurtosis Values for the Primary Variables*

	Skewness	Kurtosis
UPSA	-1.10	1.02
DPB	-0.10	-1.27
Effort	0.27	0.01
Reinforcement Learning	-0.32	-1.08

*Note.* UPSA = UCSD Performance Based Skills Assessment; DPB = Defeatist Performance Beliefs.

**Linearity.** Through examination of scatter plots of all dependent variables and plots of the residuals from regression analyses, the variables exhibited a linear relationship.

**Multicollinearity and singularity.** Singularity is considered the presence of a redundant variable (i.e., a variables composed of a combination of 2 or more of the other variables). All variables used are discrete and thus not singular. Multicollinearity was examined using a correlation matrix. Table 2 presents the correlations among all variables used in the path analysis. Variables are considered multicollinear if the correlation between them are  $> 0.90$ . As can be seen in Table 2, there were no correlations greater than 0.54 between any variable, suggesting the absence of multicollinaerity.

**Residuals.** After model estimation, the residuals (residual covariances, i.e. errors) should be small and centered around 0. Some can be large but symmetrical. When large residuals are found, this may suggest that the model is not a good fit. In each path model, medium sized residuals were present. Tabachnick and Fidell (2013) suggest examining



the Lagrange Multiplier (LM) when large residuals are found and to consider adding paths to the model. However, this may also turn path analysis into an exploratory as opposed to a confirmatory analysis. Considering the paths were determined *a priori* and the number of paths are restricted by the small number of variables included in the model, no additional paths were included.

Table 2

*Correlations among Variables to Examine Multicollinearity*

Variable	UPSA	DPB	Effort	RL
UPSA	1			
DPB	-0.39	1		
Effort	0.27	-0.10	1	
RL	0.54	-0.36	0.14	1

Note. UPSA = UCSD Performance Based Skills Assessment; DPB = Defeatist Performance Beliefs; RL = Reinforcement Learning.

**Preliminary Analyses**

Demographic data is provided in Table 3. As indicated in Table 3, groups did not significantly differ on age, gender, ethnicity, or handedness. There was a significant difference between groups for education and current and premorbid estimates of IQ, such that the patients had lower years of education and lower estimates of current and premorbid IQ.

Table 3

*Demographic Information by Group*

Variable	Group		<i>F</i>	<i>p</i>
	Control	Schizophrenia		
Age	38.08 (14.97)	40.76 (12.51)	0.67	0.42
Education	14.20 (1.94)	12.42 (1.85)	14.93	>0.001
Premorbid Estimated IQ*	111.53 (6.87)	90.36 (14.75)	46.24	>0.001
Current Estimated IQ	106.89 (11.22)	84.31 (14.06)	48.81	>0.001
			$\chi^2$	<i>p</i>
Gender (% male)	52.0	54.0	0.03	0.87
Ethnicity (%)			2.04	0.73
Caucasian	52.0	50.0		
African American	24.0	26.0		
Hispanic/Latino	8.0	14.0		
Asian American	8.0	2.0		
Biracial	8.0	8.0		
Handedness (% right)	96.0	88.0	1.26	0.26

*Note.* \* = premorbid estimated IQ based on a regression equation is reported here,

although the term ‘premorbid’ does not apply to controls.

These differences in IQ and years of education were expected based on prior research indicating that cognitive deficits are a core feature of schizophrenia and as such, individuals with schizophrenia typically obtain lower IQ scores than the general population (Aylward, Walker, & Bettes, 1984; Hedman, van Haren, van Baal, Kahn, & Hulshoff Pol, 2013), and in comparison to individuals with other psychiatric disorders (Goldberg et al., 1993). These intellectual deficits predate onset of illness (Caspi et al., 2003; Reichenberg et al., 2002), and are associated with decreased academic performance

particularly in late adolescence as the onset of the disorder approaches (Allen, Frantom, Strauss, & van Kammen, 2005; Fuller et al., 2002; Gunnell, 2002; Strauss et al., 2012; van Oel, Sitskoorn, Cremer, & Kahn, 2002). Given that diminished IQ and associated decreases in academic performance and years of education obtained are characteristic of schizophrenia, IQ and education differences were not controlled through covariance or other procedures when conducting the main analyses, since controlling for these differences would essentially control for the independent variable of interest (i.e., diagnosis).

Symptomatology information for each group is summarized in Table 4. As expected, groups significantly differed on all symptom measures, such that the patient group had significantly higher clinical symptomatology (i.e. depression, positive and negative symptoms, global assessment of functioning) than the control group.

Demographic and illness severity variables were correlated with the primary outcome variables in the schizophrenia group in order to determine if any significant associations were present (see Table 5). A Bonferroni correction was made to correct for Type I error because of the high number of correlations being examined. The Bonferroni correction took into account the 8 demographic variables being correlated with the 4 primary variables, resulting in a new  $p$  value of 0.006. Age, education, chlorpromazine equivalent, number of hospitalizations, length of illness, and global assessment of functioning were not significantly correlated with any primary outcome variables. Reinforcement learning was significantly correlated with current IQ,  $r = 0.43$ ,  $p < 0.006$ , and UPSA was significantly correlated with premorbid IQ and current IQ,  $r = 0.53$  and  $0.55$ ,  $p < 0.006$ .

Table 4  
*Symptom Ratings by Group*

Variable	Group		<i>F</i>
	Control	Schizophrenia	
CDRS	0.44 (1.33)	2.32 (2.65)	11.11**
BPRS	20.32 (2.46)	38.64 (8.87)	102.06**
Thought Disturbance	4.24 (0.60)	12.86 (4.71)	82.59**
Anergia	4.24 (0.60)	7.10 (3.38)	17.55**
Affect	6.52 (1.71)	11.34 (4.05)	32.36**
Disorganization	3.20 (0.65)	5.00 (2.20)	15.90**
SAPS	0.16 (0.63)	28.66 (18.77)	57.23**
Hallucinations	0.00 (0.00)	1.36 (1.17)	33.53**
Delusions	0.01 (0.03)	0.93 (0.66)	47.43**
Bizarre Behavior	0.01 (0.04)	0.28 (0.48)	7.78**
Thought Disorder	0.00 (0.00)	0.64 (0.64)	24.51**
SANS	4.00 (7.38)	34.38 (22.49)	43.03**
Emotional Expressivity	1.60 (3.85)	13.92 (14.10)	18.29**
Motivation/Pleasure	1.36 (2.68)	12.70 (9.96)	31.12**
GAF	83.54 (10.59)	41.12 (9.04)	319.17**
Length of Illness (years)	-	18.86 (10.34)	
Hospitalizations	-	7.34 (7.88)	

*Note.* \*\* =  $p < .001$ ; CDRS = Calgary Depression Rating Scale; BPRS = Brief

Psychiatric Rating Scale; SAPS = Scale for the Assessment of Positive Symptoms; SANS

= Scale for the Assessment of Negative Symptoms; GAF = Global Assessment of

Functioning.

Table 5

*Correlations among Demographic and Illness with Primary Variables in Schizophrenia*

Variable	Effort	RL	DPB	UPSA
Age	0.02	-0.13	0.05	0.19
Education	-0.35	0.12	-0.18	0.26
Premorbid IQ	0.16	0.28	-0.16	0.53**
Current IQ	0.19	0.43**	-0.17	0.55**
CPZ Equivalent	-0.29	-0.26	-0.07	-0.32
Hospitalizations	-0.07	0.25	-0.17	-0.05
Length of Illness	0.11	-0.03	-0.04	0.12
GAF	0.14	0.19	-0.32	0.31

*Note.* \*\* =  $p < .006$ ; SZ = schizophrenia; RL = Reinforcement Learning; DPB = Defeatist

Performance Beliefs; UPSA = UCSD Performance Based Skills Assessment

Medication information for the patient group can be found in Table 6. As can be seen from the table, all but one patient participant (2%) was taking some form of psychiatric medication. Eighty-six percent of the patient participants were taking an antipsychotic, 12% were taking a typical and 78% were taking an atypical, 36% were taking a mood stabilizer, 48% were taking an antidepressant, and 6% were taking lithium. Daily chlorpromazine equivalent dosage (CPE) in milligrams (Woods, 2003) was calculated for all participants. CPE dosage involves converting all antipsychotic medication, regardless of brand or class, into equivalent dosages to ease comparison.

Table 6

*Medication Information for the Schizophrenia Group*

Medication	Value
Chlorpromazine Equivalent	541.36 (397.33)
Antipsychotics	88%
Atypical	78%
Typical	12%
Mood Stabilizer	36%
Antidepressant	48%
Lithium	6%
No Medication	2%

**Primary analyses**

*Hypothesis 1.* One-way ANOVAs were used to investigate the differences between groups on the primary variables, i.e. reinforcement learning performance, effort, defeatist performance beliefs, and functional capacity. Table 7 summarizes these results. As can be seen from the table, the patient and control groups significantly differed on all primary variables, such that the patient group is performing significantly worse on the reinforcement, effort, and functional capacity measures, and they are experiencing significantly more defeatist performance beliefs. The patient and control groups also significantly differed on the total number of effort trials completed (i.e. trials started but not completed in time and were thus discontinued), such that the patient group completed significantly less trials. It was unclear why the patient group was completing less trials, and if this was impacting overall effort performance. To evaluate this, a one-way ANOVA evaluating effort choice performance between groups that excluded participants that completed less than 10% of the first 50 trials was completed. The difference between

groups remained significant,  $F(1,67) = 6.19, p = 0.015$ . Also to examine factors associated with effort and trial completion, these variables were correlated with various variables that may have contributed to poorer performance on this measure in the patient group. Specifically, these variables were current and premorbid intelligence estimates, working memory, processing speed, and CPE dose. Results of these correlations are presented in Table 8. As can be seen from the table, number of trials completed was not significantly correlated with any other variable, including effort choice. Effort choice was significantly correlated with CPE dose. With the available results, CPE dose was at least associated with effort choice in the patient group. It is possible the CPE dose diminishes motivation to some extent, although it is unclear whether this association is due to the medication itself, or other factors associated with increased medication dose, such as illness severity. In order to further examine the effect of CPE dose on effort choice, a regression analysis was conducted using CPE dose as the sole predictor of effort choice. The regression analysis found CPE dose to account for only 8.5% of the variance in effort performance. A one-way ANOVA was conducted between groups using the predicted effort choice values derived from the regression equation. In this analysis, significance between groups in effort performance remained significant,  $F(1,74) = 46.10, p < .001$ . Overall, these results support the hypothesis that participants in the schizophrenia group perform worse on the behavioral measures (reinforcement learning, effort, and functional capacity) and have more defeatist performance beliefs than the control group. These analyses support prior research demonstrating these impairments in individuals with schizophrenia, although all variables had not been examined in the same study.

Table 7

*Main Variable Information by Group*

Variable	Group		<i>F</i>
	CN ( <i>n</i> =25)	SZ ( <i>n</i> =50)	
DPB	31.40 (13.22)	51.20 (14.50)	32.90**
Reinforcement Learning Effort	78.07 (11.09)	69.45 (12.34)	8.68**
Easy vs Hard	0.53 (0.26)	0.39 (0.24)	5.50*
Completed	0.99 (0.03)	0.95 (0.09)	4.98*
Total trials	56.32 (9.01)	60.42 (9.00)	3.48
UPSA total	87.50 (6.71)	75.53 (14.53)	15.25**
Activities/Planning	18.46 (1.51)	16.27 (2.64)	14.77**
Finance	17.74 (2.69)	14.07 (3.95)	17.48**
Communication	15.82 (2.75)	14.49 (3.68)	2.57
Transportation	17.87 (2.13)	15.40 (3.92)	8.59**
Household	17.60 (3.27)	15.30 (4.78)	4.68*

*Note.* \* =  $p > .05$ ; \*\* =  $p > .001$ ; CN = controls; SZ = schizophrenia; DPB = defeatist performance beliefs; UPSA = UCSD Performance Based Skills Assessment.



Table 8

*Factors Potentially Impacting Effort Choice and Trial Completion in Schizophrenia*

Variable	Effort	Completed
Premorbid IQ	0.16	0.06
Current IQ	0.19	0.07
Processing Speed	0.03	0.18
Working Memory	0.01	0.14
Chlorpromazine Dose	-0.29*	-0.24
Completed	0.01	1

Note. \* =  $p < .05$

**Hypothesis 2.** Figures 5-8 depict the final proposed models with path coefficients inserted. Both groups were included in the path and regression analyses. Including only the schizophrenia group attenuated the correlations between variables by restricting the range. The majority of the schizophrenia participants were low functioning, requiring social security disability, as well as housing and other forms of assistance. If higher functioning individuals with schizophrenia had been included in the sample, the range of scores would not have been as restricted. However, this was not the case, so including controls in the analyses improved the range of scores and strengthened the correlations. Correlations were conducted between the variables used in the path and regression analyses for the schizophrenia group and the entire sample for comparison purposes (see Table 9). As can be seen from the table, including the controls did not change the pattern of correlations in a meaningful way, but provided more variability in scores thus strengthening the correlations.

Table 9

*Primary Variable Correlations in the Schizophrenia and Total Samples*

Variable	Schizophrenia (n=50)				Total Sample (N=75)			
	UPSA	DPB	Effort	RL	UPSA	DPB	Effort	RL
UPSA	1				1			
DPB	-0.19	1			-0.39**	1		
Effort	0.20	0.11	1		0.27*	-0.10	1	
RL	0.49**	-0.21	0.17	1	0.54**	-0.36**	0.14	1

*Note.* \* =  $p < .05$ ; \*\* =  $p < .01$ . UPSA = UCSD Performance Based Skills Assessment; DPB = Defeatist Performance Beliefs; RL = Reinforcement Learning.

There are a number of methods utilized to determine the significance of proposed models (Tabachnick & Fidell, 2012). Each method has certain limitations, such as restrictions caused by too small or too large of a sample size, lack of clear cut-offs for indicating a good fit, or lack of distinct ranges of fit scores (Tabachnick & Fidell, 2012). As such, multiple fit statistics are typically provided when analyzing path models. Path analysis was run using EQS software, which provides a number of commonly used methods for evaluating a model. There are a number of goodness-of-fit indices that can be examined. These statistics are provided in Table 10 and discussed in the following sections for Models 1-3. These statistics could not be computed for Model 4 because the model degrees of freedom were 0. This model was tested using a standard regression analysis.

*Independence Chi Square.* This tests the hypothesis that there is no relationship among the variables and should always be significant (Tabachnick & Fidell, 2012).

Because the same variables were used in all models, the chi-square was identical for all models,  $\chi^2 = 46.24$ ,  $p < .001$ . This suggests that there is some relationship among the variables.

*Chi-square.* The model chi-square, which examines the goodness of fit between the sample covariance matrix and the estimated population covariance matrix, should ideally be non-significant (Tabachnick & Fidell, 2012). As can be seen from Table 10, the  $\chi^2$  for Model 1 was significant while the  $\chi^2$  for Model 2 and Model 3 were not significant. These results suggest that both Model 2 and Model 3 may provide a good fit for the data. However, Model 2 approaches significance, while Model 3 does not, suggesting Model 3 is the best fitting model based on chi-square.

*Normed Fit Index (NFI).* The Normed Fit Index (NFI) compares the model  $\chi^2$  to the independence model  $\chi^2$ . A NFI value  $> 0.95$  is considered a good fitting model (Tabachnick & Fidell, 2012). Model 1's NFI = 0.52, Model 2's NFI = 0.88, and Model 3's NFI = 0.98. These results again suggest that Model 3 provides the best fit for the data.

*Comparative Fit Index (CFI).* This fit index compares the estimated and independent model  $\chi^2$  distributions based on noncentrality parameters. Again, larger values indicate better fitting models, with CFI  $> 0.95$  suggestive of a good fit (Tabachnick & Fidell, 2012). Model 1's CFI = 0.50, Model 2's CFI = 0.92, and Model 3's CFI = 1.00. These results further support the better fit of Model 3 than Model 1 or 2 for the data.

*Goodness-of-fit Index (GFI).* GFI is a weighted proportion of variance in the sample covariance accounted for by the estimated population covariance matrix. It can be considered analogous to  $R^2$  in multiple regression. A GFI  $> 0.90$  is considered an

indication of a good fitting model (Savalei & Bentler, 2006). For Model 1, GFI = 0.89, Model 2 GFI = 0.97, and Model 3 GFI = 0.99. These results further support the better fit of Model 3 and 2 than Model 1, with Model 3 providing the best fit.

*Adjusted Goodness-of-fit Index (AGFI).* AGFI is adjusted for the number of parameters estimated in the model. If the AGFI is satisfactory in 2 competing models, the more parsimonious model would be considered the better model overall. This statistic is equivalent to the adjusted  $R^2$  in multiple regression. An AGFI > 0.90 is considered an indication of a good fitting model (Savalei & Bentler, 2006). For Model 1, AGFI = 0.43, Model 2 AGFI = 0.83, and Model 3 AGFI = 0.93. These results further support the better fit of Model 3 than Model 1 or 2.

*Root-mean squared error of approximation (RMSEA).* The root-mean squared error of approximation (RMSEA) measures the average amount of misfit in the model compared to a perfect model. A RMSEA < .06 is considered an indication of a good fitting model (Tabachnick & Fidell, 2012). For Model 1, RMSEA = 0.37, Model 2 RMSEA = 0.15, and Model 3 RMSEA = 0.02. These results further support the better fit of Model 3 than Model 1 or 2 for the data.

*Akaike Information Criterion (AIC).* Like the AGFI, Akaike Information Criterion (AIC) assesses fit with a parsimony adjustment. Small AIC values indicate a good fitting model. There is no cut-off for this score, so the smallest value among competing models would be considered a good fitting model (Tabachnick & Fidell, 2012). For Model 1, AIC = 18.15, Model 2 AIC = 1.37, and Model 3 AIC = -0.98. These results further support the better fit of Model 3 than Model 1 or 2.

*Regression analysis for Model 4.* Standard multiple regression was used to assess Model 4, which assessed the ability of reinforcement learning, effort, and defeatist performance beliefs to predict functional capacity. Results of the regression indicate that these variables account for 37% of the variance,  $F(3,71) = 13.87, p < .001$ . Reinforcement learning and defeatist performance beliefs were significant in predicting functional outcome ( $p < .05$ ), with the reinforcement learning measure having the highest beta value (beta = 0.44), followed by defeatist performance beliefs (beta = -0.21). Effort was not a significant predictor of functional outcome, but approached significance (beta = 0.19,  $p = 0.054$ ).

*Conclusions regarding model fit.* The goodness-of-fit indices evaluated suggest that Model 3 is the best fitting path model for the variables considered. The standard regression analysis demonstrated that reinforcement learning and defeatist performance beliefs are significant predictors of functional capacity, while effort is not, although it was significantly associated with UPSA performance when correlations between the dependent variables were examined ( $r = .27, p < .05$ ; see Table 2).

Table 10

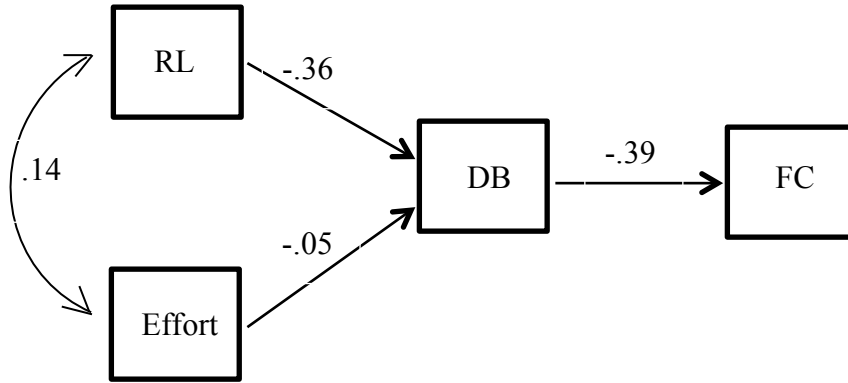
*Path Analysis Model Statistics*

Model	$\chi^2$	df	<i>p</i>	NFI	CFI	GFI	AGFI	RMSEA	AIC
1	22.15	2	>0.001	0.52	0.50	0.89	.43	0.37	18.15
2	5.37	2	0.07	0.88	0.92	0.97	.83	0.15	1.37
3	1.03	1	0.31	0.98	1.00	0.99	.93	0.02	-0.98

*Note.* df = degrees of freedom; NFI = Normed Fit Index; CFI = Comparative Fit Index; GFI = Goodness-of-fit Index; AGFI = Adjusted Goodness-of-fit Index; RMSEA = Root Mean Squared root-mean squared error of approximation; AIC = Akaike Information Criterion.

Figure 5

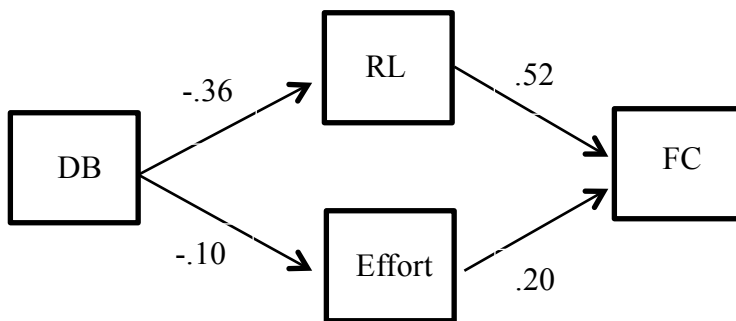
*Model 1. Model Reflecting Direct Contributions of Reinforcement Learning and Effort on Defeatist Performance Beliefs with Path Coefficients*



*Note.* RL = Reinforcement Learning. DB = Defeatist Performance Beliefs. FC = Functional Capacity.

Figure 6

*Model 2. Model Reflecting Direct Contributions of Defeatist Performance Beliefs on Reinforcement Learning and Effort with Path Coefficients*



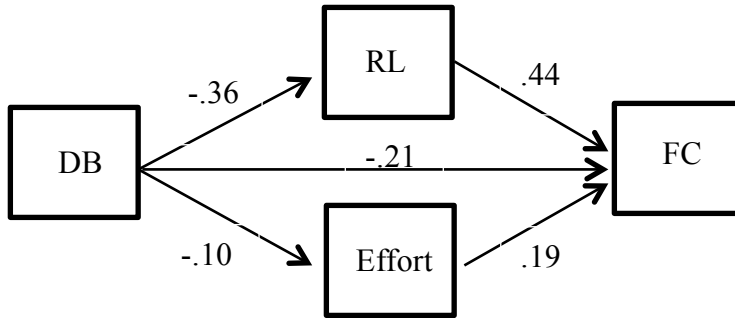
*Note.* RL = Reinforcement Learning. DB = Defeatist Performance Beliefs. FC = Functional Capacity.

Figure 7

*Model 3. Complete Model Reflecting Direct Contributions of Defeatist Performance Beliefs on Reinforcement Learning, Effort, and Functional Capacity with Path*

*Beliefs on Reinforcement Learning, Effort, and Functional Capacity with Path*

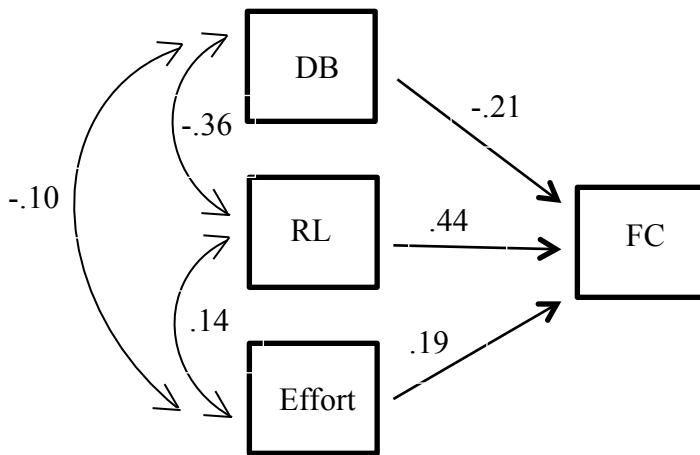
*Coefficients*



*Note.* RL = Reinforcement Learning. DB = Defeatist Performance Beliefs. FC = Functional Capacity.

Figure 8

*Model 4. Standard Regression Model with Beta Coefficients*



*Note.* RL = Reinforcement Learning. DB = Defeatist Performance Beliefs. FC = Functional Capacity.



## CHAPTER 5

### DISCUSSION

Some research has suggested that the global cognitive impairment seen in individuals with schizophrenia may be attributable to decreased effort. This decreased effort is not considered to be due to intentional poor performance, or malingering, but rather it may be due to in part by impairments in midbrain dopaminergic brain regions that are associated with effort and have been found to be affected in individuals with schizophrenia. In addition, cognitive theories of schizophrenia suggest that individuals experience defeatist performance beliefs about themselves and the environment. These defeatist performance beliefs are associated with a combination of factors, including cultural and internalized stigma and low self-esteem. Low self-efficacy, a component of self-esteem, has been found to be associated with unemployment, poor functional outcome and a lower quality of life (Corrigan et al., 2009; Vauth et al., 2007).

Individuals with mental illness that experience self-stigma and low self-efficacy may think that they are unworthy or unable to succeed in basic life functions, such as living independently. As a result, they may adopt an attitude that is consistent with the notion “why try” (Corrigan et al., 2009), thus expending less effort and attaining less. Defeatist performance beliefs in particular have been found to be a mediator between cognitive deficits and functional outcome (Grant & Beck, 2008). Effort and reinforcement learning are neurocognitive functions that have been increasingly studied in schizophrenia. They are associated with similar midbrain dopaminergic brain regions, regions that are impacted in individuals with schizophrenia. However, they have not been specifically evaluated in relation to defeatist performance beliefs, but may be a key factor in the

development and maintenance of these beliefs. The current study evaluated the predictive nature of effort, reinforcement learning, and defeatist performance beliefs on functional capacity using path analysis and standard regression. The primary model proposed that effort and reinforcement learning were correlated independent variables that predicted defeatist performance beliefs and defeatist performance beliefs then predicted functional capacity.

Results of the current study found defeatist performance beliefs and reinforcement learning to be the strongest indicators of functional capacity in all path models and in the standard regression analysis. Contrary to expectation, defeatist performance beliefs and effort were not largely associated with one another. Also, contrary to expectation, the more basic neurocognitive functions, effort and reinforcement learning, were not found to predict defeatist performance beliefs and then functional capacity. Rather, defeatist performance beliefs was found to predict reinforcement learning and effort. The best fitting model demonstrated defeatist performance beliefs to have a direct effect on reinforcement learning, effort, and functional capacity, with effort and reinforcement learning also having a direct effect on functional capacity.

As previously mentioned, Orth, Robins, and Widaman (2012) conducted an analysis of self-esteem on life outcomes across the lifespan. They found that self-esteem is not merely a by-product or result of success or failure, but that it is better conceptualized as a cause of life outcomes, such as psychological symptoms, relationships, and job satisfaction. Initially, this was interpreted as providing support solely for the predictive power defeatist performance beliefs would have on the behavioral outcome functional capacity. Instead, results of the current study suggest that

the causative influence of defeatist performance beliefs also applies to the neurocognitive abilities of reinforcement learning and effort. Furthermore, the “why try” theory (Corrigan et al., 2009) was initially interpreted as resulting in a circular effect between effort and defeatist performance beliefs, with low effort contributing to poor functioning and defeatist performance beliefs, with defeatist performance beliefs in turn contributing to poor functioning and low effort. In contrast, defeatist performance beliefs and effort were found to have a low association in the current study. The model that was found to provide the best fit for the data actually found defeatist performance beliefs to predict effort, but the model in which effort was predicting defeatist performance beliefs was not supported.

On the opposite side of the ‘why try’ effect, empowerment (the opposite of low self-efficacy) is an alternate reaction to stigma. Rather than accepting and acting on the stigmatization of mental illness, some individuals become empowered by them and react in a positive manner as if to disprove the stereotype. Empowerment has been found to be associated with positive self-esteem and a higher quality of life. Self-efficacy, whether on the negative end with defeatist performance beliefs or on the positive end with empowerment, has been theorized to result in treatment adherence and goal attainment or lack thereof (Corrigan et al., 2009). The current study found that defeatist performance beliefs have a predictive impact on reinforcement learning and functional capacity, supporting the notion that self-efficacy could lead to better outcome, possibly through treatment adherence and goal attainment. Further research would be necessary to address this issue specifically.

With regard to the relationship among defeatist performance beliefs, reinforcement learning, and functional capacity, as previously described reinforcement learning occurs when there is a discrepancy between what an individual predicts will occur and the actual outcome (Dayan & Daw, 2008; Hollerman & Schultz, 1998). Learning has been found to ensue if the outcome or reward is better than what was expected or predicted. Learning tapers off as the outcome and prediction equal one another and extinction will occur if the outcome or reward is worse than what was expected or predicted by the animal. Individuals with schizophrenia have been found to have difficulty learning from positive and negative feedback. While they have difficulty, eventual learning does occur. The relationship between defeatist performance beliefs and reinforcement learning found in the current study could suggest that defeatist performance beliefs are lowering the expectation for reward. A diminished expectation of reward would temper the learning from reinforcement. It has been found that individuals with schizophrenia, particularly those with high negative symptoms, do not show a preference for stimuli associated with a higher reward over those with no monetary gain. These results suggest that the dysfunction in the orbitofrontal cortex resulting in impaired valuations or reward is the primary mechanism for previous reward-learning impairments found in individuals with schizophrenia and high negative symptoms. Barch and Dowd (2010) examined motivation with regard to goal representations in individuals with schizophrenia. They proposed that individuals with schizophrenia exhibit an impairment in the evaluation of reward, related to reward prediction and mediated in the midbrain dopaminergic areas. The impaired evaluation of reward results in an impairment in future goal representation and lack of motivation. The results of the current study

support these findings and suggest that defeatist performance beliefs may be a factor contributing to impaired reward evaluation and future goal representation. Efforts to improve self-efficacy and lower defeatist performance beliefs could then improve ones ability to learn from choices and experiences that result in positive or negative outcomes. Doing so could result in more accurate appraisals of reward and outcome.

Negative self-appraisals and self-blame for illness has been found to be associated with greater risk of relapse, or exacerbations of psychotic symptoms, in individuals with schizophrenia (Gumley et al., 2006). Cognitive behavior therapy (CBT) has been found to improve negative and positive symptoms of schizophrenia by focusing on negative self-thought, which includes low self-esteem, defeatist performance beliefs, and fear of negative appraisals (Gould et al., 2001; Rector & Beck, 2001). Utilizing CBT and other intervention strategies aimed at defeatist performance beliefs and expectations for failure may also benefit reinforcement learning and functional capacity. The current research suggests that functional capacity could be improved by decreased defeatist performance beliefs both directly and through improved reinforcement learning and to a lesser extent effort.

Information is lacking on whether many performance-based measures of functional ability truly measure real world independence (Moore, 2007). There are many ways to evaluate functional ability, including self-report measures, observation, and performance-based measures. The current study evaluated the impact of effort, defeatist performance beliefs, and reinforcement learning on functional capacity (i.e. performance-based functioning). It is possible that evaluating the predictive value of these variables on functional outcome (i.e. self-reported real-world functional performance) may provide

differing results. Because this study was part of a larger battery of neuropsychological tests, the impact effort, defeatist performance beliefs, and reinforcement learning have on functional outcome as opposed to functional capacity could be evaluated. A standard multiple regression demonstrated that reinforcement learning, effort, and defeatist performance beliefs account for 48% of the variance of functional outcome, with defeatist performance beliefs being the strongest predictor (beta = -0.54), followed by effort (beta = 0.27), and reinforcement learning, which was not a significant predictor (beta = 0.14,  $p = 0.14$ ). Interestingly, effort becomes a significant predictor of functioning and reinforcement learning becomes non-significant when the functioning is self-reported. These results support the idea that different measures of functional ability are assessing different aspects of functioning. One possible remedy to this issue is to include multiple functional measures when evaluating functional ability, including functioning related to perceived ability as well as actual functional attainment (e.g., employment history), and ability (e.g., in person demonstration of one's ability to complete tasks of daily living).

Studies indicate that persons with mental illness who have more conspicuous illness symptoms and poorer social skills engender more negative responses from others (Dickerson, 2002). Another question that could be addressed is whether psychiatric symptoms themselves, and which ones specifically, are related and perhaps predictive of defeatist performance beliefs. This would provide additional avenues of potential treatment options to decrease defeatist performance beliefs and thus increase functional ability.

Two of the primary variables in this study are related to midbrain dopaminergic areas, areas that are also associated with schizophrenia itself and the medications used to

treat schizophrenia. Not surprisingly, effort and reinforcement learning were correlated with chlorpromazine equivalent dosage. The majority of participants with schizophrenia in the current study were taking some form of antipsychotic medication, making any causal effect of such medications on performance implausible. Future research could examine these variables and their relationship with defeatist performance beliefs and functional outcome in first-episode or drug naïve participants, or through experimental designs allowing antipsychotic medication manipulation to further clarify the impact antipsychotic medication is having on these areas.

The small number of variables examined through path analysis in the current study restricted the number of possible paths that could be evaluated. Each model is tested based on the models degrees of freedom, which is the number of distinct elements in the covariance matrix minus the number of model parameters. Degrees of freedom must be  $> 0$  in order to be evaluated, if degrees of freedom = 0 then the model is considered saturated and is unable to be analyzed. With only 4 variables composing the covariance matrix, this limited the number of paths that could be drawn in specific variable organizations. Future research could examine effort, reinforcement learning, defeatist performance beliefs, and functioning using a variety of observed variables each, rather than one measurement per variable. This would allow the examination of more path models, and further determine the components that are most salient at predicting functioning in each variable.

Because the path and regression analyses included both individuals with schizophrenia and controls, the results are not unique to schizophrenia per say. We might conclude that the path identified has explanatory power as to why individuals with

schizophrenia have poor functional outcomes. The associations between the variables found in the model help us understand how these variables, specifically reinforcement learning, defeatist performance beliefs, and effort, predict functional outcome in schizophrenia. Also, because both control and schizophrenia groups were included in these models, the findings suggest that reinforcement learning, defeatist performance beliefs, and effort are predictive of functional outcome regardless of whether severe mental illness is present. For example, the factors associated with increased blood pressure are the same, regardless of whether an individual has reached the cut-off for hypertension. Furthermore, in the cognitive model of schizophrenia previously discussed and proposed by Beck (2004), he discusses the symptoms of schizophrenia as being more extreme versions of thoughts and experiences that many people without the disorder will experience. Future research could examine these same variables with a larger sample of individuals with schizophrenia with a more diverse range of attained functional level in order to determine if the model found in this study generalizes.

## **Conclusion**

Results of the current study were surprising in that defeatist performance beliefs were found to be predictive of reinforcement learning, functional capacity, and to a lesser extent effort. It was hypothesized that the more basic neurocognitive functions of effort and reinforcement learning would predict defeatist performance beliefs, which would then predict functional capacity; however this was not the case. These results suggest that defeatist performance beliefs are negatively affecting the expected outcomes of actions, resulting in decreased learning from reinforcement. Defeatist performance beliefs were found to impact functional capacity both directly and indirectly through their impact on



reinforcement learning and effort. While the results were not what was expected, they provide important information as to the impact of self-esteem and self-efficacy and the influence these beliefs have on cognitive and behavioral outcomes. Behavioral strategies aimed at improving defeatist performance beliefs could in turn improve reinforcement learning, effort, and functional capacity. Additionally, there is an atmosphere within the health care community and the culture as a whole that depicts individuals with severe mental illness as flawed and less capable of goal attainment and independent functioning. This study further supports the potential causative influence such beliefs could be having on actual performance and functioning. There seems to be a self-fulfilling prophecy in that individuals with severe mental illness are told they will not lead a successful or independent life, and these beliefs when adopted lead to reduced neurocognitive and behavioral functioning. A continued change in the response health care providers and the community give to individuals with schizophrenia, and likely severe mental illness as a whole, aimed at increased hope for stability and independent functioning could also aid in improved self-esteem and expectancies.

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- Wright, E. R., Gronfein, W. P., & Owens, T. J. (2000). Deinstitutionalization, social rejection, and the self-esteem of former mental patients. *Journal of Health and Social Behavior, 41*(1), 68–90.
- Zimmerman, M., Coryell, W., Corenthal, C., & Wilson, S. (1986). Dysfunctional attitudes and attribution style in healthy controls and patients with schizophrenia, psychotic depression, and nonpsychotic depression. *Journal of Abnormal Psychology, 95*(4), 403–405. doi:10.1037//0021-843X.95.4.403

## VITA

SALLY J. VOGEL (Formerly Sally J. Barney)

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### EDUCATION

**Doctor of Philosophy**

University of Nevada, Las Vegas, Las Vegas, NV  
APA-Accredited Clinical Psychology Program  
Neuropsychology Track

Anticipated Graduation May 2015  
Advisor: Daniel N. Allen, Ph.D.

**Dissertation:** Functional Capacity in Schizophrenia: Relationship Among Reinforcement Learning, Effort, and Defeatist Performance Beliefs  
Defended May 14, 2014

**Clinical Internship**

University of Oklahoma Health Sciences Center, Oklahoma City, OK  
APA-Accredited Clinical Internship  
Neuropsychology Track

July 2014-July 2015

**Master of Arts in Clinical Psychology**

University of Nevada, Las Vegas, Las Vegas, NV

Advisor: Daniel N. Allen, Ph.D.

**Thesis:** Using Negative Feedback to Guide Behavior: Impairments on the First 4 Cards of the Wisconsin Card Sorting Test Relates to Psychosis

May 2012

**Bachelor of Arts**

University of Nevada, Las Vegas, Las Vegas, NV  
Summa Cum Laude with Honors

**Major:** Psychology **Minor:** Biology

**Honors Thesis:** Neurocognitive Deficits in Bipolar Disorder with Co-occurring Borderline Symptomatology

Advisor: Daniel N. Allen, Ph.D.

May 2009

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### CLINICAL TRAINING

**VA Southern Nevada Healthcare System (VASNHS)**

Las Vegas, NV

August 2013–June 2014

Supervisor: Robert F. Mirabella, Ph.D.

- **Doctoral Practicum Student:** Cognitive Processing Therapy with veterans experiencing Post Traumatic Stress Disorder, traumas include combat trauma and military sexual trauma. A weekly support group for female veterans co-facilitated with Diane Sakal-Gutierrez, LCSW.
- **Supervision and Didactics:** Weekly individual supervision meetings and case conferences are held. Training in and supervision of diagnostic interviews, treatment planning, and session notes within the CPRS medical notes system.

**Lou Ruvo, Center for Brain Health, Cleveland Clinic**

Las Vegas, NV

June 2012–June 2013

Supervisor: Sarah Banks, Ph.D., ABPP/CN

- **Doctoral Practicum Student:** Neuropsychological assessments using a flexible battery with individuals ranging from 25 to 97 in an outpatient medical setting. Further responsibilities include scoring, interpretation and partial interviewing and report writing. Commonly presented patient diagnoses include neurodegenerative disorders, specifically dementia and

movement disorders. A weekly support group for caregivers of individuals with memory problems co-facilitated with Donna Munic-Miller, PhD.

- **Supervision and Didactics:** Weekly individual supervision meetings were held in addition to weekly case conferences with only neuropsychology supervisors and students, as well as case conferences or grand rounds with neurology, physical therapy and/or social work.
- After the formal practicum training, I was hired to continue conducting neuropsychological assessments, scoring, and partial report writing on an as needed basis.

**Center for Applied Neuroscience**

June 2011–June 2012

Las Vegas, NV

Supervisor: Thomas F. Kinsora, Ph.D.

- **Doctoral Practicum Student:** Neuropsychological clinical and forensic assessments using a flexible battery with individuals ranging from 7 to 79 in an outpatient private practice setting or the Clark County Detention Center. Further responsibilities include scoring, interpretation and partial interviewing and report writing. Commonly presented patient diagnoses include cognitive disorders of varying etiologies, affective disorders, learning disabilities, and TBI.
- **Supervision and Didactics:** Weekly individual and group supervision meetings were held. Also attended weekly practicum seminars on campus, which include didactic, group supervision and case conference components.
- After the formal practicum training, I was hired to continue conducting neuropsychological assessments, scoring, and partial report writing for approx. one client per month.

**Center for Individual, Couple, and Family Counseling (CICFC)** August 2010–August 2011

University of Nevada, Las Vegas

Supervisor: Christopher A. Kearney, Ph.D. August-May

Nellie Mihelic, M.A. May-August

- **Doctoral Practicum Student:** Provided long-term individual therapy to a caseload of approximately 7-10 clients per week in an outpatient University affiliated mental health clinic. Diagnoses included personality disorders, affective disorders, anxiety disorders and adjustment disorders. Primary theoretical approach used from August to May was CBT under the supervision of Christopher Kearney, PhD. The final three months of therapy was primarily Interpersonal Process under the supervision of Nellie Mihelic, an advanced doctoral student, supervisor in training.
- **Supervision and Didactics:** Supervision consisted of weekly individual and group meetings with video tape review as well as weekly practicum seminars, which included didactic, group supervision, and case conference components.

**Psychological Assessment & Testing Clinic**

August 2010–September 2011

University of Nevada, Las Vegas

Supervisor: Michelle G. Carro, Ph.D.

- **Doctoral Practicum Student:** Neuropsychological and psychoeducational assessments using a flexible battery approach with adults and children referred from the community and the University Disability Resource Center in an outpatient University affiliated mental health clinic. Further responsibilities included interviewing, scoring, interpretation, report writing, and provision of feedback to clients.
- **Supervision:** Supervision included reviewing cases, joint determination of assessment battery and interpretation of results, report revisions, and discussion of feedback.

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**PUBLICATIONS AND PRESENTATIONS**

**Refereed Articles Published or In Press**

**Vogel, S.J.,** Strauss, G.S., & Allen, D.N., (In Press). Using negative feedback to guide behavior: Impairments on the first 4 cards of the Wisconsin Card Sorting Test predicts negative

symptoms of schizophrenia. *Schizophrenia Research*.

- Barney, S.J.**, Allen, D.N., Thaler, N.S., Park, B.S., Strauss, G.P., & Mayfield, J. (2011). Neuropsychological and behavioral measures of attention assess discrete constructs in children with traumatic brain injury. *The Clinical Neuropsychologist*, 25 (7), 1145-1157.
- Allen, D.N., Thaler, N.S., Ringdahl, E.N., **Barney, S.J.**, & Mayfield, J. (2011). Comprehensive trail making test performance in children and adolescents with traumatic brain injury. *Psychological Assessment*, doi:10.1037/a0026263
- Thaler, N.S., **Barney, S.J.**, Reynolds, C.R., Mayfield, J., Allen, D.N. (2011). Differential sensitivity of TOMAL subtests and index scores to pediatric traumatic brain injury. *Applied Neuropsychology*, 18(3), 168-178.
- Park, B.S., Allen, D.N., **Barney, S.J.**, Ringdahl, E.N., & Mayfield, J. (2009). Structure of attention in children with traumatic brain injury. *Applied Neuropsychology*, 16(1), 1-10.

### **Manuscripts/Book Chapters Submitted**

- Strauss, G.P., Thaler, N., Matveeva, T., **Vogel, S.**, Sutton, G., Lee, B., Allen, D.N. (submitted). Predicting psychosis across diagnostic boundaries: Behavioral and computational modeling evidence for impaired reinforcement learning in schizophrenia and bipolar disorder with a history of psychosis. *Biological Psychiatry*.
- Barney, S.J.** & Allen, D.N., (submitted). Connor's Continuous Performance Test. In Cecil Reynolds, R Kamphaus, & C DiStefano (Eds), *Encyclopedia of Psychological Testing*. Oxford University Press: New York.
- Vogel, S.J.** & Allen, D.N. (submitted). Assessing substance related disorders in Asian Americans. In LT Benuto, N Thaler & BD Leany (Eds.), *Guide to Psychological Assessment with Asian Americans*. New York, NY: Springer Publishing.

### **Presentations and Published Abstracts**

\* Denotes presentation has a corresponding published abstract.

- Vogel, S.J.**, Banks, S.J., Miller, J.B. (2014). Factor analysis and construct validity of the Montreal Cognitive Assessment: concordance with standard neuropsychological measures. The Annual Alzheimer's Association International Conference, July 12-17, Copenhagen, Denmark.
- \*Lee, B.G., **Vogel, S.J.**, Sisk, S.J., Yao, J.K., van Kammen, D.P., & Allen, D.N. (2014). The effects of dopamine antagonism on reward learning in schizophrenia. The 34th Annual Conference National Academy of Neuropsychology, November 12 - 15, Fajardo, Puerto Rico.
- \*Palisoc B.M., **Vogel S.J.**, Ringdahl E.N., Zenisek R., Allen D.N. (2014). The relationship between negative symptoms and functional outcome in schizophrenia and bipolar disorder. The 34th Annual Conference National Academy of Neuropsychology, November 12 - 15, Fajardo, Puerto Rico.
- \*Ciobanu C, **Vogel S.J.**, Cooper L., Hughes C., Allen D.N. (2014). Performance on a novel attention measure, the Search Identification Task, in Schizophrenia. The 34th Annual Conference National Academy of Neuropsychology, November 12 - 15, Fajardo, Puerto Rico.
- Dadis, F., **Vogel, S.J.**, Lee, B.G., Mayfield, J., & Allen, D.N. (2014). The Delis-Kaplan Trail Making Test (D-KEFS) is sensitive to injury and recovery in children with brain injury.

- The 94<sup>th</sup> Annual Western Psychological Association Conference, May, Portland, Oregon
- Cooper, L.J., Call, E.T., **Vogel, S.J.**, & Allen, D.N. (2014). Examining the validity of the Halstead Category Test-Adult Computer Version. The 94<sup>th</sup> Annual Western Psychological Association Conference, May, Portland, Oregon
- Ciobanu, C, **Vogel, S.J.**, Sisk, S. & Allen, D.N. (2014). Test-retest Reliability and Practice Effects of the Original and Computer Versions of the Halstead Category Test. The 94<sup>th</sup> Annual Western Psychological Association Conference, May, Portland, Oregon
- \***Vogel, S.J.**, Ringdahl, E.N., Freeman, A., Dadis, F., & Allen, D.N. (2013). Effort expenditure is related to every day functioning and psychiatric symptomatology. The 33rd Annual Conference National Academy of Neuropsychology, October 16-19, San Diego, CA.
- \*Cooper, L., Allen, D.N., **Vogel, S.J.**, Ciabanu, C., & Woolerey, H., (2013). Examining the factors of the Halstead Category Test. The 33rd Annual Conference National Academy of Neuropsychology, October 16-19, San Diego, CA.
- \*Ringdahl, E.N., **Vogel, S.J.**, Freeman, A.I., Call, E. T., Allen, D.N. (2013). Impaired Higher-order Social Perception Skills in Psychotic and Affective Disorders. The 33rd Annual Conference National Academy of Neuropsychology, October 16-19, San Diego, CA. (Poster award)
- \*Sisk SJ, Ringdahl EN, **Vogel SJ**, Freeman AI, Allen DN (2013) Social perception skills predict functional outcome in bipolar disorder and schizophrenia. The 33rd Annual Conference National Academy of Neuropsychology, October 16-19, San Diego, CA.
- \*Verbiest, R., Ringdahl, E.N., Thaler, N.S., Sutton, G.P, **Vogel, S.J.**, Reyes, A., & Allen, D.N. (2013). Basic auditory perception deficits are related to impaired perception of sarcasm. The 33rd Annual Conference National Academy of Neuropsychology, October 16-19, San Diego, CA.
- \*Zink, D, **Barney, S**, Gilbert, G, & Allen, D. (2013). Factor Structure of the Search Identification Task (SIT): A Novel Measure of Attention and Working Memory. The 33rd Annual Conference National Academy of Neuropsychology, October 16-19, San Diego, CA.
- Vogel, S.J.**, Ringdahl, E.N. & Allen, D.N. (2013). Presentation at the Graduate and Professional Student Research Fair, Las Vegas, NV.
- \***Barney, S.**, Thaler, N., Sutton, G., Strauss, G., & Allen D. (2012). Reward Learning Performance Predicts Functional Ability. The 32nd Annual Conference National Academy of Neuropsychology, October, Nashville, TN.
- Lee, B.G., **Barney, S.J.**, Catalano, L.T., Ringdahl, E.N., Vertinski, M., Adams, J.L., Shugarman, Y.Y., Snyder, J.S., Allen, D.N., Strauss, G.P. (2012). Anhedonia is Associated with Impaired Long-Term Memory for Positive Emotional Stimuli in Individuals with Schizophrenia. The Society for Research in Psychopathology Annual Conference, October, Ann Arbor, MI.
- Vertinski, M., Allen, D., Thaler, N., Heisler, D., Park B., **Barney, S.** (2012). Construct Validity of the Search Identification Task. Poster presented at the Graduate Student Research Fair, Las Vegas, NV. (Poster Award).
- Ramage, E.M., **Barney, S.J.**, Flores, A., Klimas, N., Vertinski, M., Ringdahl, E.N., Sutton, G.P., Allen, D.N., & Snyder, J.S. (2012). Concurrent Sound Segregation Impairments in schizophrenia. The Society of Biological Psychiatry Annual Conference, Philadelphia, PA.



- \***Barney, S.J.**, Umuhoza, D., Strauss, G.P., Knatz-Bello, D., Allen, D.N. (2011). Negative Reinforcement Learning: The first 4 cards of the WCST relates to psychosis. The 31st Annual Conference National Academy of Neuropsychology, November, Marco Island, FL.
- \*Vertinski, M., Allen, D., Thaler, N., Heisler, D., Park, B., **Barney, S.** (2011). Construct Validity of the Search Identification Task (SIT). The 31st Annual Conference National Academy of Neuropsychology, November, Marco Island, FL.
- Heisler, D.L., Umuhoza, D., Ringdahl, E.N., **Barney, S.J.**, Thaler, N.S., & Allen, D.N. (2011). Psychometric properties of a new measure of attention and working memory. The 91<sup>st</sup> Annual Convention of the Western Psychological Association, April, Los Angeles, CA.
- Hart, J.S., Locasci, T., Hadland, C., Umuhoza, D., Turner, A., **Barney, S.J.**, Thaler, N.S., Mayfield, J., & Allen, D.N. (2011). Neuropsychological and behavioral measures in children with traumatic brain injury. The 91<sup>st</sup> Annual Convention of the Western Psychological Association, April, Los Angeles, CA.
- \*Boucher, J.R., **Barney, S.J.**, Mayfield, J., Allen, D.N. (2011). Trail making tests: Comparing versions in children with TBI. The 3rd Annual American College of Professional Neuropsychology Conference, March, Las Vegas, NV.
- \*Terranova, J. **Barney, S.J.**, Mayfield, J., Allen, D.N. (2011). Construct validity of the Comprehensive Trail Making Test in children with traumatic brain injury. The 3rd Annual American College of Professional Neuropsychology Conference, March, Las Vegas, NV.
- \***Barney, S.J.**, Allen, D.N., Stolberg, P.C., Mayfield, J. (2010). Construct Validity of Reynolds Intellectual Assessment Scales (RIAS) in Brain Injured Children. Poster and Platform Session presented at the 30th Annual Conference National Academy of Neuropsychology, October, Vancouver, Canada.
- \*Sutton, G.P., Ringdahl, E.N., Thaler, N.S., **Barney, S.J.**, Mayfield, J., Pinegar, J., Allen, D.N. (2010, October). Differences in Executive Function Profiles in Normal Children and those with Traumatic Brain Injury. The 30th Annual Conference National Academy of Neuropsychology, October, Vancouver, Canada.
- \*Weintraub, D.M., Sutton, G.P., Ramage, E., Pasinski, A., Ringdahl, E.N., **Barney, S.**, Allen, D.N., and Snyder, J.S. (2010). Auditory stream segregation in schizophrenia. The Society of Biological Psychiatry 65<sup>th</sup> Annual Meeting, New Orleans, LA. *Biological Psychiatry*, 67(9), 156.
- Barney, S.J.**, Strauss, G.P., Garcia, A.E., Sanders, L.J., Allen, D.N. (2010, August). The First Ten Cards of the WCST: Comparing Bipolar and Schizophrenia. The 118<sup>th</sup> annual meeting of the American Psychological Association, August, San Diego, CA.
- \***Barney S.J.**, Pinegar J.L., Thaler N.S., McMurray J.C., Mayfield J., Allen D.N. (2010, February). Differential Impairment of Attention Component Processes in Children with TBI and ADHD. The 2<sup>nd</sup> Annual American College of Professional Neuropsychology Conference, February, Las Vegas, NV.
- \***Barney, S.J.**, Thaler, N.S., Allen, D.N., Donohue, B., Mayfield, J. (2009). WISC-IV profiles in children with traumatic brain injury: Similarities and differences to the WISC-III. The 29th Annual Conference National Academy of Neuropsychology, November. New Orleans, LA.
- \*Strauss, G.P., Ringdahl, E., **Barney, S.J.**, Jetha, S.S., Duke, L.A., Ross, S.A., Watrous, B.,

- Allen, D.N. (2009). Using Negative Feedback to Guide Behavior: Impairment on the First Four Cards of the Wisconsin Card Sorting Test Predicts Negative Symptoms of Schizophrenia. The 29th Annual Conference National Academy of Neuropsychology, November. New Orleans, LA.
- \*Kazakov, D., Haderlie, M., **Barney, S.J.**, Allen, D.N., Mayfield, J. (2009). Construct validity of the CTMT in adolescent traumatic brain injury. The 117<sup>th</sup> annual meeting of the American Psychological Association, August, Toronto, Canada.
- \***Barney, S.J.**, Ringdahl, E.N., Kazakov, D., Allen, D.N. (2008). Neurocognitive deficits in bipolar disorder with co-occurring borderline symptomatology. The 28th Annual Conference National Academy of Neuropsychology, October, New York, NY.
- \*Bello, D.T., Randall, C., Armstrong, C.M., **Barney, S.J.**, Kazakov, D., Allen, D.N. (2008). Neurocognitive deficits predict functional outcome as measured by the UCSD Performance-based Skills Assessment (UPSA) in individuals with bipolar disorder. The 28th Annual Conference National Academy of Neuropsychology, October, New York, NY.
- Barney, S.J.**, Randall, C., Bello, D.T., Armstrong, C.M., Allen, D.N. (2008). Neurocognitive deficits in bipolar disorder with co-occurring borderline symptomatology. Poster presented at the Nevada Undergraduate Research Symposium, April, Reno, NV. Also presented at the Annual Honors Poster Session, May, Las Vegas, NV
- \*Strauss, G.P., **Barney, S.J.**, Timko, C.J., Jetha, S.S., Kamalani, L., Pace, S.A., Barnes, L.M., Hoyt, T.E., Duke, L.A., Ross, S.A., Schwartz, J., Allen, D.N. (2007). Impaired olfactory hedonic judgment in deficit syndrome schizophrenia. The 27th Annual Conference National Academy of Neuropsychology, October, Scottsdale, AZ.
- \*Strauss, G.P., **Barney, S.J.**, Timko, C.J., Jetha, S.S., Kamalani, L., Pace, S.A., Barnes, L.M., Hoyt, T.E., Duke, L.A., Ross, S.A., Schwartz, J., Allen, D.N. (2007). Neuropsychological impairment patients with deficit syndrome schizophrenia. The 27th Annual Conference National Academy of Neuropsychology, October, Scottsdale, AZ.
- Barney, S.J.**, Hoyt, T.E., Cristiani, N., Timko, C.J., Park, B.S., & Allen, D.N. (2007). Factor support of the distinctiveness of a new measure for attention and working memory. The Annual Nevada State Psychological Association Conference, May, Las Vegas, NV.
- Barney, S.J.**, Jetha, S.S., Mayfield, J., & Allen, D.N. (2007). Learning and memory performance in children with ADHD subtypes. The 87<sup>th</sup> Annual Convention of the Western Psychological Association, May, Vancouver, Canada.
- Strauss, G.P., Villamar, A.J., Hoyt, T.E., **Barney, S.J.**, Crow, M., Kamalani, L., & Allen, D.N. (2006). Positive emotion buffers against the development of psychopathology. The 5<sup>th</sup> Annual International Positive Psychology Summit, October, Washington, D.C.
- Strauss, G.P., Leany, B.D., **Barney, S.J.**, Kamalani, L., & Allen, D.N. (2006). Positive emotions broaden cognitive functioning. The 86<sup>th</sup> Annual Convention of the Western Psychological Association, April, Palm Springs, CA. Also presented at the Annual Psi Chi UNLV Chapter Annual Conference, April, Las Vegas, NV (Poster award)

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## RESEARCH EXPERIENCE

### Graduate Research

**Neuropsychology Research Program**  
University of Nevada, Las Vegas

Summer 2009–Present  
Advisor: Daniel N. Allen, Ph.D.

Study: Dissertation

Responsibilities include project development, which involved selection of test battery, proposal preparation, IRB approval preparation, database creation, and organization of such materials as assessment materials, administration instructions and scoring, and subject recruitment resources, as well as phone screening potential participants, assessment of individuals with schizophrenia and bipolar disorder as well as controls using a 6-hour-long neuropsychological and neuroscience battery. IRB authorization was approved to test clinical participants off campus at Mojave Mental Health, an outpatient mental health clinic affiliated with the Nevada School of Medicine.

Study: Wechsler Intelligence Scale for Children, Fifth edition, standardization study

Responsibilities include being trained and approved in the administration of the WISC-V standardization version. To date, one child has been tested.

Study: Validation of the computer Halstead Category Test and Search Identification Task

Responsibilities include training undergraduate research assistants on administration, scoring, and data entry procedures. The project involves a two-part assessment, each lasting 2-hours and occurring within 7-14 days of one another.

Study: Longitudinal study of neuropsychological and functional deficits in bipolar disorder

Responsibilities included phone screening of potential participants, scheduling eligible participants for assessments, test scoring, data entry, and training research assistants in test scoring and entry procedures.

**Auditory Cognitive Neuroscience Laboratory**

Fall 2009–Fall 2011

University of Nevada, Las Vegas

Advisors: Joel S. Snyder, Ph.D., Daniel N. Allen, Ph.D.

Study: Neural mechanisms of perceptual processing in schizophrenia and bipolar disorder

Responsibilities included assessment of individuals with schizophrenia using a 4-hour long neuropsychological and neuroscience battery. In addition, responsibilities include phone screening of potential participants, scheduling eligible participants, scoring, data entry, and training research assistants in scoring and entry protocols of participant assessments.

**Achievement Center**

Fall 2009–Fall 2011

University of Nevada, Las Vegas

Advisor: Bradley Donohue, Ph.D.

Study: Concurrent drug abuse treatment and HIV prevention in child neglecting mothers, NIDA funded RO1 grant (DA020548-01A1)

Responsibilities included evaluating substance-abusing mothers who had been identified by Child Protective Services to participate in a therapeutic program as well as organizing efforts to regain contact with out of contact participants. Assessments were administered in the clients' homes and included the SCID, urine analysis, home safety ratings, and verbally administered self-report measures of child abuse potential, family interaction styles, and life satisfaction.

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**LEADERSHIP AND SERVICE**

**National Academy of Neuropsychology**

Professional Affairs and Information Committee Student Member

April 2011–Present

Responsibilities: Advocacy for neuropsychologists, providing practice-related information to neuropsychologists in the form of resources and information, dictate quarterly conference call meeting minutes, monitor national neuropsychology listserv for practice related issues.

Student Volunteer at Annual Conferences	
Nashville, TN	October 2012
Marco Island, FL	November 2011
Vancouver, BC, Canada	October 2010
New Orleans, LA	November 2009

**Outreach Undergraduate Mentoring Program** Fall 2011–Present

Undergraduate student mentor  
 Responsibilities: Mentor an underrepresented undergraduate student through graduate school preparation, applications, and potential career paths in psychology.

**American Red Cross, Southern Nevada Chapter** April 2013–September 2013  
 Disaster Assistance Team Member

Responsibilities: Completed a one-day training in ‘response to disaster’. Was on-call one weekend a month to respond to disasters, typically house and apartment fires, in southern Nevada in order to supply aid to the victims in the form of food, clothes, and shelter when necessary.

**UNLV Clinical Psychology Doctoral Student Committee** Fall 2010–August 2011  
 Cohort Representative and Treasurer

**American College of Professional Neuropsychology** February 2010  
 Student Volunteer at 2<sup>nd</sup> Annual Conference

**Reitan Society Meeting** February 2010  
 Student Volunteer at Conference

**National Alliance of Professional Psychology Providers** Fall 2009  
 Student Volunteer at Continuing Education Conference

**Psi Chi National Honor Society in Psychology** Fall 2008–Spring 2009  
 Vice-President, University of Nevada, Las Vegas Chapter

**Psychology Club** Spring 2008  
 Secretary, University of Nevada, Las Vegas

**PROFESSIONAL AFFILIATIONS AND HONOR SOCIETIES**

National Academy of Neuropsychology	2007–Present
International Neuropsychological Society	2014–Present
American Psychological Association	2007–Present
Division 40	2014–Present
American Psychological Association of Graduate Students	2009–Present
Nevada Psychological Association	2010–Present
Phi Kappa Phi National Honor Society	2008 –Present
Golden Key Honor Society	2008 –Present
Psi Chi, National Honor Society in Psychology	2006 –Present

**OTHER RELEVANT WORK AND TRAINING EXPERIENCE**

<b>Psychological Assessment &amp; Testing Clinic</b>	August 2009–August 2010
Las Vegas, NV	Supervisor: Michelle G. Carro, Ph.D.

Graduate Assistant responsible for conducting telephone intakes, scheduling and case assignments for 6-10 graduates students, bookkeeping, and other administrative functions at the department-sponsored community psychological assessment training clinic. (20 hours per week).

**Symptoms Ratings Training Program**

Fall 2010

University of Nevada, Las Vegas

Training Supervisor: Daniel N. Allen, Ph.D.

Completed a training program for the administration of a number of clinician administered symptom scales associated with symptoms of schizophrenia and bipolar disorder. Training was comprised of a series of workshops. Refresher workshops were held periodically.

**SCID Training Program**

Summer 2009

University of Nevada, Las Vegas

Training Supervisor: Daniel N. Allen, Ph.D.

Completed a training program over three months and made up of approximately 40 hours for administration of the Structured Clinical Interview of the DSM-IV-TR Axis I Disorders (SCID).

Provided training and mock interview assistance in two subsequent trainings held by Daniel Allen, PhD

**The Collaborative IRB Training Initiative (CITI) Program**

Spring 2005–Present

(<http://www.citiprogram.org>).

**HONORS AND AWARDS**

Graduate & Professional Student Association travel funding to attend The National Academy of Neuropsychology Convention in Fajardo, Puerto Rico (\$600)	2014
Barrick Graduate Fellowship (\$15,000)	
Summer Session Scholarship (\$2,000)	2013
Graduate & Professional Student Association research funding (\$470)	2013
President’s Graduate Research Fellowship (\$25,000)	2012
Graduate & Professional Student Association travel funding to attend and present at The National Academy of Neuropsychology Convention in Marco Island, FL (\$500)	2011
Patricia Sastaunak Scholarship (\$2,500)	2011
Graduate Access Grant (\$2,000)	2011
Edward Lovinger Psychology Scholarship (\$2,000)	2010
Psi Chi Travel Grant to attend The American Psychological Association 116 <sup>th</sup> Annual Convention in Boston, MA (\$1,800)	2008
Second Place Poster Award, Psi Chi National Honor Society Annual Conference, Las Vegas, Nevada (\$75)	2006
John P. & Mary V. Hughes Valedictorian Scholarship (\$10,000)	2004
Provost Scholarship, Nevada Board of Regents (\$12,000)	2004
Governor Guinn Millennium Scholarship (\$10,000)	2004

**GRANTS**

<b>National Science Foundation-EPSCoR Undergraduate Research Award</b>	2007-2008
University of Nevada, Las Vegas Research Funding (\$4,310)	

## PROFESSIONAL REFERENCES

### **Daniel N. Allen, Ph.D.**

Lincy Professor of Psychology Department of Psychology  
University of Nevada, Las Vegas  
4505 Maryland Parkway MS 5030  
Las Vegas, NV 89154-5030

- Email: [daniel.allen@unlv.edu](mailto:daniel.allen@unlv.edu)
- Phone: (702) 895-0121

### **Sarah Banks, Ph.D., ABPP/CN**

Head, Neuropsychology Program Lou Ruvo Center for Brain Health  
Assistant Professor of Medicine Department of Psychiatry and Psychology  
Cleveland Clinic Lerner College of Medicine  
Case Western Reserve University  
888 W Bonnevillle Ave  
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### **Thomas F. Kinsora, Ph.D.**

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