DISCOUNTING, ATTRIBUTIONAL STYLE, AND DEPRESSIVE SYMPTOMATOLOGY IN COLLEGE STUDENTS WITH AND WITHOUT ASTHMA

By
LEAFAR F-J ESPINOZA

Bachelor of Arts
Rand Afrikaans University
Johannesburg, South Africa
1999

Master of Science
Oklahoma State University
Stillwater, Oklahoma
2005

Submitted to the Faculty of the Graduate College of the Oklahoma State University In partial fulfillment of The requirements for The Degree of DOCTOR OF PHILOSOPHY July, 2007
DISCOUNTING, ATTRIBUTIONAL STYLE, AND DEPRESSIVE SYMPTOMATOLOGY IN COLLEGE STUDENTS WITH AND WITHOUT ASTHMA

Thesis Approved:

________________________  ________________________
Frank Collins, Ph.D.       Thesis Adviser

________________________
Thad Leffingwell, Ph.D.

________________________
Melanie Page, Ph.D.

________________________
Terry Stinnett, Ph.D.

________________________  ________________________
A. Gordon Emslie, Ph.D.    Dean of the Graduate College
ACKNOWLEDGEMENTS

Thank you Dr. Frank Collins for your support and for introducing me to the interesting world of behavior economics. Also, thank you to my committee members, Drs. Melanie Page, Thad Leffingwell, and Terry Stinnett, for your comments, suggestions, and an enjoyable dissertation defense experience.

I would like to express my deep gratitude to a colleague and good friend, Theodore Wagener. Your insightful reviews and willingness to help at any time of the day (or night) were greatly appreciated.

To my loving parents, Rafael and Ana Maria Espinoza and my sister, Raffaella. Thank you for your faith in me.

Most importantly my thanks, respect, and love for the one person who has been with me throughout this whole process. Thank you for your constant love and support Natalie.
TABLE OF CONTENTS

Chapter                                                                 Page

I.  INTRODUCTION ................................................................................................... 1

II. LITERATURE REVIEW ........................................................................................ 9

   Delay and Probability Discounting ................................................................. 9
   Depression and Attributional Style ............................................................... 19
   Psychological Factors Associated with Chronic Illness ................................ 26
   Summary ........................................................................................................ 34

III. PRESENT STUDY ............................................................................................... 36

IV. METHOD ............................................................................................................ 40

   Participants .................................................................................................. 40
   Procedure .................................................................................................... 41
   Measures ..................................................................................................... 41

V. RESULTS .............................................................................................................. 46

   Preliminary Analyses .................................................................................. 46
   Primary Analyses ....................................................................................... 46
   Exploratory Analyses ............................................................................... 48

VI. DISCUSSION ..................................................................................................... 51

   Strengths and Limitations .......................................................................... 57
   Future Directions ...................................................................................... 58
   Conclusions ............................................................................................... 59

REFERENCES ........................................................................................................ 60

APPENDIX A (Tables) ......................................................................................... 78

APPENDIX B (Figures) ...................................................................................... 85

APPENDIX C (IRB Form) ................................................................................... 88
## LIST OF TABLES

<table>
<thead>
<tr>
<th>Table</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>I. Participant Characteristics</td>
<td>79</td>
</tr>
<tr>
<td>II. Correlation Matrixes within Single, Combined Group</td>
<td>80</td>
</tr>
<tr>
<td>III. Correlation Matrixes within Non-Asthma Group</td>
<td>81</td>
</tr>
<tr>
<td>IV. Correlation Matrixes within Asthma Group</td>
<td>82</td>
</tr>
<tr>
<td>V. ANOVA: Non-Asthma group vs. Asthma Group</td>
<td>83</td>
</tr>
<tr>
<td>VI. ANOVA: Mild Asthma Severity vs. Mod-to-Severe Asthma Severity</td>
<td>84</td>
</tr>
</tbody>
</table>
## LIST OF FIGURES

<table>
<thead>
<tr>
<th>Figure</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>I.</td>
<td>Exponential versus Hyperbolic Discount Curves</td>
</tr>
<tr>
<td>II.</td>
<td>Hyperbolic Curve: Two Rewards, Different Sizes, Different Times</td>
</tr>
</tbody>
</table>
CHAPTER I
INTRODUCTION

Historically, research in delay/temporal discounting has focused on areas such as substance use, impulsivity, gambling, personality disorders, and executive functioning (Alessi & Petry, 2003; Barkley, Edwards, Laneri, Fletcher, & Matevia, 2001; Bickel, Odum, & Madden, 1999; Dixon et al., 2005; Kirby, Petry, & Bickel, 1999; Madden, Petry, Badger, & Bickel, 1997; Mitchell, 1999; Perry, Larson, German, Madden, & Carroll, 2005; Petry, 2002). Delay discounting has been defined as the extent to which an individual discounts the value of a reward (e.g. money, food, weight loss, etc.) as a function of having to wait for it (Reynolds, Richards, Horn, & Karraker, 2004). Temporal discounting, considered a component of the impulsiveness construct, refers to the reduction in the present subjective value of an outcome as the delay to that outcome is increased (Yi, Buchhalter, Gatchalian, & Bickel, 2007). Although it is in our nature to choose a larger reward as opposed to a smaller reward, that preference may shift to the smaller reward if a delay to the larger amount were added and gradually increased (Reynolds et al., 2004). According to Green and Myerson (2004), preference reversals occur because the subjective value of smaller, sooner rewards increases more than that of larger, later rewards when there is an equivalent decrease in the delays to the two rewards.

Temporal discounting has been typically assumed to be exponential by economists and researchers in decision analysis, with subjective value decreasing by a constant percentage per unit of time (Green & Myerson, 2004). However, an exponential formula does not adequately explain preference reversals and alternatives have been proposed. Chief among them is Mazur’s
(1987) proposal that the discounting function is a hyperbola, as expressed in the following equation:

\[ V = A/(1 + kD) \]

Where \( k \) is a parameter governing the rate of decrease in value, \( V \) is the subjective value of a future reward, \( A \) is its amount, and \( D \) is the delay until its receipt. A hyperbolic function attempts to relate the subjective value of a reward to the time it is received (Green, Fristoe, & Myerson, 1994). According to Ainslie (2005), when asked their preferences between pairs of a smaller amount of money at delay \( D \) and a larger amount at delay \( D + L \), people regularly reverse their preferences between the same pair as a function of \( D \), and show an overall pattern of choice described by: Value = Value-if-immediate / (1 + (Constant X Delay )). The finding that a hyperbolic function better fits temporal discounting than an exponential function has been replicated in several studies (Green, Myerson, & McFadden, 1997; Kirby, 1997; Kirby & Marakovic, 1995; Kirby & Santiesteban, 2003; Rachlin, Raineri, & Cross, 1991; Simpson & Vuchinich, 2000). This hyperbolic equation, where the relative decline in subjective value decreases as delay increases, has proven to be a superior model of discounting behavior to the exponential equation (Yi, Gatchalian, & Bickel, 2006). Put simply, hyperbolic curves predict an urge to satisfy appetite prematurely (Ainslie, 2005). Figure 1 demonstrates the difference between hyperbolic and exponential curves.

Delay discounting is generally assessed by having the respondents choose between a relatively small but immediate outcome and a relatively large but delayed outcome. The value of the immediate outcome is typically titrated across choices. The goal is to find the indifference point – the point at which the smaller sooner outcome and larger delayed reward outcome have equal value (Odum, Baumann, & Rimington, 2006). Collins, Vincent, Soracco, and Lovallo
conceptualize $k$ as an indifference point where the individual finds the immediate value equal to the delay value. Smaller values of $k$ are indicative of slower rates of discounting, while larger values of $k$ represent faster and steeper rates of discounting. Thus, individuals who quickly devalue delayed rewards produce large values of $k$ and have a very small, immediate indifference point (Collins et al., 2005).

In essence, the technique determines the present value of a delayed outcome. When this procedure is repeated at various delays to the larger later outcome, it generates a series of indifference points that can be plotted to examine the manner in which the value of an outcome decays with delay to its receipt (Odum, Baumann, & Rimington, 2006). It should be noted that in general, studies of delay discounting use hypothetical outcomes. However, several studies (Johnson & Bickel, 2002; Lagorio & Madden, 2005; Madden, Begotka, & Raiff, 2003; Madden, Raiff, & Lagorio, 2004) have demonstrated that discounting rates are similar between hypothetical and actual outcomes.

Similar results have been found when employing tasks that change the probability of the reward rather than the delay (Mitchell, 1999; Richards, Zhang, Mitchell, & de Wit, 1999). The value of a probabilistic reward decreases as its probability decreases (Richards, Zhang, Mitchell, & de Wit, 1999). While delay and probability discounting are mathematically equivalent, they present differently in measures of discounting. For example, a delay discounting task would include items such as “Would you prefer $20 today or $30 in 20 days?” A probability discounting task, on the other hand, would include items such as “Would you prefer $20 for sure, or $30 with a 75% chance?”

In general, delay and probability discounting are correlated with one another, with correlations ranging from 0.40 to 0.75 (e.g., Mitchell, 1999; Richards et al., 1999), which
represents substantial between-study variability in DD/PD correlations (Reynolds et al., 2004). However, research has found some differences not only in how these tasks present outcomes, but possibly in what they actually measure. In a study by Mitchell (1999) probability discounting was not found to be as good at differentiating between adult smokers and non-smokers as delay discounting (Mitchell, 1999). However, another study by Reynolds, Karraker, Horn, and Richards (2003) found that probability discounting differentiated better than delay discounting those adolescents who had recently tried smoking versus those who had never tried smoking. Further, those adolescents who had tried smoking demonstrated significantly higher levels of impulsivity than those who had never tried smoking.

Extant literature indicates that there is indeed an association between substance use and rates of discounting rewards, with individuals such as problem drinkers, smokers, and marijuana smokers tending to discount the value of future hypothetical rewards more steeply than their peers (e.g., Kollins, 2003; Petry, 2002; Reynolds, Richards, Horn, & Karraker, 2004; Vuchinich & Simpson, 1998). Various explanations for these choices include delayed rewards taking too much effort, uncertainty of obtaining delayed rewards, and poor impulse control (Burlingame, 1998; Mitchell, 2004; Monterosso & Ainslie, 1999; Petry, 2003; Petry & Casarella, 1999).

Related to this is literature describing the frequent comorbidity of substance use with depression (Chang, 1997; Kelly, McKellar, & Moos, 2003; Miller, Hoffmann, Ninonuevo, & Astrachan, 1997; Swendsen & Merikangas, 2000). A study by Miller, Klamen, Hoffmann, and Flaherty (1996) analyzed 6,355 alcohol- and drug-dependent patients from 41 sites. The results indicated that not only was there a relationship between substance use and depression, but that 44% of these individuals had a lifetime history of major depression. Naturally, in the past few decades, discounting research has begun to investigate the relationship between discounting and
depression, with much literature already demonstrating a link between depression and rates of reinforcement (Heiby, Ozaki, & Campos, 1984; Schill & Kramer, 1991; Welker, 1989). The literature indeed seems to indicate that individuals with depression engage in activities that provide them with immediate reinforcement (e.g., using substances, staying in bed, staying at home) rather than seeking the reinforcers that require more effort and may be more uncertain (e.g., going to the gym, taking a walk in the park, socializing with friends at a party).

The Problem

Research concerning the relationship between delay discounting and depression has yielded mixed findings. An early study by Rehm and Plakosh (1975) found that individuals scoring high on measures of depression were less inclined to choose a delay over an immediate reward. In other words, depressed individuals preferred immediate rewards as opposed to delayed rewards. However, other research (e.g., Burlingame, 1998; O’Hara & Rehm, 1982) were unsuccessful in finding a significant relationship between individuals’ scores on a measure of depression (Beck Depression Inventory-II – BDI-II) and the length of delay of the reward.

Theoretically, individuals with significant levels of depression will seek immediate reinforcement rather than delayed reinforcement (Rehm & Plakosh, 1975). Behavioral models of depression (Ferster, 1973; Lewinsohn, 1974) further support the notion that individuals demonstrating depressive symptomatology lack the skills necessary to obtain the type of long-term reinforcement that would promote healthy behavior (e.g., working out at the gym, adhering to the diet). Instead, short-term, immediate reinforcers are chosen, such as staying at home and watching television or violating the rules of a diet.

However, not all research has shown a correlation between rates of reinforcement and discounting. A possible explanation for these incongruent findings is the presence of certain
confounds, namely the methods used to measure levels of depression and discounting. If attributes of depression were to be approached and measured using alternative methods, it is possible that a stronger relationship might be observed without requiring subjects to endorse high levels of depressive symptomatology.

An application of the reformulated helplessness theory to depression by Abramson, Seligman, and Teasdale (1978) resulted in predictions that individuals who habitually explain bad events by internal, stable, and global causes will be more prone to depressive episodes than persons without this maladaptive explanatory style. In other words, individuals with a negative attributional style are more prone to experience depression. To reiterate, by investigating an individual’s attributional style in addition to their levels of depressive symptomatology, researchers may find a more stable relationship to rates of discounting.

Notably, a relatively unexplored area in the literature concerns how probability discounting tasks are associated with depression. The inclusion of a probability discounting task may help reflect aspects of a negative attributional style and depression by addressing an individual’s negative beliefs and pessimistic attitude when making a choice that relies on chance. In the same way that a delay discounting task addresses an individual’s reluctance to exert any effort to obtain rewards, a probability discounting task may address the pessimism and negative attributional style that is often characteristic of individuals with depression. Therefore, by approaching these aspects of depression and discounting, it is expected that stronger relationships will be found.

The target population is another factor that may influence the strength of correlations found between depressive symptomatology and rates of discounting. More specifically, a healthy population may exhibit lower levels of depressive symptomatology and a more optimistic
attributional style than a chronic illness population. For exploratory purposes, individuals with asthma were identified so as to compare the two groups.

Research by Mullins, Chaney, Balderson, and Hommel (2000) found a significant association between increased illness uncertainty and increased levels of depression in a college sample of 40 students with histories of childhood asthma. Additionally, other researchers have found a correlation between chronic illnesses and levels of depression and psychological distress (Halligan, 1983; Ludman et al., 2004; Mullins, Chaney, Pace, & Hartman, 1997; Paschalides et al., 2004; Silverglade, Tosi, Wise, & D’Costa, 1994; Whittemore, Melkus, & Grey, 2004). Recent research by Chaney et al. (2004) provides further support for the relationship between attributional style and depression in individuals with chronic illnesses. More specifically, Chaney et al. (2004) found that individuals who endorsed a more helpless cognitive style characterized by pervasive personal causal inferences (internal attributions) across a wide array of negative life events (global attributions) were at increased risk for later depressive symptoms. Earlier research by Schiaffino, Shawaryn, and Blum (1998) has also shown a relationship between internal attributions and the likelihood of experiencing helplessness and depression in individuals with Rheumatoid Arthritis.

Consequently, it seems that individuals with a chronic illness provide a unique population to investigate variables of depression, attributional style, and rates of reinforcement. According to the extant literature, it is more likely that a sample of college students with asthma will exhibit higher levels of depression and/or a negative attributional style than a sample of healthy college students (e.g., Bender, Klinnert, Kotses, & Harver, 1998; Harm, Marion, Kotses, & Creer, 1984; Miller, 1987). Chaney et al. (1999) and Badoux-Levy, Robin, Lavarde, and Grygielski (2004) have reported prevalence rates of depression in individuals with asthma at 21% and 25%
respectively. Notably, research indicates that individuals with asthma are at a greater risk for developing anxiety and anxiety disorders, although, given the overlap of anxious and depressive symptomatology, it is sometimes difficult to distinguish between anxiety and depression and to determine which is more influential to adjustment (Hommel, Chaney, Wagner, & McLaughlin, 2002). Thus, differential assessment of these affective expressions is essential in determining their independent contributions to asthma-related variables (Hommel et al., 2002).

The following literature review will begin with a description of delay and probability discounting, including recent research conducted using discounting tasks. This is followed by a review of the literature regarding depression and attributional style. More specifically, the development of Seligman’s (1975) Learned Helplessness theory into the Reformulated Theory of Learned Helplessness (Abramson, Seligman, & Teasdale, 1978) will be explained. A discussion of relevant behavioral models of depression will follow.

Research on psychological factors associated with chronic illnesses in general, and asthma in particular, will then be examined. The psychosocial variables attributional style and depression will be discussed in the context of chronic illnesses. The hypothesized relationships between the variables will then be set forth. For the purposes of this study, individuals with asthma were identified to run exploratory analyses comparing healthy college students to college students with asthma.
CHAPTER II
LITERATURE REVIEW
Delay and Probability Discounting

Definition and Description

It is in the nature of humans and animals to choose a larger reward over a smaller reward, a reward that is given sooner than later, and to choose a high probability of obtaining a reward rather than a low probability of obtaining the same reward. However, there are times when we need to make a choice between small, highly probable rewards and larger, less probable rewards. For example, a radio station held a quiz competition where individuals called in and, if they answered a question correctly, they were given a choice between $50 and what was in “the box”. There was a small chance that the prize in the box was a sports car, although the alternative was a prize worth less than $50. Another example is taken from the world of horse racing, where an individual has the option of placing a lesser paying but more reliable bet on the favored horse, or the individual may choose to place a bet of a higher paying horse that is considered to be a long shot. This is a choice between a smaller, less risky reward and a larger, more risky reward (Green & Myerson, 2004).

According to Green and Myerson (2004), there are three dimensions on which rewards can differ, namely amount, delay, and probability. Amount refers to the size of the reward; the larger the reward, the more value associated with it. Delay refers to a temporal measurement; the longer the delay, the longer the individual has to wait for his/her reward. This delay may be
measured in seconds, minutes, hours, days, and even years. Finally, the probability of obtaining the reward may be measured in terms of percentages, with a low percentage being a low probability of obtaining the reward. Further, Yi, Gatchalian, and Bickel (2006) report that the extensive literature on future discounting indicates some consistent findings. More specifically, the hyperbolic equation better explains temporal discounting, large-magnitude outcomes are discounted less than small-magnitude outcomes (magnitude effect), and positive outcomes (gains) are discounted more than negative outcomes (losses) (Yi, Gatchalian, & Bickel, 2006).

Many researchers have investigated the influence of delay and probability discounting in the context of alcohol and drug abuse, gambling, personality disorders, and impulsivity (e.g., Alessi & Petry, 2003; Baker, Johnson, & Bickel, 2003; Bickel & Marsch, 2001; Eysenck, Eysenck, & Barrett, 1985; Holt, Green, & Myerson, 2003; Ostaszewski, 1996; Petry, 2001a; Petry, 2001b; Vuchinich & Simpson, 1998). Findings indicate that impulsive individuals, such as gamblers, psychiatric outpatients, children with ADHD, and drug abusers, discount the value of delayed rewards more and, therefore, demonstrate poor behavioral inhibition (de Wit & Richards, 2004; Green & Myerson, 2004). These studies also reveal that extraverts and individuals high on impulsivity show steeper discounting of hypothetical delayed rewards than introverts and individuals low on impulsivity (Green & Myerson, 2004).

Research findings concerning the relationship between delay discounting and depression have been mixed. More specifically, an early study by Rehm and Plakosh (1975) found that individuals scoring high on measures of depression were less inclined to choose a delay over an immediate reward. In other words, they preferred immediate rewards as opposed to delayed rewards. However, other findings (Burlingame, 1998; O’Hara & Rehm, 1982) were unsuccessful
in finding a significant relationship between individuals’ scores on a measure of depression (Beck Depression Inventory-II – BDI-II) and the length of delay of the reward.

The current research will investigate the relationship between probability discounting and depression. It is believed that the probabilistic nature of the choices in the experimental task may better discriminate between higher and lower levels of depression. To date, there has been less research on the construct of probability discounting than there has on delay discounting. Research findings comparing the two constructs (Christensen, Parker, Silberberg, & Hursh, 1998; Du, Green, & Myerson, 2002; Green, Myerson, & Ostaszewski, 1999; Myerson, Green, Hanson, Holt, & Estle, 2003) indicate that, although the same mathematical function describes both delay and probability discounting, humans may discount smaller probabilistic rewards less steeply than larger probabilistic rewards. Therefore, although the two constructs may appear to be mathematically equivalent, they appear to have different underlying processes. With this in mind, the current study will also investigate the correlation between probability discounting and attributional style, in an attempt to find a correlation with a construct that may reflect depression in another manner. Extant literature suggests that there is indeed a relationship between depression and attributional style. It is believed that by measuring an individual’s attributional style, a correlation may be found between a depressive attributional style and steeper rates of probability discounting.

According to Reynolds, Karraker, Horn, and Richards (2003), the outcome measure of interest in probability discounting is the extent to which an individual discounts the value of a larger reinforcer as a function of a decreasing probability of its delivery. In other words, individuals who engage in probability discounting place less value on a reinforcer as the probability of attaining the reinforcer decreases. The size of the certain reward that just balances
a probabilistic reward may be called the ‘certain value’ of the probabilistic reward. Certain Value would be expected to be a monotonic increasing function of probability; when probability equals 1.0, certain value equals the full size of the reward; as probability approaches zero, certain value approaches zero (Rachlin, Brown, & Cross, 2000). Therefore, as the probability of obtaining the reward decreases, humans begin to place less value on the reward. For example, if there was a 100% chance of receiving a reward, that reward’s value would be a 10 (an arbitrary number). However, if there was a 25% chance of receiving a reward, that reward would decrease in value and may become a 3. The less chance an individual has of obtaining that reward, the less value that person places on it. It is clear that certain value and probability are directly proportional and have a positive correlation. An examination of current research findings will now be presented.

Research on Discounting

The number of experimental studies that approach choice from the discounting perspective has been rapidly increasing in recent years and appears to be because the concept of discounting provides a potentially unifying theoretical approach that may be applied to diverse issues in psychology, such as self-control, impulsivity, and risk taking (Green & Myerson, 2004). Indeed, much of the current discounting research to date has sought to determine its relation to impulsivity, drug-use, cigarette smoking, gambling, and alcohol, exploring both temporal (delay) and probability discounting, determining differences and similarities between the two constructs.

Recently, deWit, Flory, & Acheson (2007) examined both self-reported impulsivity (using the Barratt Impulsiveness Scale - BIS) and delay discounting in a large sample of adults ($N = 606$). The researchers also examined the scores from the self-report questionnaire and behavioral measure of discounting in relation to demographic characteristics such as age, sex, race, IQ, years of school, and family income. Results of the study indicated that preference for
immediate rewards was related to the Nonplanning impulsiveness subscale of the BIS, thus further supporting research linking impulsivity to discounting. Also, after controlling for other variables, the researchers found that preference for immediate rewards was related to intelligence.

Other studies looking at discounting differences between outcomes have shown steeper discounting with consumable products than with money (Odum & Rainaud, 2003; Petry, 2001a). Odum, Baumann, and Rimington (2006) proposed that these results could be questioned due to the large and unrealistic amounts of consumables that were presented. In a study to discover whether relatively small amounts of food would be discounted more steeply than relatively small amounts of money (Odum et al., 2006), it was still found that college students ($N = 102$) discounted food more steeply than money. This finding is concurrent with previous findings demonstrating steeper discounting with consumable products than with money.

Researchers have also studied the relationship between discounting of future and past outcomes. To examine the possibility that outcomes in the past are discounted hyperbolically and at a similar rate to outcomes in the future, Yi, Gatchalian, and Bickel (2006) used a paper-and-pencil questionnaire of future and past discounting to obtain indifference points of college students. Their study revealed that the students did indeed discount temporally past outcomes similarly to future outcomes. In fact, discounting of past outcomes is orderly, hyperbolic, and consistent with most empirical observations from studies of future discounting (Yi, Gatchalian, & Bickel, 2006).

In an attempt to determine whether discounting rates may be adaptable and that drug abstinence may be associated with reductions in discounting, Heil, Johnson, and Higgins (2006) studied discounting differences between currently abstinent and currently using cocaine-
dependent outpatients. The researchers investigated whether a period of abstinence (30 days) resulted in altered discounting rates of money in a population of primary cocaine-dependent patients compared to current cocaine users and non-drug-using matched controls (Heil et al., 2006). The results indicated that there was no difference in discounting between currently abstinent and currently using cocaine-dependent outpatients. When drug users were combined into a single group and compared to the non-drug-using controls, cocaine-dependent patient showed higher rates of discounting, further supporting evidence that higher rates of discounting are found in substance-users than non-drug-users.

Extensive research has been conducted regarding nicotine use and discounting. Recently, Reynolds, Richards, Horn, and Karraker (2004) examined relations between adult smokers and non-smokers and the devaluation of monetary rewards as a function of delay and probability discounting. They considered those individuals who discounted the most as being more impulsive. Participants in the study included 25 female adult smokers and 29 non-smokers, with the smokers reporting smoking at least 20 cigarettes per day and the non-smokers having never smoked. Results of the study indicated that there was a significant difference in discounting between smokers and non-smokers, with the smokers discounting more by both delay and probability. However, delay discounting was found to be a significantly stronger predictor of smoking than probability discounting. This study further supported the literature regarding a link between discounting behavior and cigarette smoking.

A prior study by Reynolds, Karraker, Horn, and Richards (2003) investigated the relation between different patterns of adolescent cigarette smoking and discounting of monetary rewards due to delay and probability discounting. Participants included 55 adolescents (28 females) between the ages of 14 and 16 years who were categorized according to one of three patterns of
smoking behavior: never smokers, triers, and current smokers. Contrary to previous literature, results indicated that there were no differences in discounting between current smokers and never smokers. However, significantly more probability discounting behavior was found in the “trier” group than the never- and current-smoker groups. According to the researchers, these results suggest that impulsive discounting may be more related to adolescents trying cigarettes than to their becoming regular smokers. However, it seemed that number of peer friends who smoke and parent level of education better differentiated between individuals who have smoked to some extent and those who have not even tried cigarettes.

Holt, Green, and Myerson (2003) examined temporal and probability discounting in gambling and non-gambling students, using discounting tasks to determine whether the two groups differed in the degree to which they discounted delayed and probabilistic rewards. Participants included 48 18- to 24-year old college undergraduates at a state university in the Midwest. Each group consisted of 19 students (13 male and 6 female). Differences in temporal and probability discounting were noted, as both groups discounted large delayed amounts less steeply than small delayed amounts, but discounted large probabilistic amounts more steeply than small probabilistic amounts. Also, results indicated that gamblers discounted probabilistic rewards less steeply than non-gamblers, suggesting that gamblers are impulsive in the sense that they are less affected by risk than non-gamblers (Holt, Green, & Myerson, 2003). The authors suggest that those individuals who are less affected by risk are not necessarily more affected by delay; therefore the mechanisms underlying the discounting of delayed and probabilistic rewards can be viewed as separate. The results further argue against the view that impulsivity is a general trait that includes both an inability to delay gratification and a tendency to take risks (Holt, Green, & Myerson, 2003).
Myerson, Green, Hanson, Holt, and Estle (2003) examined discounting of delayed and probabilistic rewards in two samples. Findings indicate that the amount of reward had opposite effects on temporal and probability discounting. More specifically, smaller delayed rewards were discounted more steeply than larger delayed rewards, whereas larger probabilistic rewards were discounted more steeply than smaller probabilistic rewards. This further supports the notion that, although the temporal and probability discounting functions are similar mathematically, separate processes may underlie the discounting of delayed and probabilistic rewards (Myerson et al., 2003).

Du, Green, and Myerson (2002) studied delay and probabilistic discounting in 28 American, 28 Chinese, and 23 Japanese graduate students in an effort to investigate cross-cultural differences. Initial analyses revealed that, for all three groups, the rate at which delayed rewards were discounted was higher for the smaller amount whereas the rate at which probabilistic rewards were discounted was lower for the smaller amount, thus further supporting the idea that there are differences in delay and probabilistic discounting with regard to the size of the amount. Cross-cultural differences were found, with the Americans and Chinese discounting delayed rewards more steeply than the Japanese. In addition, the Americans discounted probabilistic rewards the most, whereas the Chinese discounted probabilistic rewards the least. This study further indicates that, although similar, delay and probabilistic discounting tasks may differ in the extent to which they can note differences between groups, with one task often being more sensitive than the other.

Rachlin, Brown, and Cross (2000) investigated whether two equations accounted for judgments as well as choices among delayed and probabilistic rewards and to see whether the amount was eliminated or reversed by using a judgment procedure. Consistent with previous
findings, the delayed and probabilistic discount functions obtained were hyperbolic in nature, rather than exponential, thus demonstrating their mathematical similarity. However, differences were noted, as steepness of delay discounting was not systematically related to standard money amount but probabilistic discounting was steeper for higher standard amounts than for lower amounts. In other words, there was more discounting in the probabilistic discounting task when larger values were involved, than there was in the delay discounting task.

Richards, Zhang, Mitchell, and de Wit (1999) assessed impulsivity in humans using a computer task that measured delay and probability discounting. Twenty-four adults ingested a moderate dose of ethanol or a placebo before completing the discounting task. The results indicated that delay and probability discounting were mathematically similar, both being well described by a hyperbolic function. In addition, delay and probability discounting were positively correlated within subjects, indicating that individuals who engaged in higher discounting of delayed rewards also engaged in higher discounting of probabilistic rewards. With regard to the alcohol, no significant effect of alcohol on discounting was observed. The researchers noted that the degree of discounting was modestly correlated with scores on paper-and-pencil tests of impulsivity, suggesting that the behavioral discounting procedures may be an efficient and sensitive measure of impulsive behavior.

A study by Ostaszewski (1997) examined the relationship between several temperamental traits (sensation seeking, extraversion, and impulsivity) and discounting of delayed and probabilistic monetary rewards in 18-39 yr old 1st-year psychology students. The results revealed that discounting of delayed rewards was related to extraversion and impulsivity, but not sensation-seeking. More specifically, extroverts and individuals scoring high on measures of impulsivity engaged in steeper delay discounting than individuals scoring high on sensation-
seeking. However, discounting of probabilistic rewards did not differ between high and low impulsive individuals or between introverts and extraverts, suggesting that delay and probability discounting tasks are not functionally equivalent (Ostaszewski, 1997).

It is apparent that there have been mixed findings regarding group differences in delay and probability discounting. In fact, the literature indicates that researchers often have difficulty replicating a study to obtain similar results. The research studies mentioned above indicate that, although delay and probability discounting appear to be mathematically identical, and are positively correlated when predicting certain personality traits (such as impulsivity), they sometimes predict other behaviors differently. For example, Reynolds et al. (2004) revealed that delay discounting was a better predictor of smoking than probability discounting. Holt et al. (2003) indicated that both gambling and non-gambling students discounted large delayed amounts less steeply than small delayed amounts, but discounted large probabilistic amounts more steeply than small probabilistic amounts. Rachlin, Brown, and Cross (2000) found that, although mathematically similar, differences in steepness of discounting in delay and probabilistic discounting tasks were noted, with more discounting occurring in the probabilistic discounting task than the delay discounting task when larger values were involved.

Notably, a relatively unexplored area in the discounting literature concerns the effectiveness of levels of depression and attributional style as predictors of scores in delay and probabilistic discounting tasks. Indeed, historical and extant research on depression and attributional style suggests that these factors most likely have a notable effect on an individual’s decision-making style as well as reinforcement-seeking behaviors. The following section will discuss the link between behavioral models of depression and economic theory of discounting. The development of Seligman’s (1975) Learned Helplessness theory of depression into
Abramson, Seligman, and Teasdale’s (1978) reformulated theory to include attributional style will be then presented. Also, the relationship between depression and attributional style will be expounded, in order to explain the inclusion of both these constructs in the current study.

Depression and Attributional Style

Behavioral Models of Depression

According to Hopko, Lejuez, and Ruggiero (2003), original behavioral models of depression implicated decreases in response-contingent reinforcement for non-depressive behavior as the causal factor in eliciting depressive affect. A functional analytic view would suggest that continued engagement of depressed behavior must result from some combination of reinforcement for depressed behavior and a lack of reinforcement or even punishment of more healthy alternative behavior (Ferster, 1973). More specifically, Ferster (1973) emphasized depression as a function of positive reinforcement deprivation – in terms of low density of positive reinforcement. Other theorists (Hoberman & Lewinsohn, 1985; Lewinsohn, 1974) viewed depression in terms of low rates of response-contingent positive reinforcement.

Behaviorists often place an emphasis on the role of the environment on depressive behavior. This contextual perspective (a) sees behavior as a function of the environmental contingencies that shape and maintain its occurrence and (b) encourages the identification of environment-behavior relations that may be measured objectively and reliably (Zuriff, 1986).

In other words, according to these behavioral theories of depression, depression is the result of a lack or decrease of positive reinforcement in an individual’s environment, thereby reducing overall behavioral frequency and variability. Functionally, the important point is that it is a generalized reinforcer that is lost and extinction over-generalizes to many behavioral repertoires at least partially controlled by or associated with the lost reinforcer (Kanter, Cautilli,
Depressive behavior, such as passivity and negative affect, strengthens as a result of environmental contingencies that function to decrease the rate of “healthy” responses within one’s behavioral repertoire and increase avoidance of aversive stimuli (Ferster, 1973).

Lewinsohn (1974) attributed the reduction of positively reinforced healthy behavior to a decrease in the number and range of reinforcing stimuli available to an individual for such behavior and/or a lack of skill in obtaining reinforcement. Individuals with depression can therefore be described as not having the skills to seek out positive reinforcement from their environment. From a discounting perspective, the value of the delayed reinforcers (e.g., working out at the gym or going for a walk in the park) has decreased or become a low level reinforcer for the depressed individual. In turn, the value of more immediate reinforcers (e.g., staying at home, over-eating) increases and individuals demonstrate steeper levels of discounting. Lewinsohn (1974) theorizes that depression involves a functional attempt to gain immediate reinforcement, often at the expense of more valuable, long-term reinforcement. In fact, the Behavioral Activation treatment approach to depression views this depressed behavior (e.g., inactivity, withdrawal) as a coping strategy to avoid environmental circumstances that provide low levels of positive reinforcement or high levels of aversive control (Jacobson, Martell, & Dimidjian, 2001). Behavioral Activation has as its theoretical underpinnings, Ferster’s (1973) functional analysis of depression, which considers an assessment of the function of behavior rather than the form as important in facilitating clinical change. After establishing recognition of avoidance patterns, the principal objective becomes one of helping the patient to reengage in various healthy behaviors through the development of alternative coping strategies (Hopko et al., 2003).
Herrnstein (1970) states that the absolute rate of any response is proportional to its associated relative reinforcement. Herrnstein’s mathematical statement of the law of effect improves on Skinner’s view of reinforcement by asserting (a) that response rate varies hyperbolically with reinforcement rate and (b) that responding is governed not only by reinforcement obtained for responding but also by reinforcement obtained from all other concurrent sources (McDowell, 1982). In sum, researchers (Herrnstein, 1970; McDowell, 1982) have proposed that, in the context of depression, matching theory suggests that the relative value of reinforcement obtained for depressed versus non-depressed behavior is directly proportional to the time and effort allocated to exhibiting depressed relative to non-depressed behavior.

According to Hopko et al. (2003), when the value (e.g., accessibility, duration, immediacy) of reinforcers for depressed behavior is increased through environmental change (e.g., increased accessibility to social attention, increased opportunities to escape aversive tasks), the relative value of reinforcers for healthy behavior decreases, increasing the likelihood of depressive behavior. On the flip side, when the value of reinforcers for healthy behavior is decreased through environmental change (e.g., decreased availability of peers), the relative value of reinforcers for depressed behavior is simultaneously increased.

Individuals with depression therefore appear to demonstrate hyperbolic discounting, temporarily preferring options that pay off quickly to richer, but slower-paying alternatives (Ainslie, 2005). Figure 2 depicts hyperbolic discount curves from two rewards of different sizes available at different times. The smaller reward is temporarily preferred for a period just before it is available, as shown by the portion of its curve that projects above that from the later, larger reward.
According to Harris and Laibson (2001), discount curves which are more bowed (steeper discounting) than exponential ones better fit people’s lifetime patterns of spending and saving, and might explain such notorious anomalies as borrowing at 15% while keeping investments that earn 5%. While exponential curves may seem more logical, there is more and more evidence that people’s natural discount curve is not only non-exponential, but specifically hyperbolic (Ainslie, 2005).

Learned Helplessness Theory of Depression

Learned Helplessness theory was developed by Martin Seligman in the 1970s and had a major impact on psychological research in depression. Seligman discovered the phenomenon of helplessness while investigating the effects of inescapable shock on active avoidance learning in dogs. The central idea in the original Learned Helplessness theory (Maier & Seligman, 1976; Maier, Seligman, & Solomon, 1969; Seligman, 1975) is the notion that all animals (including humans) are able to learn that reinforcers are uncontrollable; learned helplessness is believed to result from learning that reinforcement and responding are independent. So, the learned helplessness model of depression predicts that depressed subjects should perceive reinforcement as more response-independent than non-depressed subjects in skill tasks where reinforcement is actually response-dependent; in chance tasks, where reinforcement and responding are actually independent, both depressed and non-depressed subjects should perceive reinforcement as response-independent (Miller & Seligman, 1975). Further, Miller and Seligman (1975) state that depressed subjects should exhibit smaller expectancy changes than non-depressed subjects in skills tasks, while depressed and non-depressed subjects should exhibit similar expectancy changes in chance tasks. Therefore individuals who have experienced non-contingent reinforcers exhibit smaller expectancy changes.
This expectation of no control leads to motivational deficits (lowered response initiation and lowered persistence), cognitive deficits (inability to perceive existing opportunities to control outcomes), and, in humans, emotional deficits (sadness and lowered self-esteem); these deficits are collectively known as learned helplessness deficits (Nolen-Hoeksema, Girgus, & Seligman, 1986).

However, not all individuals exposed to the helplessness-producing circumstances will become helpless (Buchanan & Seligman, 1995). The theory was reformulated (Abramson, Seligman, & Teasdale, 1978) in order to take account of explanatory style (i.e., the way people explain negative events to themselves). The attribution reformulation addresses this issue of individual differences and predicts who is more vulnerable or resistant to learned helplessness (Abramson et al., 1978; Seligman, Abramson, Semmel, & von Baeyer, 1979). The reformulated model thus is a diathesis-stress model, in which a bad explanatory style is viewed as a factor that predisposes the individual to helplessness in the face of bad events (Nolen-Hoeksema et al., 1986).

**Attributional Style**

Abramson et al. (1978) proposed that once people perceive a non-contingency, they attribute their helplessness to a cause. This cause can be stable or unstable, global or specific, and internal or external. The stable-unstable dimension is concerned with the permanence of transience or that cause; the global-specific dimension involves attributing the event to a cause that will affect many areas of an individual’s life or just one particular event; and the internal-external dimension that involves attributing the event to something about the individual or something outside of the individual (Buchanan & Seligman, 1995). Therefore, according to the reformulation, individuals who habitually attribute negative events to internal, stable, and global
causes and positive events to external, unstable, and specific causes (the pessimistic style) are at greater risk for helplessness deficits than those people with the opposite, optimistic style (Buchanan & Seligman, 1995).

Therefore the hypothesized depressive explanatory style in individuals with depression is characterized by internal, stable, and global attributions for negative events and external, unstable, and specific attributions for positive events (Buchanan & Seligman, 1995). Abramson et al. (1978) applied the reformulated helplessness theory to depression and predicted that individuals who habitually explain bad events by internal, stable, and global causes will be more prone to depressive episodes than persons without this maladaptive explanatory style. Studies using adults and children have confirmed this prediction (e.g., Alloy, Peterson, Abramson, & Seligman, 1984; Peterson & Seligman, 1984).

The attribution chosen influences whether expectation of future helplessness will be chronic or acute, broad or narrow, and whether helplessness will lower self-esteem or not (Abramson et al., 1978). In 1975, Miller and Seligman found that individuals with depression were less likely to be motivated and initiate responses than individuals who were not depressed. Also, Peterson and Seligman (1984) employed a variety of new investigations of the helplessness reformulation, including cross-sectional correlational studies, longitudinal studies, experiments of nature, laboratory experiments, and case studies. The researchers demonstrated that people who have an explanatory style characterized by internal, stable, and global explanations for bad events are more likely to become depressed following uncontrollable, aversive events (Buchanan & Seligman, 1995), thereby further supporting Seligman’s (1975) reformulated Learned Helplessness model of depression. The 12 studies they reviewed (Peterson & Seligman, 1984) confirmed the reformulation of Seligman’s (1975) Learned Helplessness theory.
A longitudinal study by Nolen-Hoeksema, Giegus, and Seligman (1986) measured the depressive symptoms, life events, and explanatory styles of 168 school children 5 times during the course of 1 year. Participants included 87 males and 81 females, ranging in age from 8 to 11 years. Results found that the maladaptive explanatory style not only correlated with concurrent depression but also predicted later depression (Nolen-Hoeksema et al., 1986). In other words, children with the maladaptive explanatory style at time $n$ had higher levels of depression at time $n + 1$ than did children with the optimistic explanatory style. Further, the researchers found that depression at time $n$ predicted explanatory style at time $n + 1$. Other researchers (Andrews & Debus, 1978; Chapin & Dyck, 1976; Dweck, 1975; Folwer & Peterson, 1981) have shown that children who are given attribution retraining show decreases in helplessness deficits in cognitive tasks and that the effects of retraining persist for at least several months (Nolen-Hoeksema et al., 1986).

It is apparent that individuals who engage in a pessimistic attributional style have difficulties functioning in various areas such as cognitions, motivation, and emotional well-being. Several studies have shown how depression and attributional style are interlinked (Andrews & Debus, 1978; Chapin & Dyck, 1976; Dweck, 1975; Folwer & Peterson, 1981; Nolen-Hoeksema et al., 1986). Studies have also shown that individuals with chronic illnesses may often exhibit depressive symptomatology due to the uncertain and intermittent nature of their illness (Bennett, 1994; Burke & Elliott, 1999; MacLean, Perrin, & Gortmaker, 1992; Symister & Friend, 2003). In fact, Schoenherr, Brown, and Baldwin (1992) demonstrated that attributional style was a significant predictor of youths’ self-reports of depressive symptoms. These individuals’ experiences with the non-contingent nature of their illness might result in not only an unhealthy attributional style, but also motivational and cognitive deficits (Gaudino,
Masur, & Kaufman, 1995; Prescott, Richardson, & Gillespie, 1990). If assessed with a discounting task, these cognitive deficits may emerge in a similar pattern as individuals who have depression or impairments in working memory. The following section will discuss attributional style in the context of chronic illnesses. Chronic illness may be another dimension in which to look at attributional style, as not only are individuals with depression at risk for a pessimistic attributional style and cognitive deficits, but so too are individuals with chronic illnesses.

Psychological Factors Associated with Chronic Illnesses

Attributional Style

Mullins, Chaney, Pace, and Hartman (1997) state that as a result of repeated exposure to the unpredictable nature of asthma (e.g., the number of attacks varying across time), individuals with asthma tend to have a cognitive style that includes an expectation of negative outcomes for both asthma-related and non-asthma related events, and the expectation that these negative outcomes cannot be avoided. In addition, greater perceived asthma uncertainty and increased stable attributions for negative events were significantly associated with poorer psychological adjustment after controlling for demographic and disease variables. It appears that the uncertainty about the asthma management and the expectation of negative outcome for events may contribute to the psychological distress experienced by individuals with asthma (Van Pelt, 2004).

Van Pelt (2004) investigated self-focus and psychological distress in adolescents and young adults with long standing asthma. Participants included 81 college-age students with asthma (32 males and 49 females). These students were paired with healthy controls, although three students with asthma were unable to be matched with a healthy control participant. The
participants ranged in age from 18- to 22-years. The results of her study indicated that college students with asthma, in particular those with moderate to severe asthma, evidence higher rates of general psychological distress, depression, and anxiety compared to same-age and gender peers without a chronic illness history. Asthma severity was measured on a 7-point Likert scale with 1-2 indicating mild severity and 3-7 indicating moderate to severe severity. The results of this study appear to be consistent with other research suggesting that asthma severity increases the risk for psychological distress (McQuaid, Kopel, & Nassau, 2001; Silverglade et al., 1994; Vila, Nollet-Clemencon, de Blic, Mouren-Simeoni, & Scheinmann, 1998). Concerning attributions, Van Pelt (2004) hypothesized that participants with asthma would make more internal attributions when presented with a non-contingent task. Although it was found that the participants made more external attributions, Van Pelt (2004) argued that the participants may have been aware of the deceptive nature of the experiment. She further posited that, in a real world situation, individuals with asthma may be more inclined to make internal attributions for failure than individuals with asthma and suggested that further research utilizing more subtle experimental manipulation to examine differences in attributions for task performance may be needed (Van Pelt, 2004).

Thus far the literature indicates that individuals with chronic illnesses are indeed at risk for the development of depressive attributional styles which, in turn, may affect their judgment in cognitive tasks. It has been demonstrated that individuals with a depressive attributional style are more likely to engage in discounting. In other words, individuals with a depressive attributional style, when presented with complex decisions that tax their working memory, are more likely to choose the more immediate and simple reinforcer, thereby obtaining higher k-values. It is therefore likely that individuals with a chronic illness will engage in more discounting than
individuals without a chronic illness. Naturally this may be mediated by depression, anxiety, and psychological distress.

A study by Burlingame (1998) examined the relationship between depressed mood, substance use, and temporal discounting, hypothesizing that self-reported depression scores would predict individual’s discounting rates after statistically controlling for substance use. Participants included 59 male and 45 female undergraduate students with a mean age of 20 years (SD = 3.27). Results indicated that depressed mood did not predict rates of temporal discounting. Among other problems with the study, Burlingame (1998) states that overall, the sample of students was not very depressed, with a median Inventory to Diagnose Depression (IDD) score of 9 (out of a possible 72).

It is hoped that this study will find an interaction between depressed mood, attributional style, and probability discounting. If the depression in the sample is not severe enough, then it may be possible that an individual’s attributional style may influence their discounting style. Also, it is possible that \textit{probability} discounting may more effectively measure the differences in individuals with a chronic illness and healthy individuals regarding attributional style and discounting.

\textit{Depression}

In any given 1-year period, 9.5 percent of the population, or about 18.8 million American adults, suffer from a depressive illness (Robins & Regier, 1991). According to Kessler et al. (1994) depression affects 10.3\% of the US population in any year. Further, it has become the leading cause of disability in adults, between the ages of 15 and 44, in Western Europe and North America (Murray & Lopez, 1996). It is associated with increased mortality in coronary artery disease, increased morbidity in chronic illnesses, impaired quality of life, and increased
health care cost and utilization (Januzzi, Stern, Pasternak, & DeSanctis, 2000; Mazure, Maciejewski, Jacobs, & Bruce, 2002). According to the National Institute of Mental Health [NIMH] (2003), the term medical comorbidity can be used to distinguish psychiatric-medical co-occurrence from the co-occurrence of two or more psychiatric disorders, without regard to causality. Although it is clear that depression worsens the outcome of medical illnesses (NIMH, 2003), research is still being conducted to determine whether effective treatment of depression actually improves medical conditions. Conversely, we need more data to determine whether the presence of a medical illness impairs the treatment of depression (NIMH, 2003).

Although there is much research aimed at understanding how depression arises and can best be prevented or treated in the context of general medical illness and chronic illness, for the purposes of this paper, only extant and relevant literature pertaining to chronic illness, depression, and illness uncertainty will be discussed.

Paschalides et al. (2004) examined the interrelationships of anxiety, depression, and personal illness representations with glycemic control and health-related quality of life in adults with Type 2 diabetes. Participants included 184 patients with DM2 who completed measures pertaining to perception of control, depression, and other demographic measures. Results indicated that anxiety, depression, and negative beliefs about illness influence physical and mental functioning, but not metabolic control in patients with diabetes. The results of this study provide further support for the negative influence of depression on cognitive and physical functioning in both healthy individuals as well as individuals with a chronic illness.

Across a wide range of chronic medical illnesses, patients with psychological distress also report more medical symptoms and more functional impairment than do patients with medical illnesses alone even when controlling for severity of medical illness (Katon, Sullivan, &
Walker, 2001). In a recent meta-analysis by Anderson, Freedland, Clouse, and Lustman (2001), twofold higher odds of depression in persons with diabetes compared to individuals without diabetes was reported. Indeed, there is extensive evidence that diabetes and depressive disorder occur together more often than would be expected by chance association (Eaton, 2002). A review of 9 studies with a control group (Gavard, Lustman, & Clouse, 1993) revealed that the current prevalence of depressive disorder was about twice or three times as common in diabetics as in controls. Although blood sugar levels have also been discussed as playing a role in the development of depression in individuals with diabetes, extant literature indicates that the psychological and psychosocial functioning of individuals with a chronic illness may be negatively impacted, resulting in higher levels of depression, anxiety, and general psychological distress.

Ludman et al. (2004) examined the relationship among patient-reported diabetes symptoms, severity of depressive illness, and objective measures of diabetes control and severity among a population-based sample of patients with diabetes. Participants included 4168 patients with diabetes from nine primary care clinics of a Health Maintenance Organization. Participants were mailed surveys aimed at measuring depression, diabetes symptoms, and other medical data. Results revealed that among 4168 patients with diabetes, those with major depression (487) reported significantly more diabetes symptoms that participants without depression after adjusting for demographic characteristics, objective measures of diabetes severity and medical comorbidity (Ludman, et al., 2004). Although the data does not allow conclusions regarding causation, depression can be viewed as an integral part of any chronic illness that is accompanied by significant physical symptoms (Ludman et al., 2004).
In a similar study, Whittemore, Melkus, and Grey (2004) examined the differences in diabetes-related health outcomes (physiological, psychosocial, and health functioning variables) with respect to depressed mood. Participants included 53 women between the ages of 30 and 70 years who were medically stable and evidenced no advanced complications in diabetes. Data were collected on demographic, physiologic, psychosocial, and health functioning variables. Forty-four percent of the women in the sample reported a depressed mood “a lot or some” of the time. The results of the study indicate that women with depressed mood demonstrated poorer psychosocial adjustment and health functioning compared to women without a depressed mood, although no differences in physiological outcomes were demonstrated. Findings of this study do corroborate the potential suffering and disability associated with depressive symptoms and a comorbid chronic illness (Whittemore, Melkus, & Grey, 2004).

In a longitudinal study, Chaney, Mullins, Wagner, Hommel, Page, and Doppler (2004) examined longitudinal relationships between causal attributions and depression symptoms in adults with rheumatoid arthritis. Participants included 34 females and 8 males, ranging in age from 25 to 75 years. They were recruited from a private outpatient rheumatology clinic and included if they had been diagnosed with RA according to American College of Rheumatology criteria. Results indicated that, whereas initial causal attributions for negative events predicted later depressive symptoms beyond the influence of perceived disability and pain, initial levels of depression were unrelated to subsequent variations in causal attributions (Chaney et al., 2004). Therefore, the results highlight the contribution of disease-unrelated cognitive appraisals to the development of depressive symptoms. Individuals in this study who endorsed a more helpless cognitive style characterized by pervasive internal attributions across a wide array of negative life events (i.e., global attributions) were at increased risk for later depressive symptoms (Chaney
et al., 2004). Indeed, the study indicates that individuals who develop a pervasive sense of personal blame for negative outcomes in the face of an unpredictable disease such as RA are more likely to experience helplessness and depression (Chaney et al., 2004).

Chaney et al. (2004) propose that, in line with a more contemporary cognitive diathesis conceptualization of depression, their findings suggest that individuals predisposed toward a more pessimistic cognitive style (independently of the disease) are at increased risk for developing symptoms of depression when the physical and psychological proximal challenges of RA are superimposed on this preexisting cognitive style. Therefore, this study indicates that individuals who demonstrate a cognitive vulnerability characterized by pessimistic appraisals across a wide array of life domains are more at risk for developing depressive symptoms than individuals with a healthier attributional style.

Thus far it is apparent that depression caused by a chronic illness may often worsen symptoms related to the illness, particularly illnesses that require high levels of daily management. Individuals with chronic illness may find themselves more prone to depression, which in turn may influence physiological, cognitive, and social functioning. In addition, researchers are also investigating the interaction of cognitive functioning, attributional style, and depression in the context of chronic illness.

In a review article, Katon and Ciechanowski (2002) discussed the impact of major depression on chronic medical illness. Katon and Ciechanowski (2002) report that major depression has been shown in multiple studies in patients with chronic medical illness to increase symptom burden, lead to additive function impairment, increase medical costs and to impair self-care and adherence. Indeed, it appears that comorbidity with depressive and chronic medical illness is often associated with increased symptom reporting and amplification of chronic
medical illness symptoms (Katon & Ciechanowski, 2002). Earlier studies also indicate that patients with both psychiatric disorders as well as subsyndromal psychological distress report significantly more physical symptoms on medical review of systems than nondistressed populations when controlling for severity of medical illness (Katon & Sullivan, 1990; Katon, Sullivan, & Walker, 2001).

Hommel, Chaney, Wagner, and McLaughlin (2002) examined the influence of psychological comorbidity on asthma-specific quality of life. More specifically, Hommel et al. (2002) investigated the extent to which anxiety and depression influence quality of life in individuals with asthma. Participants included 34 male and 30 female older adolescents and young adults between the ages of 18 and 25 with childhood onset chronic asthma. Participants completed a measure pertaining to asthma symptomatology, completed three recorded peak expiratory flow rate measurements, were administered a semi-structured illness severity assessment interview, and provided demographic information. The results of the study suggested that psychological factors (i.e., anxiety and depression) play a significant role in asthma, as they were found to be salient predictors of asthma-specific quality of life (Hommel et al., 2002). However, the researchers also found that when compared to depressive symptomatology, anxiety accounted for the majority of variance in asthma quality of life. This provides further support for psychological variables such as anxiety and depression negatively influencing quality of life and other psychosocial functioning in individuals with a chronic illness.

Chaney, Mullins, Uretsky, and Doppler (1996) examined the moderating influence of perceived daily illness control on the relationship between disease-unrelated causal attributions and DSM-IV depressive symptomatology. Participants included 47 female and 11 male patients diagnosed with RA that were seen in a private outpatient rheumatology clinic. They ranged in
age from 25 to 75 years, with a median age of 51 years. Fourteen percent of the sample met DSM-IV criteria for current major depression. Findings revealed that internal and global attributions for negative events were associated with increased levels of depression under conditions of decreased perceived illness control (Chaney et al., 1996). The findings of this study provided further support for examining general attributional style in studies of depression in RA and for cognitive-diathesis conceptualizations of adjustment to chronic illness (Chaney et al., 1996).

It is clear that individuals with chronic illnesses are at higher risk for developing a negative attributional style as well as depressive symptoms. Several studies above investigated the interaction between depression and attributional style, revealing that these individuals may also experience higher levels of psychological distress, psychosocial impairment, and to an extent, cognitive impairment. It is believed that this study will find differences between healthy individuals and individuals with chronic illnesses in the realm of attributional style, levels of depression, and amount of discounting. Individuals with chronic illnesses are not only at risk for developing more depressive symptoms and a negative attributional style, but also for engaging in more probabilistic discounting than healthy individuals.

Summary

A review of the literature indicates that, although there has been much emerging research in the field of discounting, there is a relative lack of substantial research investigating the relationship between probability discounting, attributional style, and depressive symptomatology. Further, research investigating these variables in a chronic illness population is particularly incomplete. The current research will contribute significantly to the literature in several ways: 1) alternative methods of exploring the relationship between depression and
discounting will be investigated, 2) research on the relationship between probability discounting in particular and depressive symptomatology will be supplemented, 3) discounting research will be expanded to include a chronic illness population (asthma) – a relatively unexplored field in terms of behavioral economics, 4) the correlation between depression and attributional style will be investigated in two different groups, and 5) the current research could provide insights into impulsivity and attributional style in two different groups, possibly influencing treatment interventions.
CHAPTER III

PRESENT STUDY

Research concerning the relationship between delay discounting and depression has yielded mixed findings. While Rehm and Plakosh (1975) found that individuals scoring high on measures of depression were less inclined to choose a delay over an immediate reward, other research (e.g., Burlingame, 1998; O’Hara & Rehm, 1982) were unsuccessful in finding a significant relationship between individuals’ scores on a measure of depression (Beck Depression Inventory-II – BDI-II) and the length of delay of the reward. A possible explanation for these incongruent findings is the presence of certain confounds, namely the methods used to measure levels of depression and discounting. If attributes of depression were to be approached and measured using alternative methods, such as attributional style, it is possible that a stronger relationship might be observed without requiring subjects to endorse high levels of depressive symptomatology. Abramson, Seligman, and Teasdale (1978) theorize that individuals who habitually explain bad events by internal, stable, and global causes (negative attributional style) will be more prone to depressive episodes than persons without this maladaptive attributional style.

The association between probability discounting and depression is relatively unexplored. The inclusion of a probability discounting task may aid in reflecting aspects of a negative attributional style and depression by addressing an individual’s negative beliefs and pessimistic attitude when making a choice that relies on chance. By investigating these aspects of depression
and discounting, it is expected that stronger relationships will be found. For exploratory purposes, a chronic illness group (asthma) was identified in order to investigate and compare the relationship of discounting, attributional style, and depressive symptomatology.

The purpose of the current research was to determine whether the relationship between symptoms of depression and delay discounting may be better demonstrated by using alternative measures of both. More specifically, the ASQ Composite Negative and Probability Discounting task were expected to yield a stronger relationship between depressive symptomatology and discounting compared to the Negative Affect and Negative Sadness scales of the PANAS-X and delay discounting. While not all participants may exhibit high levels of depression, attributional style has been shown to be a significant predictor of depressive symptomatology (Schoenherr et al., 1992) and may provide a better measure of an underlying pessimistic approach. In addition, the Probability Discounting task is expected to be more sensitive to a pessimistic/depressed decision-making approach. Specifically, this research aimed to:

1. Investigate the correlation between measures of delay and probability discounting within the single, combined group.

   *Hypothesis 1:* It was predicted that Delay Discounting scores and Probability Discounting scores would be significantly correlated.

2. Investigate the correlation between a measure of depressive symptomatology and a measure of attributional style within the single, combined group.

   *Hypothesis 2:* It was predicted that PANAS-X depressive symptomatology (Negative Affect; Sadness) and attributional style (CoNeg) would be significantly correlated.

3. Evaluate the relationship between measures of discounting and depressive symptomatology within the single, combined group. More specifically, correlations between a) PANAS-X
depressive symptomatology (Negative Affect; Sadness) and delay discounting; b) attributional style (CoNeg) and delay discounting; c) PANAS-X depressive symptomatology (Negative Affect; Sadness) and probability discounting; and d) attributional style (CoNeg) and probability discounting were evaluated within the single, combined group.

Hypothesis 3a: It was predicted that the effect size between attributional style (CoNeg) and probability discounting would be significantly larger than the effect size between symptoms of depression (Negative Affect; Sadness) and Delay Discounting.

Hypothesis 3b: It was predicted that symptoms of depression and negative attributional style would predict Delay and Probability discounting scores.

The present study conducted exploratory analyses with a chronic illness population (college students with asthma) to:

4. Determine whether or not the chronic illness group asthma differs to the healthy group in terms of depressive symptomatology, attributional style, delay discounting, and probability discounting.

Hypothesis 4a: It was predicted that there would be higher levels of reported depressive symptomatology and negative attributional style in the chronic illness group.

Hypothesis 4b: It was further predicted that there would be significantly higher delay discounting and probability discounting in the chronic illness group than in the healthy group.

Hypothesis 4c: It was predicted that differential correlational relationships would exist between NegAffect, NegSadness, ASQ CoNeg, and Delay and Probability discounting within the two groups (asthma versus healthy). More specifically, it was predicted that there would be stronger correlations between variables within the chronic illness group than there would
be within the healthy group. Literature has demonstrated generally higher levels of depressive symptomatology in a chronic illness population as well as certain attributional differences between individuals with a chronic illness and healthy individuals. This may more clearly demonstrate the link between the variables in question.

5. Determine whether or not there are higher rates of discounting, depressive symptomatology, and a more pessimistic attributional style in individuals endorsing moderate to high levels of asthma severity compared to individuals endorsing mild severity of asthma.

*Hypothesis 5:* It was predicted that there would be higher levels of these variables within the group of individuals endorsing moderate to high levels of asthma severity compared to the group of individuals endorsing mild severity of asthma. Literature indicates that individuals endorsing more severe asthma are at an increased risk for general psychological distress, depression, and anxiety (McQuaid, Kopel, & Nassau, 2001; Van Pelt, 2004; Vila, Nollet-Clemenccon, deBlic, Mouren-Simeoni, & Scheinmann, 1998).
CHAPTER IV

METHOD

Participants

Participants (N = 507; 115 males, 357 females, 35 unreported) were undergraduate students attending Oklahoma State University, ranging in age from 18 to 41 years (M = 20.1). Of these students, 59 were identified to have asthma (47 participants endorsed mild symptoms of asthma and 12 participants endorsed moderate-to-severe symptoms of asthma). See Table 1 for more detailed demographic information regarding the asthma, non-asthma, and combined groups. To be eligible to participate, participants had to be at least 18 years of age. The combined sample reported their ethnicity as 81.3% Caucasian, 5.5% Native American, 4.7% African American, 2.6% Asian American, .6% Hispanic/Latino, 2.8% Other, and 2.6% did not report race or ethnicity.

Participants were recruited through the Oklahoma State University Psychology department’s research subject pool, which is managed using Experimetrix internet-based software. Participants were required to register for each experiment through Experimetrix, thereby preventing them from participating in the same study multiple times. To further control duplicated data, participants’ IP addresses and month and date of birth were recorded, allowing the researcher to delete these cases. Participants were informed that the purpose of the study was to examine the relationship between attributional style and other variables, such as decision-making skills.
Procedure

Participants were given the URL address (http://fp.okstate.edu/collinslab/leo/) and requested to follow the address to the study’s homepage. After reading the introduction, participants clicked the “submit” button where they were taken to a consent page, informing them of the nature of the study. To ensure confidentiality, after clicking the “submit” button, the participant’s IP address was securely transferred into our research database. Other identifying information that was automatically included was the time and date of completion. Upon completion of data collection, the URL address (http://fp.okstate.edu/collinslab/leo/) displayed a message thanking the students for their participation and informing them that the study has completed its data collection and was closed.

Measures

Background Information Questionnaire. The Background Information Questionnaire is designed to gather information regarding the participant’s gender, age, and ethnic origin. This questionnaire also provided the participants with a question aimed at identifying chronic illnesses. In addition, participants with asthma were asked to report their age of asthma diagnosis, type of asthma (seasonal versus perennial), current treatment status, and provide ratings of asthma severity and controllability.

Positive and Negative Affect Schedule – Expanded Form (PANAS – X). The PANAS-X (Watson & Clark, 1994) is a 60-item, expanded version of the PANAS. There are two higher order scales – Positive and Negative affect. The Negative affect scale contains the following terms: afraid, scared, nervous, jittery, irritable, hostile, guilty, ashamed, upset, and distressed. In addition to the two higher order scales (Positive and Negative affect), the PANAS-X measures 5 basic negative emotions: Fear, Hostility, Guilt, and Sadness; 3 basic positive emotions: Joviality, Self-Assurance,
and Attentiveness; and 4 other affective states: Shyness, Fatigue, Serenity, and Surprise. More specifically, the Negative Sadness scale contains the following terms: sad, blue, downhearted, alone, and lonely. The PANAS-X consists of a number of words and statements that describe different feelings and emotions. The respondent selects one of 5 descriptive words (1 – very slightly or not at all; 2 – a little; 3 – moderately; 4 – quite a bit; 5 – extremely) that best describes that feeling.

Regarding internal consistency, the alpha reliabilities for both higher order scales are high, generally ranging from .83 to .90 for Positive Affect, and from .85 to .90 for Negative Affect (Watson & Clark, 1994). Furthermore, scores on the PANAS-X Negative Sadness scale had a correlation of .59 with the Beck Depression Inventory (BDI; Beck, Ward, Mendelson, Mock, & Erbaugh, 1961) and the Negative Affect scale showed a correlation of .66 with the BDI-II (Tafarodi, Marshall, & Milne, 2003). Internal consistency was calculated for the PANAS-X Negative Affect and Negative Sadness scales. The PANAS-X Negative Affect Scale revealed a Cronbach’s α of .86 and the Negative Sadness Scale revealed a Cronbach’s α of .83, with both scales demonstrating very good internal consistency. For the purposes of this study, the Negative Affect and Negative Sadness scores were used to measure depressive symptomatology.

Attributional Style Questionnaire (ASQ). The ASQ (Peterson, Semmel, von Baeyer, Abramson, Metalsky, & Seligman, 1982) is the most widely known global measure developed to test predictions from Abramson’s reformulated theory of learned helplessness depression (Fernandez-Ballesteros, Diez-Nicolas, Caprara, Barbaranelli, & Bandura, 2002). It is a 48-item questionnaire containing 12 hypothetical events, half describing positive events (e.g., “you meet a friend who compliments you on your appearance”) and half describing negative events (e.g., “you go out on a date and it goes badly”). The respondent is instructed to read each event and rate the cause of the event. Using seven-point Likert scales, the perceived cause of each event is rated along the
dimensions of locus (1 = totally due to other people or circumstance; 7 = totally due to me), stability (1 = will never again be present; 7 = will always be present), and globality (1 = influences just this particular situation; 7 = influences all situations in my life). It provides three composite attributional style scale scores that measure the attributional style for negative events (CoNeg), the participants’ attributional style for positive events (CoPos), and the difference between the participants’ attributional style for positive events and negative events (CPCN).

For the purposes of the current study, low CoNeg scores were interpreted as a more optimistic attributional style for negative events and high CoNeg scores as a more pessimistic attributional style for negative events. The composite scores have consistently demonstrated acceptable internal consistency ranging from .71 to .75 (Peterson et al., 1982; Welter, 2002).

As the ASQ is frequently used in both research and practice to predict symptomatology, it is important to have some form of theoretical justification for the measurement of internality, stability, and globality dimensions of attributional style (Hewitt, Foxcroft, & MacDonald, 2004). Because attributions for positive events are not central to learned helplessness theory (Peterson, 1993; Chaney, Mullins, Wagner, Hommel, Page, & Doppler, 2004), only attributions for negative events were examined in the current study, the rationale being that they are more likely to be associated with depression than the positive events (Peterson & Seligman, 1984). The negative items of the ASQ have been found to have moderate internal consistency (Peterson et al., 1982; Seligman, Abramson, Semmel, & von Baeyer, 1979), test-retest reliability (Golin, Sweeney, & Shaeffer, 1981) and construct validity (Schulman, Castellon, & Seligman, 1989). Internal consistency was calculated for the Attributional Style Questionnaire Composite Negative Scale (ASQ CoNeg). The ASQ CoNeg revealed a Cronbach’s α of .74, demonstrating a moderate level of internal consistency.
**Delay Discounting.** A monetary-choice questionnaire developed by Kirby, Petry, and Bickel (1999) was used to measure delay discounting. Participants were presented with a fixed set of 27 choices between smaller, immediate hypothetical rewards and larger, delayed hypothetical rewards (Kirby et al., 1999). There are 9 discounting rates ranging from a k-value of 0.00016 to a k-value of 0.25, with three choices associated with each of the 9 k-values. Therefore the total number of items for this task is 27. The indifference points between each of the choices correspond to one of the 9 k-values. For example, one item is a choice between “$25 today” and “$30 in 80 days.” If the participant chose the immediate reward of “$25 today,” it was assumed that s/he had a discounting rate greater than 0.0025 (k = 0.0025). However, if the participant chose the delayed reward of “$30 in 80 days,” it was assumed that s/he had a discounting rate less than 0.0025. Another choice utilizing the same k-value of 0.0025 is “$69 today,” or “$85 in 91 days,” where the difference between the immediate and delayed reward is mathematically equivalent to a k-value of 0.0025. Based on participants’ choices of the immediate or delayed reward across trials, the scoring procedure involved each participant being assigned a k-value corresponding to the geometric midpoint of one of the eight ranges or one of the two endpoint values.

**Probability Discounting.** A modified version of a probability discounting task created by Richards, Zhang, Mitchell, and de Wit (1999) was included in this study. Although mathematically equivalent in nature, the probability discounting task uses different indifference points. In other words, the k-values used in this task will differ from the delay discounting task. The same k-values used by Richards et al. (1999) in their probability discounting task were incorporated into the current task. Indifference points for three different probabilities were determined, namely 25%, 50%, and 75%, as opposed to Richards et al. (1999) task which determined indifference points for 25%, 50%, 75%, 90%, and 100%. There are 9 discounting rates ranging from a k-value of 0.20155 to a k-value
of 12, with three choices associated with each of the 9 k-values. Therefore the total number of items for this task is 27. Individuals will be given a choice between, for example, “$33.20 for sure,” and “$60 with a 25% chance.” The corresponding k-value for this item is 0.20155. Accordingly, another item with the same k-value would be presented as: “$27.60 for sure,” or “$35 with a 75% chance.”
CHAPTER V

RESULTS

Preliminary Analyses

Correlation Matrices. See Table 2 for a comprehensive correlative matrix of interest to the present study. Analyses of the results from the PANAS-X indicated that the current sample’s NegAffect mean and standard deviation \((M = 15.4; SD = 6.4)\) and NegSadness mean and standard deviation \((M = 9.1; SD = 4.4)\) were not significantly different from the NegAffect mean and standard deviation \((M = 16.4; SD = 4.1)\) and NegSadness mean and standard deviation \((M = 9.4; SD = 4.4)\) that was seen in the college sample used when designing the measure. When using Likert-type scales it is imperative to calculate and report Cronbach’s alpha coefficient for internal consistency reliability for any scales or subscales one may be using (Gliem & Gliem, 2003).

Primary Analyses

Hypothesis 1: Within the single, combined group Pearson correlations indicated that the correlation of Delay Discounting scores \((M = .026; SD = .034; N = 396)\) and Probability Discounting scores \((M = 1.346; SD = 1.403; N = 319)\) were significantly correlated \([r(269) = .202, p = .001]\). Hypothesis 1 was supported.

Hypothesis 2: Within the single, combined group Pearson correlations indicated that the correlation of Negative Affect \((M = 15.4; SD = 6.4; N = 507)\) and ASQ CoNeg \((M = 71.7; SD = 12.5; N = 507)\) was significant \([r(505) = .109, p = .014]\), the correlation of Negative Affect and Negative Sadness \((M = 9.1; SD = 4.4; N = 507)\) was significant \([r(505) = .616, p = .001]\), and the
correlation of Negative Sadness and ASQ CoNeg was significant \([r(505) = .124, p = .005]\).

Hypothesis 2 was supported.

*Hypothesis 3a:* Within the single, combined group Pearson correlations did not reveal any significant correlation between ASQ CoNeg and Probability discounting scores \([r(317) = -.059, p = .296]\). Further, no significant correlations were found between NegAffect and Delay discounting \([r(394) = -.033, p = .518]\) or NegSadness and Delay discounting scores \([r(394) = -.083, p = .099]\). In addition, correlation coefficients were transformed using Fisher’s \(r\)-to-\(z\) transformation. Null hypothesis testing for the difference between the two correlations revealed that there was no significant difference between the ASQ CoNeg/Probability discounting correlation and the NegAffect/Delay discounting correlation \((z = 1.217, p > .05)\) or the ASQ CoNeg/Probability discounting correlation and the NegSadness/Delay discounting correlation \((z = 0.317, p > .05)\). To obtain significance at the .05 level and .01 levels, the absolute \(z\) needs to exceed 1.96 and 2.58 respectively. The results indicate that Hypothesis 3a was not supported in that the effect size for the relation between attributional style (CoNeg) and Probability discounting was not larger than the effect size for the relation between symptoms of depression (Negative Affect; Sadness) and Delay discounting.

*Hypothesis 3b:* A hierarchical regression analysis was conducted to assess whether Negative Affect would predict Delay discounting. Delay discounting was not significantly predicted by Negative Affect alone \([F(1,394) = .419, p = .52, R^2 = .001]\) with Negative Affect alone accounting for only 0.1% of the variance in Delay discounting, or by Negative Sadness alone \([F(1,394) = 2.731, p = .09, R^2 = .007]\) with Negative Sadness alone accounting for only 0.7% of the variance in Delay discounting. However, it appeared that there was an additive effect with these two variables. Placing both Negative Affect \(B = .001 (.0001), t(393) = 2.155, p = .032\) and Negative
Sadness [$B = -.001 (.001), t(393) = -2.641, p = .009$] into the model, resulted in 1.8% of the variance being accounted for in Delay discounting and this overall model was significant [$F(2,393) = 3.70, p = .026$].

Hierarchical regression analyses were conducted to assess whether Negative Affect would predict Probability discounting. Probability discounting was not significantly predicted by Negative Affect alone [$F(1,317) = .147, p = .70, R^2 = .0001$] with Negative Affect alone accounting for an insubstantial part of the variance in Probability Discounting, or by Negative Sadness alone [$F(1,317) = .155, p = .69, R^2 = .0001$], with Negative Sadness alone also accounting for an insubstantial part of the variance in Probability Discounting. There was no additive effect with these two variables. Placing both Negative Affect [$B = .003 (.016), t(316) = .173, p = .863$] and Negative Sadness [$B = .005 (.024), t(316) = .194, p = .846$] into the model, resulted in 0.1% of the variance being accounted for in Probability Discounting and this overall model was not significant [$F(2,316) = .092, p = .912$].

Hierarchical regression analyses were conducted to assess whether ASQ CoNeg would predict Delay and Probability discounting. Delay discounting was not significantly predicted by ASQ CoNeg [$F(1,394) = .168, p = .68, R^2 = .0001$]. Probability Discounting was not significantly predicted by ASQ CoNeg [$F(1,317) = 1.1, p = .296, R^2 = .003$], with ASQ CoNeg accounting for 0.3% of the variance in Probability Discounting.

**Exploratory Analyses:**

**Hypotheses 4a and 4b:** A one-way analysis of variance (ANOVA) was conducted to examine mean levels of NegAffect (asthma group: $M = 15.1, SD = 6.0$; non-asthma group: $M = 15.5, SD = 6.5$), NegSadness (asthma group: $M = 8.8, SD = 4.2$; non-asthma group: $M = 9.1, SD = 4.4$), ASQ CoNeg (asthma group: $M = 70.1, SD = 16.2$; non-asthma group: $M = 72, SD = 12$), Delay
discounting (asthma group: $M = .017, SD = .021$; non-asthma group: $M = .027, SD = .035$), and
Probability discounting (asthma group: $M = 1.420, SD = 1.546$; non-asthma group: $M = 1.331, SD = 1.389$) between college students with asthma and college students without asthma (refer to Table 5).

The analyses revealed that the two groups did not differ significantly on the NegAffect score $[F(1,500) = .201, p = .654]$, the NegSadness score $[F(1,500) = .182, p = .670]$, the ASQ CoNeg score $[F(1,500) = 1.079, p = .299]$, the Delay discounting score $[F(1,390) = 3.387, p = .066]$, or the Probability discounting score $[F(1,313) = .136, p = .713]$. Hypothesis 4a and 4b were not supported in that there were not higher levels of reported depressive symptomatology, negative attributional style, delay discounting, or probability discounting.

**Hypothesis 4c:** Within the Asthma group ($N = 59$), Pearson correlations indicated that Negative Affect was significantly correlated with Negative Sadness $[r = .610, p = .001]$. No other correlations were significant (see Table 4). Hypothesis 4c was not supported in that there were not stronger correlations between variables within the chronic illness group than the healthy group ($N = 443$).

Correlation coefficients for both groups (healthy versus asthma) were transformed using Fisher’s $r$-to-$z$ transformations. Null hypothesis testing for the difference between the two correlations revealed that there was no significant difference between Delay and Probability discounting ($z = .326, p > .05$), Negative Affect and ASQ CoNeg ($z = 1.027, p > .05$), or Negative Sadness and ASQ CoNeg ($z = 1.356, p > .05$) for the two groups.

**Hypothesis 5:** Finally, a one-way analysis of variance (ANOVA) was conducted to examine mean levels of NegAffect (mild: $M = 14.5, SD = 5.9$; mod-to-severe: $M = 17.2, SD = 5.8$), NegSadness (mild: $M = 8.5, SD = 4.3$; mod-to-severe: $M = 10.0, SD = 4.0$), ASQ CoNeg (mild: $M = 70.0, SD = 17.4$; mod-to-severe: $M = 70.8, SD = 11.0$), Delay discounting (mild: $M = .014, SD =
.019; mod-to-severe: $M = .024, SD = .025$), and Probability discounting (mild: $M = 1.444, SD = 1.676$; mod-to-severe: $M = 1.341, SD = 1.075$) between college students endorsing mild severity of asthma (n = 47) and college students endorsing moderate-to-severe severity of asthma (n = 12). The analyses (refer to Table 6) revealed that the two groups did not differ significantly on the NegAffect score ($F(1,57) = 1.952, p = .168$), the NegSadness score ($F(1,57) = 1.197, p = .279$), the ASQ CoNeg score ($F(1,57) = .027, p = .869$), the Delay discounting score ($F(1,45) = 2.001, p = .164$), or the Probability discounting score ($F(1,37) = .029, p = .865$).
CHAPTER VI
DISCUSSION

The purpose of this study was to investigate the relationship between Delay discounting, Probability discounting, depressive symptomatology, and attributional style in college students. Previous research has shown inconsistent correlations between Delay discounting tasks and measures of depression. While Rehm and Plakosh (1975) found a significant correlation between depressive symptomatology and higher rates of delay discounting, other researchers (e.g., Burlingame, 1998; O’Hara & Rehm, 1982) were unable to replicate those findings. The Probability discounting task and Attributional Style Questionnaire were introduced as possible alternative measures to better demonstrate the relationship between discounting and depressive symptomatology respectively. Further, for exploratory purposes, the study sought to explore differences in discounting, attributional style, and depressive symptomatology between healthy college students and college students identified to have asthma.

Primary analyses were conducted within the single, combined group. First, the correlation between the Delay and Probability discounting scores was examined. Specifically, it was predicted that the two tasks would be correlated with each other. The analysis revealed that Delay and Probability discounting scores were indeed significantly correlated. This further supports the research indicating that in general, delay and probability discounting tasks are correlated with one another (Mitchell, 1999; Richards et al., 1999). However, it should be noted that research has also
demonstrated that these tasks may tap into different constructs (e.g., Reynolds, Karraker, Horn, & Richards, 2003), which may influence their sensitivity in measuring the same variables.

Second, the correlation between depressive symptomatology as measured by the PANAS-X (Negative Affect and Negative Sadness) and the Composite Negative (ASQ CoNeg) score on the Attributional Style Questionnaire were investigated. Results of the analysis revealed that the second hypothesis was supported in that the Negative Affect and Negative Sadness scales of the PANAS-X were significantly correlated with the Composite Negative score on the Attributional Style Questionnaire. These results are consistent with research demonstrating the relationship between attributional style and depressive symptomatology (e.g., Alloy, Peterson, Abramson, & Seligman, 1984; Peterson & Seligman, 1984). This evidence further supports the reformulated helplessness theory (Abramson et al., 1978) which predicted that individuals who habitually explain bad events by internal, stable, and global causes will be more prone to depressive episodes than persons without this maladaptive explanatory style. Put simply, individuals with a negative attributional style (CoNeg) are more likely to experience symptoms of depression (NegAffect and NegSadness).

Third, effect sizes between attributional style, probability discounting, depressive symptomatology, and delay discounting were examined. Specifically, it was predicted that the effect size between attributional style (CoNeg) and Probability discounting would be significantly larger than the effect size between depressive symptomatology (Negative Affect; Negative Sadness) and Delay discounting. Analyses revealed no significant correlations between ASQ CoNeg and Probability discounting, NegAffect and Delay discounting, or NegSadness and Delay discounting. In addition, no significant differences in effect sizes were noted. Although hypothesis 3a was not supported, these results mimic past research in that significant correlations between delay
discounting and depressive symptomatology were found inconsistently (e.g., Burlingame, 1998; O’Hara & Rehm, 1982; Rehm & Plakosh, 1975). It is likely that, due to the nature of the current sample (non-clinical, college students), levels of depressive symptomatology were not high enough to demonstrate a substantial relationship with delay discounting. Another possible confound includes the measure used in the current study to assess symptoms of depression (PANAS-X). While the PANAS-X Negative Sadness and Negative Affect scales have demonstrated a correlation of .59 and .66 with the BDI and BDI-II respectively, this broadband measure may not have been sensitive enough to more accurately assess for symptoms of depression. Future studies investigating the relationship between delay discounting and depression should consider the influence of level of depressive symptomatology on this relationship. In addition, alternate measures of depression may be more sensitive to depressive symptomatology.

The relationship between probability discounting and attributional style is relatively unexplored. Theory and research have demonstrated significant relationships between delay and probability discounting, between depression and attributional style, and between delay discounting and depression. Theoretically, a relationship between probability discounting and attributional style should be seen. It is possible that the null findings are due to the nature of the sample (non-clinical) influencing the levels of negative attributional style, thereby preventing significant relationships from being found. Future studies exploring these variables may need to obtain data from a clinically significant sample to better demonstrate the strength of the hypothesized relationships. In addition, not only has the literature shown that researchers often have difficulty replicating a study demonstrating the link between depression and delay discounting, but there is also inconsistency in the relationship between delay and probability discounting, suggesting that these discounting tasks may measure differing constructs. Recent research (Reynolds & Schiffbauer, 2004) has suggested
that there may be more sensitive measures of discounting requiring that participants experience choice consequences *during* the measurement period. The experiential discounting task developed by Reynolds and Schiffbauer (2004) did indeed demonstrate more sensitivity to state changes in participants, such as sleep deprivation. Future research may wish to consider employing experiential, in-lab data collection to increase measurement sensitivity of discounting.

Next, the extent to which symptoms of depression and negative attributional style can predict Delay and Probability discounting scores was investigated. Analyses revealed that Delay discounting was significantly predicted by Negative Affect and Negative Sadness when these variables were both placed in a regression, although neither Negative Affect nor Negative Sadness alone significantly predicted Delay discounting. Hypothesis 3b was therefore partly supported in that Delay discounting was significantly predicted by Negative Affect and Negative Sadness. These results further support the theory and research demonstrating how Delay discounting can be significantly predicted by depressive symptomatology (Ferster, 1973; Lewinsohn, 1974; Rehm & Plakosh, 1975). In fact, Ainslie (2005) suggested that individuals with depression appear to demonstrate hyperbolic discounting, temporarily preferring options that pay off quickly to richer, but slower-paying alternatives. While the finding concerning the extent to which Delay discounting is predicted by depressive symptomatology is statistically significant, it does not emerge as clinically significant. As mentioned earlier, it is possible that more significant results may have been seen from a population that was more severely depressed. Future research will need to consider obtaining data from a population with severe depression to better demonstrate this relationship. It should be noted that the Negative Affect scale includes descriptors of general psychological distress (e.g., afraid, scared, nervous, jittery, irritable) as opposed to the more depression-specific descriptors in the Negative Sadness scale (sad, blue, downhearted, alone, and lonely). Future research may wish to
obtain data not only of depressive symptomatology but of general psychological distress in particular to determine the extent to which Delay discounting can be predicted by these variables.

Regarding the null finding that attributional style did not significantly predict Delay discounting, it was mentioned earlier that, while the theory regarding these relationships appears sound, the actual instruments measuring the construct of Delay discounting may not be sensitive enough to obtain significant results. Future research would do well not only to employ more sensitive measures of discounting in an in-lab setting, but should consider manipulating levels of attributional style during the data collection. In other words, exercises designed to alter participants’ attributional style before completing a discounting task (with the obvious use of control groups) may yield significant differences. Further, employing an alternate measure of attributional style may result in more sensitivity to the construct. Peterson and Seligman (1984) argued that the reformulated model of learned helplessness is more likely to be supported when a large number of events are employed. In fact, improvements on attributional style measures (an increase in internal consistency) have been shown by increasing the number of items from 6 bad events to 24 (e.g., Peterson & Villanova, 1988) and by selecting items that fit the target population (Anderson & Deuser, 1991).

While Delay discounting was significantly predicted by the combination of Negative Affect and Negative Sadness, Probability discounting was not predicted by Negative Affect, Negative Sadness, a combination of Negative Affect and Negative Sadness, or ASQ CoNeg. The fact that Delay and Probability discounting were significantly correlated but Probability discounting was not significantly predicted by depressive symptomatology provides further support for the theorizing that these discounting tasks, while similar in several respects, may indeed assess different constructs to varying degrees (e.g., Mitchell, 1999; Reynolds, Karraker, Horn, & Richards, 2003).
The in-lab experiential discounting task (EDT) described earlier contains a combination of temporal and probabilistic choices. It is possible that by providing consequence feedback to participants while completing the task, researchers may obtain more substantial moment-to-moment intra-individual variability in discounting (Reynolds & Shiffbauer, 2004). Previous research has also shown that discounting tasks containing more items (e.g., Hinson, Jameson, & Whitney, 2003) may be more likely to find larger effect sizes. The delay and probability discounting tasks in this study contained 27 items each, as opposed to the 80-item delay discounting measure employed by Hinson et al. (2003) in a study examining memory, impulsiveness, and discounting. Strong relationships between measures of impulsiveness, working memory load, and discounting of delayed rewards were found. Future research should consider employing discounting tasks with more items so as to increase the sensitivity of the measure.

For exploratory purposes, the single group was divided into two groups: individuals with asthma and healthy individuals. Research indicates that individuals with a chronic illness are at a higher risk for psychological distress (McQuaid, Kopel, & Nassau, 2001; Silverglade et al., 1994; Vila, Nollet-Clemecon, deBlic, Mouren-Simeoni, & Scheinmann, 1998). In particular, individuals with asthma tend to have a cognitive style that includes an expectation of negative outcomes for events (Mullins, Chaney, Pace, & Hartman, 1997). Also, Chaney et al. (2004) found that individuals who endorsed a more helpless cognitive style characterized by pervasive internal attributions were at increased risk for later depressive symptoms. Although the literature clearly indicates these relationships, the current study did not find that the two groups differed significantly on any of the variables. This could be explained by several reasons. As mentioned earlier, given the limited sample size of the asthma group, the power of the study may have diminished. Also, asthma severity was in general, not very high. Research (e.g., Van Pelt, 2004; Hommel et al., 2002) indicates that
college students with asthma, in particular those with moderate to severe asthma, evidence higher rates of general psychological distress, depression, and anxiety compared to healthy same-age and gender peers. It is therefore likely that significant differences may have been seen in this study if the asthma group comprised individuals with more severe asthma. Research examining discounting processes and attributional style in a chronic illness group should include participants that are significantly impaired if any significant differences between a healthy group and a chronic illness group are to be found.

Further analyses indicated that there were fewer significant correlations between variables within the asthma group compared to the healthy group. Again, to a certain degree this can be explained by the limited sample size of the asthma group. Future research may need to include larger samples so as to increase power and ensure the detection of any differences.

*Strengths and Limitations*

Several strengths of the current study are notable. First, the significant predictability of delay discounting by depressive symptomatology in this study further supports the behavior economics research findings. Second, to date, there has been limited research conducted to document the relationship between probability discounting and attributional style. Thus, the study represents a relatively unique effort to assess whether attributional style does, to an extent, predict probability discounting. Third, the exploratory analyses further represent an effort to expand the literature investigating discounting in a chronic illness population. Fourth, the online nature of the study enabled the researcher to obtain data from a larger sample. Regarding validity of responses, studies have demonstrated that individuals completing information online are typically more willing to provide sensitive information as compared to in-lab paper and pencil tasks (e.g., Elford et al., 2004;
Hirshfield, Remien, & Humberstone, 2004; Newman et al., 2002). In addition, only discounting data from consistent responders was used in an effort to increase validity.

Several important limitations are acknowledged in the current study. First, the sample used was a college sample that did not demonstrate clinical levels of depression or negative attributional style, thereby most likely reducing the likelihood of significant findings. Second, in the interest of efficiency, the measures used to calculate discounting, depression, and attributional style in this study did not contain as many items as other longer and possibly more sensitive measures. Third, data collected online, while having its advantages, prevents the manipulation of variables of such as discounting and attributional style in the laboratory using the EDT or a task aimed at increasing internalizing respectively. Fourth, within the context of statistical analyses, it is possible that significant differences between the healthy group and asthma group were not detected due to the relatively small sample size of the asthma group. The sample size may have resulted in a larger standard error and weaker power. Although the relatively low incidence of asthma makes it difficult to obtain large sample sizes within the general population, future research may need to include larger samples so as to increase power and ensure the detection of any differences. Finally, asthma severity was generally in the mild range in the sample and may not have been severe enough for individuals to demonstrate higher levels of depressive symptomatology and negative attributional style.

Future Directions

Future studies should consider employing more sensitive measures of discounting, such as the in-lab experiential discounting task (EDT). Also, expanded measures of discounting and attributional style are suggested to increase sensitivity to the constructs, such as the 80-item discounting task and the Expanded Attributional Style Questionnaire (Peterson & Villanova, 1988). The fact that Delay discounting was predicted by depressive symptomatology and Probability
discounting was not indicates that more research needs to be conducted to investigate how Delay and Probability discounting tasks differ and what constructs are actually measured. Research with a chronic illness population should ensure that the chronic illness group is sufficiently large and has a significant degree of impairment related to the chronic illness. Other chronic illnesses should be explored to expand the literature investigating behavior economics in a chronic illness population and possibly guide interventions with such a population such as treatment adherence.

Conclusions

In sum, this study partially supported the literature concerning the relationship between depressive symptomatology and delay discounting in that depressive symptomatology significantly predicted rates of delay discounting. In addition, this study obtained similar results to research findings indicating that delay and probability discounting, while similar in many regards, may indeed measure different constructs. No significant differences between the healthy group compared to the asthma group emerged.
VIII. References.


Gliem, J.A. & Gliem, R.R. (2003, October). *Calculating, interpreting, and reporting Cronbach’s Alpha Reliability Coefficient for Likert-type scales*. Paper presented at the meeting of the Midwest Research to Practice Conference in Adult, Continuing, and Community Education, Columbus, OH.


psychiatric disorders in the United States: Results from the National Comorbidity Survey. 

*Archives of General Psychiatry, 51*, 8-19.


APPENDIX A

Tables
Table 1. Participant Characteristics.

<table>
<thead>
<tr>
<th></th>
<th>Combined Group</th>
<th>Healthy Group</th>
<th>Asthma Group</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Gender</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>115 (22.7%)</td>
<td>101 (22.8%)</td>
<td>12 (20.3%)</td>
</tr>
<tr>
<td>Female</td>
<td>357 (70.4%)</td>
<td>309 (69.8%)</td>
<td>46 (78%)</td>
</tr>
<tr>
<td>No response</td>
<td>35 (6.9%)</td>
<td>33 (7.4%)</td>
<td>1 (1.7%)</td>
</tr>
<tr>
<td><strong>Age</strong></td>
<td>20.1 (2.8)</td>
<td>19.9 (2.5)</td>
<td>20.7 (3.4)</td>
</tr>
<tr>
<td><strong>Ethnicity</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>African American</td>
<td>24 (4.7%)</td>
<td>21 (4.7%)</td>
<td>3 (5.1%)</td>
</tr>
<tr>
<td>American Indian</td>
<td>28 (5.5%)</td>
<td>25 (5.6%)</td>
<td>2 (3.4%)</td>
</tr>
<tr>
<td>Asian American</td>
<td>13 (2.6%)</td>
<td>13 (2.9%)</td>
<td>0 (0%)</td>
</tr>
<tr>
<td>Caucasian</td>
<td>412 (81.3%)</td>
<td>356 (80.4%)</td>
<td>52 (88.1%)</td>
</tr>
<tr>
<td>Hispanic/Latino</td>
<td>3 (0.6%)</td>
<td>2 (0.5%)</td>
<td>1 (1.7%)</td>
</tr>
<tr>
<td>Other</td>
<td>14 (2.8%)</td>
<td>13 (2.9%)</td>
<td>1 (1.7%)</td>
</tr>
<tr>
<td>No Response</td>
<td>13 (2.6%)</td>
<td>13 (2.9%)</td>
<td>0 (0%)</td>
</tr>
</tbody>
</table>

* a mean (standard deviation) in years.
Table 2. Correlation matrix of delay discounting, probability discounting, attributional style CoNeg, negative affect, and negative sadness within the single combined group.

<table>
<thead>
<tr>
<th></th>
<th>DD</th>
<th>PD</th>
<th>ASQ CoNeg</th>
<th>NegAffect</th>
<th>NegSadness</th>
</tr>
</thead>
<tbody>
<tr>
<td>DD</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PD</td>
<td></td>
<td>0.202**</td>
<td>1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>ASQ CoNeg</td>
<td>-0.021</td>
<td>-0.059</td>
<td>1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>NegAffect</td>
<td>0.033</td>
<td>0.022</td>
<td>0.109*</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>NegSadness</td>
<td>-0.083</td>
<td>0.022</td>
<td>0.124*</td>
<td>0.616**</td>
<td>1</td>
</tr>
</tbody>
</table>

Note: * Correlation is significant at the 0.05 level (2-tailed). ** Correlation is significant at the 0.01 level (2-tailed). DD = Delay Discounting; PD = Probability Discounting; ASQ CoNeg = Attributional Style Questionnaire Composite Negative; NegAffect = PANAS-X Negative Affect Scale; NegSadness = PANAS-X Negative Sadness Scale.
Table 3. Correlation matrix of delay discounting, probability discounting, attributional style CoNeg, negative affect, and negative sadness within the non-asthma group.

<table>
<thead>
<tr>
<th></th>
<th>DD</th>
<th>PD</th>
<th>ASQ CoNeg</th>
<th>NegAffect</th>
<th>NegSadness</th>
</tr>
</thead>
<tbody>
<tr>
<td>DD</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PD</td>
<td>.220**</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ASQ CoNeg</td>
<td>-.033</td>
<td>-.115</td>
<td>1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>NegAffect</td>
<td>.036</td>
<td>.000</td>
<td>.132**</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>NegSadness</td>
<td>-.080</td>
<td>.039</td>
<td>.160**</td>
<td>.616**</td>
<td>1</td>
</tr>
</tbody>
</table>

Note: * Correlation is significant at the 0.05 level (2-tailed). ** Correlation is significant at the 0.01 level (2-tailed). DD = Delay Discounting; PD = Probability Discounting; ASQ CoNeg = Attributional Style Questionnaire Composite Negative; NegAffect = PANAS-X Negative Affect Scale; NegSadness = PANAS-X Negative Sadness Scale.
Table 4. Correlation matrix of delay discounting, probability discounting, attributional style CoNeg, negative affect, and negative sadness within the asthma group.

<table>
<thead>
<tr>
<th></th>
<th>DD</th>
<th>PD</th>
<th>ASQ CoNeg</th>
<th>NegAffect</th>
<th>NegSadness</th>
</tr>
</thead>
<tbody>
<tr>
<td>DD</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PD</td>
<td>.159</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ASQ CoNeg</td>
<td>-.038</td>
<td>.225</td>
<td>1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>NegAffect</td>
<td>.056</td>
<td>.152</td>
<td>-.013</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>NegSadness</td>
<td>-.032</td>
<td>-.107</td>
<td>-.031</td>
<td>.610**</td>
<td>1</td>
</tr>
</tbody>
</table>

Note: * Correlation is significant at the 0.05 level (2-tailed). ** Correlation is significant at the 0.01 level (2-tailed). DD = Delay Discounting; PD = Probability Discounting; ASQ CoNeg = Attributional Style Questionnaire Composite Negative; NegAffect = PANAS-X Negative Affect Scale; NegSadness = PANAS-X Negative Sadness Scale.
Table 5. One-way ANOVA: Non-Asthma group vs. Asthma group.

<table>
<thead>
<tr>
<th>Source</th>
<th>SS</th>
<th>df</th>
<th>MS</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>DD</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Between Groups</td>
<td>.004</td>
<td>1</td>
<td>.004</td>
<td>.066</td>
</tr>
<tr>
<td>Within Groups</td>
<td>.461</td>
<td>390</td>
<td>.001</td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>.465</td>
<td>391</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>PD</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Between Groups</td>
<td>.269</td>
<td>1</td>
<td>.269</td>
<td>.713</td>
</tr>
<tr>
<td>Within Groups</td>
<td>621.524</td>
<td>313</td>
<td>1.986</td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>621.793</td>
<td>314</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>ASQ CoNEG</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Between Groups</td>
<td>170.616</td>
<td>1</td>
<td>170.616</td>
<td>.299</td>
</tr>
<tr>
<td>Within Groups</td>
<td>79043.615</td>
<td>500</td>
<td>158.087</td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>79214.231</td>
<td>501</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>NEG AFFECT</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Between Groups</td>
<td>8.353</td>
<td>1</td>
<td>8.353</td>
<td>.654</td>
</tr>
<tr>
<td>Within Groups</td>
<td>20798.756</td>
<td>500</td>
<td>41.598</td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>20807.110</td>
<td>501</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>NEG SADNESS</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Between Groups</td>
<td>3.543</td>
<td>1</td>
<td>3.543</td>
<td>.670</td>
</tr>
<tr>
<td>Within Groups</td>
<td>9720.210</td>
<td>500</td>
<td>19.440</td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>9723.753</td>
<td>501</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Table 6. One-way ANOVA: Mild Asthma Severity vs. Moderate-to-Severe Asthma Severity.

<table>
<thead>
<tr>
<th>Source</th>
<th>SS</th>
<th>df</th>
<th>MS</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>DD</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Between Groups</td>
<td>.001</td>
<td>1</td>
<td>.001</td>
<td>.164</td>
</tr>
<tr>
<td>Within Groups</td>
<td>.020</td>
<td>45</td>
<td>.000</td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>.021</td>
<td>46</td>
<td></td>
<td></td>
</tr>
<tr>
<td>PD</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Between Groups</td>
<td>.072</td>
<td>1</td>
<td>.072</td>
<td>.865</td>
</tr>
<tr>
<td>Within Groups</td>
<td>90.769</td>
<td>37</td>
<td>2.453</td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>90.842</td>
<td>38</td>
<td></td>
<td></td>
</tr>
<tr>
<td>ASQ CoNEG</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Between Groups</td>
<td>7.334</td>
<td>1</td>
<td>7.334</td>
<td>.869</td>
</tr>
<tr>
<td>Within Groups</td>
<td>15305.582</td>
<td>57</td>
<td>268.519</td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>15312.915</td>
<td>58</td>
<td></td>
<td></td>
</tr>
<tr>
<td>NEG AFFECT</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Between Groups</td>
<td>68.430</td>
<td>1</td>
<td>68.430</td>
<td>.168</td>
</tr>
<tr>
<td>Within Groups</td>
<td>1997.739</td>
<td>57</td>
<td>35.048</td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>2066.169</td>
<td>58</td>
<td></td>
<td></td>
</tr>
<tr>
<td>NEG SADNESS</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Between Groups</td>
<td>21.763</td>
<td>1</td>
<td>21.763</td>
<td>.279</td>
</tr>
<tr>
<td>Within Groups</td>
<td>1036.406</td>
<td>57</td>
<td>18.183</td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>1058.169</td>
<td>58</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
APPENDIX B

Figures
Figure 1. An exponential discount curve and a hyperbolic (more bowed) curve from the same reward. As time passes (rightward along the horizontal axis), the motivational impact-- the value-- of the goal gets closer to its undiscounted size, which is depicted by the vertical line (Ainslie, 2005).
Figure 2. Hyperbolic discount curves from two rewards of different sizes available at different times. The smaller reward is temporarily preferred for a period just before it's available, as shown by the portion of its curve that projects above that from the later, larger reward (Ainslie, 2005).
APPENDIX C

IRB Form
Oklahoma State University Institutional Review Board

Date: Tuesday, August 22, 2006
IRB Application No. AS06107
Proposal Title: Probability Discounting and Attributional Style in College Students

Reviewed and Processed as: Expedited

Status Recommended by Reviewer(s): Approved Protocol Expires: 8/21/2007

Principal Investigator(s)

Leafar Francesco-Jose  Frank L Collins
215 North Murray  215 N Murray
Stillwater, OK 74078  Stillwater, OK 74078

The IRB application referenced above has been approved. It is the judgment of the reviewers that the rights and welfare of individuals who may be asked to participate in this study will be respected, and that the research will be conducted in a manner consistent with the IRB requirements as outlined in section 45 CFR 46.

The final versions of any printed recruitment, consent and assent documents bearing the IRB approval stamp are attached to this letter. These are the versions that must be used during the study.

As Principal Investigator, it is your responsibility to do the following:

1. Conduct this study exactly as it has been approved. Any modifications to the research protocol must be submitted with the appropriate signatures for IRB approval.
2. Submit a request for continuation if the study extends beyond the approval period of one calendar year. This continuation must receive IRB review and approval before the research can continue.
3. Report any adverse events to the IRB Chair promptly. Adverse events are those which are unanticipated and impact the subjects during the course of this research; and
4. Notify the IRB office in writing when your research project is complete.

Please note that approved protocols are subject to monitoring by the IRB and that the IRB office has the authority to inspect research records associated with this protocol at any time. If you have questions about the IRB procedures or need any assistance from the Board, please contact Beth McTernan in 415 Whitehurst (phone: 405-744-5700, beth.mcternan@okstate.edu).

Sincerely,

Sue C. Jacobs, Chair
Institutional Review Board
VITA

Leafar Francesco-Jose Espinoza

Candidate for the Degree of

Doctor of Philosophy

Dissertation: DISCOUNTING, ATTRIBUTIONAL STYLE, AND DEPRESSIVE SYMPTOMATOLOGY IN COLLEGE STUDENTS WITH AND WITHOUT ASTHMA.

Major Field: Psychology

Biographical

Education: Graduated from Sasolburg High School, Sasolburg, South Africa in December 1995; received a Bachelor of Arts degree in Psychology from Potchefstroom University Vaal Triangle Campus, Vanderbijlpark, South Africa in December 1998; graduated Cum Honoribus in December 1999 and received a Post Graduate Higher Education Diploma in December 2000 from Rand Afrikaans University, Johannesburg, South Africa; received a Master of Science degree with a major in Psychology at Oklahoma State University in July, 2005. Completed the requirements for the Doctor of Philosophy Degree with a major in Clinical Psychology at Oklahoma State University in July 2007.

Experience: Employed by Oklahoma State University as a graduate research assistant, graduate teaching assistant, graduate instructor, and psychological associate from 2001-2004. Served as a practicum student in the A Better Chance (ABC) clinic at University of Oklahoma Health Science Center 2003- 2005. Employed as a pre-doctoral intern at the Children’s Mercy Hospital, Kansas City, KS from 2006-2007.

Professional Memberships: Association for the Advancement of Behavior Therapy and the American Psychological Association.
Name: Leafar Francesco-Jose Espinoza Date of Degree: July, 2007

Institution: Oklahoma State University Location: Stillwater, Oklahoma

Title of Study: DISCOUNTING, ATTRIBUTIONAL STYLE, AND DEPRESSIVE SYMPTOMATOLOGY IN COLLEGE STUDENTS WITH AND WITHOUT ASTHMA.

Pages in Study: 89 Candidate for the Degree of Doctor of Philosophy

Major Field: Clinical Psychology

Scope and Method of Study: It was the purpose of this study to investigate alternative methods of exploring the relationship between delay discounting and depression. A sample of 507 college students completed the Delay and Probability discounting tasks, the Attributional Style Questionnaire (ASQ), and the Positive and Negative Affect Scale (PANAS-X) in an online study. Individuals with asthma were identified to run exploratory analyses comparing the non-asthma group to the asthma group.

Findings and Conclusions: This study supported the literature concerning the relationship between depressive symptomatology and delay discounting in that depressive symptomatology significantly predicted rates of delay discounting. This study provided further evidence that delay and probability discounting may indeed measure different constructs. Attributional style and probability discounting did not demonstrate a significant relationship. No significant differences between the healthy group and the asthma group emerged. Strengths, limitations, and future directions are discussed.