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Joan E. McDowell

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Attributions Others Assign to Depressed Individuals and their Relationship to Severity of Depressive Symptoms, Amount of Contact, and Familiarity with Depressed Individuals

by

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Master’s Thesis

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in

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Ypsilanti, Michigan
ATTRIBUTIONS OTHERS ASSIGN TO DEPRESSED INDIVIDUALS AND THEIR RELATIONSHIP TO SEVERITY OF DEPRESSIVE SYMPTOMS, AMOUNT OF CONTACT, AND FAMILIARITY WITH DEPRESSED INDIVIDUALS

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Abstract

While there has been valuable research critical for furthering our understanding of how an individual’s social network affects recovery from depression, we need to know more about the interplay of other people’s attributions and their relationships with depressed individuals that may impact recovery from the disorder. This research investigated causal and controllability attributions that others assign to individuals with depression by distributing a questionnaire to faculty and administrative staff at Eastern Michigan University to obtain ratings of attributions for depression. The level of intimacy within a relationship and the severity of depression were related to others’ beliefs about the controllability of depressive symptoms. Exploring these relationships may expand our understanding of how and when specific attributions begin and how they change. This information may be useful in developing treatments for depression that not only help the individual suffering with the disorder but also others around the individual who are affected.
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Attributions Others Assign to Depressed Individuals and the Relationship to Severity of Depressive Symptoms, Amount of Contact, and Familiarity with Depressed Individuals

Introduction

The purpose of this thesis was to test the hypothesis that relationship variables, such as the length of time a person has known a depressed person and the severity of the depression, are related to beliefs about the controllability of specific symptoms. Differences in these attributions were expected based on relationship variables. Exploring the relationship between these variables and attributions may expand our understanding of how and when specific attributions begin and whether they change over time. Others’ attributions for depression may foster or hinder the process of healing in depressed individuals by creating an environment of acceptance and understanding or one of hostility and criticism. The bidirectional influence between depression and an individual’s social environment suggests that researching the relationships between these dimensions may provide insight into methods of treating depression that shorten depressive episodes and reduce relapse rates (Casten, Rovner, Scmuely-Dulitzki, Pasternak, Pelchat, & Ranen, 1999; Ezquiga, Garcia, Bravo, & Pallares, 1998; Klerman, Weismann, Rounsaville, & Chevron, 1996).

Nineteen million adult Americans will have some form of depression each year (Young, Weinberger, & Beck, 2001). Developing new treatments for depression will have far-reaching impact not only for depressed individuals but for millions more who are affected by those with depression. The stress and strain caused by having a family member with a mental illness can be considerable and can lead to dysfunctional patterns of interaction that can have adverse effects on the patient (Hill, Shepherd, & Hardy, 1998). Depressive behaviors such as complaining, lack of interest in social life, fatigue, and feelings of
helplessness may create difficulties within the family. Excessive reassurance-seeking and depressive mood contribute to negative partner attitudes (Benazon, 1998). Others may hold the ill person responsible for his or her actions when they come to feel that certain behaviors are under the control of the depressed person. In reaction to the affected individual’s depressive behaviors, family members may become hostile, critical, or withdrawn from the family member with a depressive disorder (Benazon, 2000; Coyne, Kessler, Tal, & Turnbull, 1987).

These studies suggest that there is a bi-directional influence between depression and an individual’s social environment. Major depression affects not only the individual diagnosed with the condition, but also those around the person, which then creates patterns of interacting that maintain depressive symptoms. There is a need for developing more knowledge regarding the complex relationship between depression and its influence on the depressed individual’s social support system. Research indicates that social support has a strong relationship to recovery (Ezquiga, Garcia, Bravo, & Pallares, 1998). Moreover, therapy aimed at the interpersonal context facilitates recovery and protects against relapse (Klerman, Weismann, Rounaville, & Chevron, 1996). Previous research gives support for the potential of a theory of depression encompassing the idea that family members are reactive to the presence of a depressive person in the household. Researching the relationships between attributions and various levels of exposure to depression may provide insight into methods of treating depression that shorten depressive episodes and reduce relapse rates.
Interpersonal Links

It is well known that life events and the social environment affect mood. Depression often follows major negative life events such as divorce, death of a loved one, or loss of a job (American Psychiatric Association, 2000). These events can cause changes in the individual's social environment and, therefore, an individual's mood. The strong inverse relationship between the quality of close relationships and depressive symptomatology has been demonstrated with research encompassing many types of subjects and across many disciplines.

For example, Field, Diego, and Sanders (2001) identified parental relations, affection, and intimacy as important contributors to the psychological health of adolescents. Critical communication within the family has also been associated with poorer outcomes for depressed youth (Asarnow, Goldstein, Tompson, & Guthrie, 1993). Social support, particularly the size of the social network and everyday psychological support from a partner, also has a strong relationship to incomplete recovery in major depression (Ezquiga, Garcia, Bravo, & Pallares, 1998). Likewise, Cronkite, Moss, Twohey, Cohen, and Swindle (1998) found that an individual without social resources is at considerable risk for partial or non-remission. As suggested by these examples, the link between depression and interpersonal factors has important implications for treatment.

The strong association between depression and social factors raises the question of whether there is a causal relationship. There may be a direct causal effect of interpersonal dysfunction on depression, the depression may trigger relationship difficulties, or there may be a bi-directional influence for both the disorder and the dysfunction. There is evidence of an impact of depression on close relationships as well as evidence of the impact of the
interpersonal environment on depression. This leaves open the question of the direction of the effect. Even when there are no existing causal factors, familial factors that exacerbate or maintain depression may develop over the course of the illness.

**Bi-directional Influences**

The research literature indicates that a reciprocal model of effect for depression and interpersonal factors is most promising. The environment affects mood; however, mood also influences social functioning and one’s environment (Markowitz, 1998). For example, the environment can affect mood when the actions of an overprotective spouse create threats to self-esteem and reduce feelings of self-efficacy (Holahan, Moos, & Bonin, 1999). Conversely, mood influences the environment as the patient and partner’s moods become correlated. Many studies have shown that significant others of patients with depression have an increased risk for distress (Benazon, 2000; Coyne, Thompson, & Palmer, 2002; Coyne, Wortman, & Lehman, 1988). For example, Coyne (2001) found that subjects who had spoken to depressive patients were more depressed, anxious, hostile, and rejecting. This finding suggests that the environmental response to an individual with depression may play an important role in the maintenance of depressive symptoms.

People suffering from depression often engage in reassurance-seeking behavior in an attempt to offset their feelings of hopelessness. Excessive reassurance-seeking and depressive mood, however, contribute to negative partner attitudes (Benazon, 1998). In a study of dating relationships, men were more likely to exhibit relationship discord when partners reported depressive symptoms, reassurance-seeking, and interest in negative self-relevant feedback (Katz & Beach, 1997).
Coyne, Kahn, and Gotlib (1987) describe an interactional theory that explains how families affect and are affected by depressed family members. This interactional theory suggests that the demands of depressed persons for reassurance and support contribute to partner rejection. For instance, depressed individuals may be preoccupied with thoughts of others leaving them. Their excessive demands for reassurance may serve to decrease empathy from nondepressed partners which, in turn, is perceived as a lack of support, worsening the depressive symptoms.

The reciprocal effect of environment and mood serves to maintain the illness. Living with a depressed individual creates an environment in which patterns of negativity, hostility, and criticism may become pervasive. It is not clear which aspects of living with a depressed person lead to negativity. Is it the closeness of the relationship, the amount of time the individual has spent with the depressed person, or the severity of the depression that leads caretakers to feel resentful, hostile, or frustrated? This paper addresses this question by evaluating aspects of living with a depressed person that relate to attributions that may affect the interpersonal relationship.

*Effects on Family Members*

After finding out that a person they love has been diagnosed with a mental illness, family members must learn to cope with intense emotional responses to the illness (Karp & Tanarugsachok, 2000). They may also find that they are caring for the individual without much help, education, or training (Hill, Shepherd, & Hardy, 1998). Caretakers have to monitor their relative’s behavior while being extremely sensitive to their own reactions. Caretakers often find that they have to “walk on eggshells” because of the depressed person’s sensitivity to criticism, especially from partners (Hill, Shepherd, & Hardy). Attempts to
support and care for the individual with a mental illness can fail as emotional overinvolvement breaks down proper functioning within the family (Coyne, Wortman, & Lehman, 1988).

The effects on quality of life for family members in terms of family relationships, friends, social life, and work prospects are generally negative (Fadden, Bebbington, & Kuipers, 1987; Hill, Shepherd, & Hardy, 1998; Yarrow, Schwartz, Murphy, & Deasy, 1955). When children are involved, parenting roles may shift, with the non-depressed partner having to assume more responsibility for the children in addition to taking care of the depressed individual. Caregivers report stress, anxiety, and depressive symptoms of their own, as well as negative effects on their self-esteem and confidence that often fluctuates with their partner’s mood state (Hill, Shepherd, & Hardy, 1998).

The burden associated with having a family member with a mental illness has been well documented (Baronet, 1999; Perlick, Clarkin, Sirey, Raue, Greenfield, Struening, & Rosenheck, 1999). Tessler and Gamache (1994) reported that the greatest caretaking burdens fall on spouses and parents who live with mentally ill individuals. Coyne, Kessler, Tal, and Turnbull (1987) found that living with a person in a depressive episode produces numerous burdens in response to the patient’s symptoms that lead to psychological distress of the caregiver.

These burdens and the resulting distress may create dysfunctions that affect the course of the illness. Coyne, Wortman, and Lehman (1988) found that the quality of others’ involvement and support deteriorates over time. New patterns of relating within the family may be formed, resulting in a situation that exacerbates or maintains the depressive symptoms.
Expressed Emotion

One of the patterns of behavior in a family, which may be counterproductive to recovery from depression, has been labeled expressed emotion (EE). Hooley and Campbell (2002) describe EE as having three components: criticism, hostility, and emotional over-involvement. High levels of EE correspond to critical, hostile, and over-involved behaviors. Low EE is characterized by empathic, calm, and respectful conduct (Leff, Kuipers, Berkowitz, & Sturgeon, 1985). Research suggests that EE affects the course of illness and the likelihood of relapse in depression as well as several other disorders (Asarnow, Goldstein, Tompson, & Guthrie, 1993; Butzlaff & Hooley, 1998; Greene, 1998; Hooley, Orley, & Teasdale, 1986; Licht, 2001; Vaughn & Leff, 1976). In one 9-month follow-up study linking high levels of EE with relapse rates, none of the patients in a family with low levels of EE relapsed, whereas 59% of persons living in a family with high levels of EE relapsed (Hooley, Orley, & Teasdale, 1986). Similarly, McCleary and Sanford (2002) found that high EE within a family predicts a worse clinical course for depression, and low EE predicts remission among an adolescent sample. In a study of patients with bipolar depression, patients in families with more critical EE had more symptoms after ten months than those in families with low EE (Greene, 1998). Hooley and Teasdale (1989) studied the predictive validity of EE, marital distress, and patients’ perceptions of criticism from spouses and found that the single best predictor of relapse for unipolar depression was perceived criticism.

In addition, Butzlaff and Hooley (1998) found that the association between EE and relapse was stronger for more chronically ill patients. Hooley, Orley, and Teasdale’s study (1986) found that high EE, marital distress, and the patient’s perceptions of criticism by spouses were associated with depressive relapse in patients who had been hospitalized for
depression. A replication and extension of a study of hospitalized schizophrenic patients added a group of depressive patients. This study considered EE the best single predictor of symptomatic relapse during the nine months after discharge from the hospital (Vaughn & Leff, 1976).

High levels of EE may develop over time as family members are exposed to depressive symptomatology. The depressive communication style (hostile, argumentative, and demanding), self-disclosure, and negative facial expressions of individuals with depression evoke unfavorable reactions (Schwartz, Fair, Salt, Mandel, & Klerman, 1976; Waxer, 1974). Therefore, this inappropriate self-disclosure and complaining guarantees the partner seeing and confirming the individual’s negative qualities. It becomes easier for family members to deliver the desired negative feedback as they react to these depressive symptoms. Individuals are especially sensitive to feedback if it comes from a close relationship partner (Swann & Predmore, 1985). This change in family functioning may lead to the development of high levels of EE that serve to maintain the illness.

Interventions to help friends and relatives caring for people with depression generally share the core idea of reducing levels of EE (criticism, hostility, and over involvement) (Anderson, Hogarty, Bayer, & Needleman, 1984; Leff, Kuipers, Berkowitz, & Sturgeon, 1985; Vaughn, 1989). Family-based treatments that reduce EE have shown an associated reduction in relapse rates (Asarnow, Tompson, Woo, & Cantwell, 2001). Thus, understanding and recognizing the causes of EE in families with a depressed loved one may help in developing treatment programs that target specific beliefs and behaviors.
Attributions

The adjustment for families of individuals with depression is difficult because of enduring social and cultural stigma and the tendency to perceive mentally ill persons as weak, dangerous, and unpredictable (Crisp, Gelder, Rix, Meltzer, & Rowlands, 2000). Many people view depression as a sign of weakness, not an illness. Embarrassment, shame, and denial function to create a poor environment for adjusting to living with a mentally ill family member. In addition, many practitioners deal with families on the basis of etiological assumptions about their role in the causation of the illness. One study found that almost half of social work practitioners believe that the aim of therapy for severe mental illness should be to get family members to recognize their own culpability in their family member’s illness (Rubin, Cardenas, Warren, Pike, & Wambach, 1998), even though there are indications that doing so would be ineffective, create stress and burden for the family, and perhaps be harmful to the individual with the disorder (Lefley, 1998). There is a currently a lack of detailed information about the role that attributions play in affecting relationships between depressed and non-depressed individuals. Because information regarding how best to help is insufficient, people sometimes do what is counterproductive for recovery from depression. Caregivers’ attributions about the cause of depression, as well as their beliefs about the controllability of various behaviors associated with depression, can be crucial in helping the individual recover from depression.

This current project adds to the work that has already been done concerning attributions by taking a closer look at several dimensions. Studying how attributions are related to exposure to mental disorders may help identify family members as secondary victims to the illness regardless of the cause of the disorder. This may allow the therapist and
the family to become partners in treatment. Using nonjudgmental psychoeducation and teaching coping skills that foster recovery may lead to better outcomes for depressed individuals.

Research has been conducted that suggests a link between attributions and the course of the illness (Casten, Rovner, Scmuely-Dulitzki, Pasternak, Pelchat, & Ranen, 1999). In Casten et al.’s study of geriatric psychiatry inpatients, having fewer depressive symptoms that the caregiver perceived to be within the patient’s control predicted remission of the depression at discharge. However, there are also studies that have found no link between attributions and risk for relapse (Hooley & Licht, 1997; Licht, 2001). These conflicting results may be due to differences in the composition of the samples. The first study’s sample was composed of caregivers for geriatric inpatients while the latter studies used participants related to depressed individuals with a much lower mean age of whom only about half had been hospitalized. Therefore, severity of depression and the level of exposure participants had to the depressed individuals may account for the difference in results. Another possible explanation is that the attributions under study were somewhat different. Are attributions concerning the cause of the illness or attributions concerning the controllability of symptoms more related to the course of depression? Different constructs of causal attributions and attributions of controllability may have important implications for caregiver and patient outcomes.

Many previous studies in this area of research have used a five dimensional model of attributions composed of internal/external, global/specific, stable/unstable, personal/universal, and controllable/uncontrollable dimensions, or some combination of these dimensions (Barrowclough, Johnston, & Tarrier, 1994; Hooley & Licht, 1997; Licht, 2001).
Part of the difficulty in interpreting the results of these studies is that these dimensions are often blurred. In many studies it is not clear whether participants are reporting causal attributions for the disorder or attributions of responsibility for current symptoms. In other words, when a person attributes controllability to an individual with a mental illness, it could mean that the individual should have been able to control the onset of the disorder, or that the individual should be responsible for recovery from the illness or maintenance of symptoms.

Furthermore, attributions are sometimes blurred by definition. Individuals are assumed to be responsible for internal causes while external causation relieves the person of responsibility for causing the problem. However, in some studies, biological causes are included in the internal dimension because the cause resides with the individual. In this case, the assumptions of the internal/external dimension break down because the individual may not be blamed for a biological cause. Likewise, the stable dimension falls short when we consider the case of laziness. Stability is assumed to correlate with attributions of uncontrollability. However, habitual laziness is labeled as a stable attribution, and most people would assume an individual had control in this case.

In addition, controllability beliefs appear to change over time. Volunteers working with the mentally ill were given Rotter’s Internal-External Locus of Control Scale before their volunteer work began and three months later. Their pre-work scores did not differ from a control group. After working with the mentally ill for three months, the volunteers had higher measures of internal control than did the control group (Miller, 1974). This contradicts the current thinking that people maintain an attributional style. Studying whether controllability attributions change with various levels of exposure to depression may provide some insight into better treatments for depression.
Brickman et al.'s attributional theory (Brickman, Rabinowitz, Karuza, Coates, Cohn, & Kidder, 1982) suggests four models regarding personal responsibility that take into account the difference between blame and control. Blame is when an individual is held responsible for the origin of a problem, a past event. Controllability is holding a person responsible for the solution to a problem, a future event. People can believe that individuals are responsible - or not - for future solutions regardless of whether they are responsible - or not - for the cause of the problem. The moral model proposes that an individual is responsible for causing and solving problems. The compensatory model suggests that responsibility for solving, but not causing, problems lies with the individual. Responsibility for causing problems, but not for the solution to the problems, is the theme of the enlightenment model. Finally, the medical model suggests that an individual has no responsibility for causing or solving their problems.

Kleinke and Kane (1997) researched how people assigned responsibility for mental disorders to individuals with mental health problems. In general, people assigned more responsibility for solving problems than for causing them, consistent with the compensatory model. Thus, the inconsistency in the research on causal attributions may be due to confusion about attributions regarding cause or solutions for problems as well as the belief that attributions are a stable style or trait.

Relation of Attributions to Expressed Emotion

Coyne, Kessler, Tal, and Turnbull (1987) assert that while depression initially evokes sympathy and support, it subsequently produces impatience, frustration, and withdrawal by those in the environment as they come to see the symptoms as willfully unpleasant. Benazon
(2000) found that those around the patient become hostile, critical, and withdraw from what they see as morally weak behavior.

High levels of expressed emotion (EE) have been linked to familial attributions in which mentally ill persons are perceived as being in control of and accountable for their symptomatic behavior (Barrowclough, Johnston, & Tarrier, 1994; Hooley & Campbell, 2002; Licht, 2001). In a study of 43 spouses of psychiatric inpatients who met criteria for major depressive episode, Hooley and Licht (1997) found that relatives who expressed high levels of EE attributed more control to their ill family members. Criticism was the dimension of EE that was most strongly linked to these attributions of controllability.

Because attributions are related to EE, we would expect measures of attributions to have predictive power for relapse similar to EE measures. Yet previous research results are inconsistent regarding the predictive value of attributions. Some studies have found that attribution dimensions do not predict patient relapse (Hooley & Licht, 1997; Licht, 2001). However, in a study of patients with schizophrenia, attribution variables were better predictors of relapse than were EE measures (Barrowclough, Johnston, & Tarrier, 1994). Further study of attributional styles with depressed persons is necessary to clarify the unexpected results.

It is probable that there are various dimensions that affect how much impact relatives’ attributions have on a depressed person. Although a relationship between high EE and negative attributions has been established, Hooley and Campbell (2002) discovered that, even among relatives with a negative attributional style, most believed the patient was genuinely ill. Furthermore, the relatives believed that not all of the depressive symptoms were under the patient’s control. This suggests that family members may have different
attributions for the disorder in general than they have for specific symptoms of the disorder. In other words, causal attributions may be independent from the controllability of symptoms.

**Summary of Current Literature**

Psychotherapeutic intervention aimed at the interpersonal context can facilitate patients’ recovery from an acute episode of depression and may have preventative effects against relapse (Klerman, Weismann, Rounsaville, & Chevron, 1996). Frank, Kupfer, Wagner, McEachran, and Cornes (1991) found that patients whose therapy focused on interpersonal factors had longer time before relapse compared to a group with less focus on interpersonal aspects of their lives. The fact that these types of therapy are effective in treating depression suggests that the familial environment must be a predominant factor in any theory of depression.

Individuals who are significant in the lives of persons with depression have something to contribute to the recovery of the patients, but they are also likely to have unmet needs themselves (Coyne, 1999). Because of the chronic nature of depression and the burden on those caring for a relative with a mental illness, family members need to learn skills to help them cope. High levels of expressed emotion from family members have been associated with depression but may be a response to the illness, instead of a causal factor. One model suggests that mood influences environment, the environment influences mood, and the cyclical effect serves to maintain the illness.

In sum, changes associated with depressive symptomatology may set the stage for negative familial behavior. Depressed persons may exhibit depressive behaviors such as complaining, social withdrawal, and fatigue, thereby inducing negative affect and rejection from others. Ultimately, this pattern of responding may result in increased distress for both
the patient and their social network by producing family functioning difficulties such as EE and negative attributional style.

These interpersonal dysfunctions can have a cumulative effect over time, turning into ongoing patterns of behavior. These cycles may lock depressed persons into longer or more serious episodes of depression as well as increase the risk for future recurrent episodes. Coyne and Benazon (2001) explored the reasons why some people have longer periods of remission between depressive episodes. They cautioned against concluding that poor marital functioning causes a quicker relapse since the factor most associated with relapse was recent recovery. However, the fact that there are differences in the length of time people are remitted suggests the presence of mediating or moderating factors. Further exploration of these factors may lead to a better understanding of the course of depression as well as a means for improving the well-being of family members living with a depressed person.
Purpose of Current Study

The present study is concerned with the types of attributions participants make about depression and whether relationship variables are related to differences in these attributions. The literature is sparse concerning the breakdown of attributions by type (causal or controllability) or by relationship variables (familiarity, chronicity, etc.). In a study of 42 couples in which one member had depression, Hooley and Campbell (2002) reported no relationship between attributions of control and the age of the patients or the length of time the couple had been married. There were differences by gender, with women being more likely to attribute control to their spouse, and differences by the amount of time spent together each week, with more time associated with lower attributions of control.

Two dimensions of attributions were explored in this study. Previous research by Robinson (1996) found that biogenetic causal attributions were the most highly endorsed responses for clients and family members who had been involved in a program emphasizing a strong biological and genetic cause for depression. Likewise, more clients were found to hold etiology beliefs similar to their therapist after counseling than before counseling (Atkinson, Worthington, Dana, & Good, 1991). In addition, Read and Law (1999) found that undergraduate students' etiology beliefs changed after a series of four lectures presenting the causes and solutions to mental health problems.

The implication is that causal attributions may be related to the theoretical orientation of the setting or therapist rather than other factors. It is likely that causal attributions may develop through psychoeducation about mental disorders, contact with mental health professionals, and so on, rather than through any specific experiential variables. Attributions regarding causality and controllability are not necessarily related, even though previous
researchers often interpreted attributions in that manner. In other words, an individual can believe that depression occurs because of bad luck, yet also believe that personal control can effect change. Therefore, controllability attributions were measured separately from causal attributions. The first hypothesis suggested that causal attributions about the etiology of depression were expected to be unrelated to controllability attributions.

The second dimension of attributions explored participants’ beliefs about the controllability of depressive symptoms. Previous research reported that the more time spouses spent together, the lower the attributions of control over behavior the spouse assigned to the depressed partner (Hooley & Campbell, 2002). Moreover, the developmental course of depression suggests that at the initial phases of depression family members may be sympathetic to the person’s symptoms seeing them as products of the disorder and not within the individual’s control. As the disorder progresses, family members may become less tolerant of symptoms, and believe the depressed individual is willfully engaging in these behaviors. Nevertheless, as the disorder continues and no change occurs, family members may return to earlier beliefs about the controllability of symptoms.

Consequently, this study’s second hypothesis is that the attributions participants make concerning depression are associated with the amount of contact that an individual has had with depressed individuals, the severity of the depression, and the familiarity of the individual to the depressed person. In other words, those who had closer relationships for longer periods of time with severely depressed individuals were expected to have different attributions about depression than those who were less familiar with depressed individuals. This was expected to follow a curvilinear trend; participants with moderate ratings on
relationship variables would have higher controllability ratings than those with low or high relationship variable ratings.
Method

Participants

Many of the analyses conducted in this study used four groups: no contact, mild, moderate, and high/severe contact with depressed individuals. Hypothesizing a moderate effect size on dependent measures and conducting analyses with alpha set to 0.05, then an 80% chance of detecting a significant effect may be achieved with a sample size of forty-five per group (Cohen, 1992). Thus, the goal was to recruit 180 to 200 participants across groups.

Testing for curvilinearity requires sampling across the entire range in a given context in order to have enough participants in each area of the curve. Thus, using a convenience sample rather than a clinical sample was desirable for this study in order to acquire a full range of exposure to depressed individuals.

Participants included 209 faculty and administrative staff members at Eastern Michigan University. One case was removed because the participant noted that their answers reflected a professional relationship rather than a personal relationship. Participants were 69.7% female, with one not indicating gender. Age was designated by category. There were six participants aged 17-21 (2.9%), 30 individuals aged 22-30 (14.4%), 52 individuals aged 31-40 (25%), 52 participants aged 41-50 (25%), and 67 participants aged 51 and over (32.2%). One participant did not indicate an answer for the age item. Participants included 175 Whites/non-Hispanics, 17 Blacks, 4 Latinos/Hispanics, 5 Asians, and 4 Other, with 3 missing data on this item.

Measure and Procedure

Questionnaires were distributed to participants through departmental mailboxes. A letter of introduction briefly described the study and provided instructions to return
completed instruments through campus mail. Questionnaires were mailed primarily to non-academic departments in an attempt to oversample individuals with a lower level of education to control for the high levels expected within a university setting. Each participant anonymously and voluntarily filled out a questionnaire regarding their attributions for causality and the amount of control that they expect individuals to have over symptoms of depression (see Appendix A for sample questionnaire).

A self-report questionnaire was developed to determine whether specific attributions are related to participant variables concerning their amount of contact with a depressed individual. Each participant was asked to indicate the demographic information of age, sex, educational level, and ethnicity. In order to limit the study to attributions for clinical depression, the survey instructed participants to rate items with respect to a person that they know who has been diagnosed with clinical depression, hospitalized, or placed on antidepressants for depression. Questions regarding relationships with depressed individuals asked the participant to focus on the one person who the respondent feels has the most severe depression. The questionnaire also included an item regarding whether the participant has ever personally been diagnosed with clinical depression, hospitalized, or placed on antidepressants for depression. This allowed for separate analyses to be performed for this portion of the subject pool to determine if personal experience affected the attributions that people have for depression.

*Relationship with depressed person.* Items inquired about the respondent’s relationship to the depressed person, how close the respondent feels to the depressed person, how long the participant has known that the person suffers from depression, how many episodes of depression there have been since the respondent has known the person with
depression, the typical length of the depressive episodes, a subjective rating of the severity of the depression, and whether the participant lived with the depressed individual while they had a depressive episode. Respondents who do not know anyone with depression were instructed to skip those questions and continue to the next section.

**Controllability.** Next, respondents were asked to rate the amount of controllability they believe a depressed individual has over 15 symptoms of depression. The items were developed by the researcher. Items were chosen based on descriptors from the major depressive episode section of the DSM-IV-TR (American Psychiatric Association, 2000). Respondents who do not know a depressed individual were instructed to rate attributions and controllability items as they believed they would apply to most individuals with depression. The items were rated on a 5-point scale anchored by “no control = 1” and “complete control = 5.” In addition, items could be excluded by answering “not observed/don’t know.”

**Causality.** Next, the participants were asked to rate the degree to which they agree with seven statements concerning the etiology of depression (e.g. Clinical depression is due to genetic/biological factors, chance/bad luck, family of origin conflict, etc.) using a 5-point scale anchored by “strongly agree = 1” and by “strongly disagree = 5.” These items were also developed by the researcher. The items were chosen based on a review of the literature concerning depression attributions. Finally, the survey asked respondents to estimate the percentage to which each of the seven causes is responsible for depression. Respondents could assign each item any percentage value, including 0%, as long as the total for the seven items was 100%. Both questions instructed participants to answer based on their beliefs about the person that they knew with depression or, if the participant did not personally know
anyone with depression, to answer based on how they believed it would apply to most individuals with depression.

The two ways of measuring causality were developed so people’s beliefs about the absolute and relative contributions of genetics and other environmental factors to depression could be explored. The absolute ratings and relative percentages could also be examined in terms of age, gender, and education.
Results

Preliminary Analyses

Principal components factor analysis was conducted to determine if any underlying factor structures exist for the fifteen controllability variables:

- crying (crying),
- irritability (irritable),
- no interest in activities (no interest),
- change in appetite (appetite),
- insomnia (insomnia),
- sleeping all the time (sleeping),
- too tired to get out of bed (tired),
- fatigue that keeps normal activities from being accomplished (fatigue),
- talk of worthlessness, hopelessness, or guilt (hopelessness),
- impaired concentration or decision-making (concentration),
- talk of death or suicide (suicidal),
- low self-esteem (self-esteem),
- delusions or hallucinations (delusions),
- complaining of aches (aches), and
- social withdrawal (withdrawal).

The initial analysis retained two components. The eigenvalue criterion was considered unreliable because all but two communalities were < .70. In addition, the components were uninterpretable after Varimax rotation. Therefore, the scree plot was
examined to determine the appropriate number of components to retain. This criterion indicated that only one component should be retained. This component accounted for 50.44% of the total variance in the original variables. There was a strong positive loading for all fifteen items on this one factor indicating that all the items measure the same construct (See Appendix B, Table B1). Therefore, a composite for controllability was created by computing the mean of all fifteen items to obtain a rating for global controllability \((M = 2.06, SD = .69)\). If a participant endorsed some items as “not observed, don’t know,” those items were not used to compute the mean. A minimum of seven items were required for this composite. This resulted in 25 participants with missing data. A reliability analysis revealed an alpha of .93 for the fifteen items.

In order to determine which variables are most salient for attributions, exploratory analyses were conducted to identify which variables should be explored further. Correlations were calculated for the controllability attribution items and the items concerning the respondent’s relationship to the depressed person:

- how close the respondent feels to the depressed person (close),
- how long the participant has known the person suffers from depression (time known),
- how many episodes of depression there have been since the respondent has known the person with depression (episodes),
- the typical length of the depressive episodes (length of episode), and
- a subjective rating of the severity of the depression (severity).

Results are shown in Appendix B, Table B2. In addition, ANOVAs were conducted to identify differences between the participants who lived with a depressed individual while
they had a depressive episode and those who did not \textit{(lived with/did not live with)} and differences between ratings for various types of relationships \textit{(relationship)}. There were no differences in global controllability ratings between those who lived with a depressed individual and those who did not, $F(1,121) = .03, p > .05$. Differences in global controllability ratings by type of relationship approached significance, $F(7,115) = 1.91, p = .07$. Given the preliminary results, relationship and severity of episode were explored in depth.

The preliminary results suggested that there may be differences in beliefs between those in various types of relationships to a depressed individual. Additional post hoc testing using the LSD method was conducted to identify which types of relationships were different. Results are shown in Appendix B, Table B3. The data were manipulated in order to combine participants who had similar responses for controllability beliefs and create categories with sufficient numbers of participants. Using the results from post hoc testing, a new variable was created combining those with a spouse or parent (14.4\%, $n = 30$), those with a child or sibling (15.9\%, $n = 33$), and those with other types of relationships (36.1\%, $n = 75$) into separate categories. Those who do not know someone (33.2\%, $n = 69$) were used as the first category in this new variable \textit{(relationship4)} in order to include participants with a full range of experience to depression.

\textit{Differences between Those Who Knew Someone with Depression and Those Who Did Not}

The absolute ratings (Please think about the following items and rate the extent to which you agree with each statement using the following scale) and relative percentages (Please estimate the percentage to which each of the causes listed below is responsible for depression in the person that you know) for causal beliefs were examined for differences
between participants who knew someone with depression and those who did not personally know anyone with depression. Independent samples $t$ tests for differences in absolute ratings of causal beliefs showed that individuals who knew someone were significantly more likely to rate genetic/biological factors, $t(202) = 3.79, p < .001$, conflicts within the family of origin, $t(203) = 3.64, p < .001$, and conflict with others, $t(202) = 2.70, p < .01$, as more likely to have caused the depression than those who did not know anyone with depression.

The ratings for all 7 items are reported in Table 1.

Table 1

**Causal Ratings for Those Who Know and Do Not Know Someone with Depression.**

<table>
<thead>
<tr>
<th>Causal variable</th>
<th>Know Someone $M (SD)$</th>
<th>Do not know someone $M (SD)$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Environmental</td>
<td>4.18 (0.81)</td>
<td>4.09 (0.70)</td>
</tr>
<tr>
<td>Genetic-biological **</td>
<td>4.04 (0.90)</td>
<td>3.48 (1.16)</td>
</tr>
<tr>
<td>Family of origin conflict **</td>
<td>3.92 (0.84)</td>
<td>3.45 (0.95)</td>
</tr>
<tr>
<td>Intrapersonal</td>
<td>3.84 (0.85)</td>
<td>3.64 (0.85)</td>
</tr>
<tr>
<td>Conflict with others *</td>
<td>3.76 (0.95)</td>
<td>3.37 (1.08)</td>
</tr>
<tr>
<td>Chance</td>
<td>2.12 (1.16)</td>
<td>2.25 (1.08)</td>
</tr>
<tr>
<td>Higher power/evil</td>
<td>1.85 (1.13)</td>
<td>1.72 (0.99)</td>
</tr>
</tbody>
</table>

*Note. Ratings were made on 5-point scales (1 = strongly disagree that variable is the cause of depression, 5 = strongly agree that variable is the cause).*

* $p < .01$. ** $p < .001$.

Independent samples $t$ tests for differences in the relative percentage of causation participants reported resulted in only one cause showing significant differences between the groups. Those who knew someone with depression reported that a significantly higher
The percentage of causation was due to conflict within the family of origin than those who did not know anyone with depression, $t(1, 196) = 2.69, p < .01$. The percentages for all 7 items are reported in Table 2.

Table 2

<table>
<thead>
<tr>
<th>Causal variables</th>
<th>Know someone $M (SD)$</th>
<th>Do not know someone $M (SD)$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Genetic-biological</td>
<td>31.09 (26.87)</td>
<td>30.29 (25.87)</td>
</tr>
<tr>
<td>Environmental</td>
<td>22.38 (18.85)</td>
<td>23.03 (17.29)</td>
</tr>
<tr>
<td>Family of origin conflict *</td>
<td>16.91 (12.59)</td>
<td>12.19 (9.77)</td>
</tr>
<tr>
<td>Intrapersonal</td>
<td>15.05 (15.15)</td>
<td>15.50 (11.63)</td>
</tr>
<tr>
<td>Conflict with others</td>
<td>12.65 (11.06)</td>
<td>14.13 (12.55)</td>
</tr>
<tr>
<td>Chance</td>
<td>2.26 (4.56)</td>
<td>2.23 (4.78)</td>
</tr>
<tr>
<td>Higher power/evil</td>
<td>2.22 (10.61)</td>
<td>3.74 (13.85)</td>
</tr>
</tbody>
</table>

*Note.* Participants could assign any percentage between 0 and 100 with instructions that all 7 percentages should total 100.

* $p < .01$.

Independent samples $t$ tests were also conducted for the 15 controllability items and the global controllability composite to determine if there were differences in controllability attributions between those who knew someone with depression and those who did not know anyone. There were no significant differences in global controllability attribution ratings between those who knew someone with depression and those who did not, $p > .05$. $T$ tests on individual controllability items did reveal one significant difference between the two groups. Those who knew someone with depression were more likely to believe the individual had
more control over delusions and hallucinations than did participants who did not know anyone with depression, \( t(1, 123) = 3.47, \ p < .01 \). The results for all 16 variables are shown in Table 3.

Table 3

<table>
<thead>
<tr>
<th>Controllability attributions</th>
<th>Know someone ( M (SD) )</th>
<th>Do not know someone ( M (SD) )</th>
</tr>
</thead>
<tbody>
<tr>
<td>Global controllability</td>
<td>2.08 (0.75)</td>
<td>2.04 (0.56)</td>
</tr>
<tr>
<td>Suicidal</td>
<td>2.74 (1.31)</td>
<td>2.54 (1.21)</td>
</tr>
<tr>
<td>Delusions *</td>
<td>2.42 (1.60)</td>
<td>1.53 (1.00)</td>
</tr>
<tr>
<td>Crying</td>
<td>2.32 (1.07)</td>
<td>2.07 (0.83)</td>
</tr>
<tr>
<td>Hopelessness</td>
<td>2.30 (1.06)</td>
<td>2.18 (0.98)</td>
</tr>
<tr>
<td>Irritable</td>
<td>2.28 (0.92)</td>
<td>2.28 (0.82)</td>
</tr>
<tr>
<td>Tired</td>
<td>2.22 (1.11)</td>
<td>2.29 (0.98)</td>
</tr>
<tr>
<td>Aches</td>
<td>2.13 (1.15)</td>
<td>2.25 (0.88)</td>
</tr>
<tr>
<td>Withdrawal</td>
<td>2.13 (0.96)</td>
<td>2.18 (0.98)</td>
</tr>
<tr>
<td>Sleeping</td>
<td>2.10 (1.09)</td>
<td>2.25 (1.00)</td>
</tr>
<tr>
<td>Fatigue</td>
<td>1.97 (0.98)</td>
<td>2.10 (0.99)</td>
</tr>
<tr>
<td>Self-esteem</td>
<td>1.95 (1.10)</td>
<td>1.84 (0.92)</td>
</tr>
<tr>
<td>No interest</td>
<td>1.90 (1.00)</td>
<td>1.97 (0.92)</td>
</tr>
<tr>
<td>Concentration</td>
<td>1.89 (1.05)</td>
<td>2.10 (0.81)</td>
</tr>
<tr>
<td>Appetite</td>
<td>1.88 (0.96)</td>
<td>1.75 (0.83)</td>
</tr>
<tr>
<td>Insomnia</td>
<td>1.64 (1.02)</td>
<td>1.44 (0.76)</td>
</tr>
</tbody>
</table>

*Note.* Ratings were made on 5-point scales (1 = no control over symptom, 5 = complete control over symptom).
It is worth noting that all of the items regarding attributions for the controllability of depressive symptoms had relatively low ratings. Most of the means for these items fell around a descriptor indicating that individuals have only a little control of the symptom. In other words, most participants do not believe individuals are able to control the symptoms of depression.

Because there appeared to be some differences between those who knew someone with depression and those who did not, the remaining analyses were conducted both with the entire sample and with only the portion of the sample who reported a personal relationship with a depressed individual.

**Relationship of Causal and Controllability Attributions**

Analyzing the entire sample, bivariate correlations for the relationship of the absolute causal ratings and global controllability attributions suggested that the belief in chance, \( r(183) = 0.20, \ p < .01 \), is related to how much control participants believed individuals had over all the symptoms of depression. Subjects who rated chance higher as a cause of depression were more likely to believe individuals have control over their symptoms. Correlations for all causal beliefs and individual controllability items are listed in Table 4.
### Table 4

**Correlations between Absolute Causal Ratings and Controllability Attributions for the Whole Sample.**

<table>
<thead>
<tr>
<th>Attributions</th>
<th>Biogenetic</th>
<th>Chance</th>
<th>Family of origin conflict</th>
<th>Conflict w/ others</th>
<th>Higher power/evil</th>
<th>Environment</th>
<th>Intrapersonal</th>
</tr>
</thead>
<tbody>
<tr>
<td>Global controllability</td>
<td>-.05 (182)</td>
<td>.20 (183)**</td>
<td>.04 (183)</td>
<td>.03 (183)</td>
<td>.07 (182)</td>
<td>.05 (183)</td>
<td>.14 (179)</td>
</tr>
<tr>
<td>Crying</td>
<td>-.02 (177)</td>
<td>.04 (178)</td>
<td>.00 (178)</td>
<td>.01 (178)</td>
<td>.04 (176)</td>
<td>-.06 (178)</td>
<td>.09 (174)</td>
</tr>
<tr>
<td>Irritable</td>
<td>-.06 (183)</td>
<td>.10 (184)</td>
<td>.10 (184)</td>
<td>.03 (184)</td>
<td>.01 (1832)</td>
<td>-.09 (184)</td>
<td>.12 (180)</td>
</tr>
<tr>
<td>No interest</td>
<td>-.12 (183)</td>
<td>.11 (184)</td>
<td>.05 (184)</td>
<td>-.00 (184)</td>
<td>.04 (183)</td>
<td>-.03 (184)</td>
<td>.13 (180)</td>
</tr>
<tr>
<td>Appetite</td>
<td>-.09 (175)</td>
<td>.19 (176)*</td>
<td>.01 (176)</td>
<td>-.00 (176)</td>
<td>.17 (175)*</td>
<td>.02 (176)</td>
<td>-.03 (172)</td>
</tr>
<tr>
<td>Insomnia</td>
<td>-.18 (163)*</td>
<td>.18 (164)*</td>
<td>.04 (164)</td>
<td>.06 (164)</td>
<td>.18 (163)*</td>
<td>.09 (164)</td>
<td>.07 (160)</td>
</tr>
<tr>
<td>Sleeping</td>
<td>-.06 (171)</td>
<td>.10 (172)</td>
<td>.06 (172)</td>
<td>-.05 (172)</td>
<td>.05 (171)</td>
<td>.07 (172)</td>
<td>.11 (168)</td>
</tr>
<tr>
<td>Tired</td>
<td>-.08 (169)</td>
<td>.17 (170)*</td>
<td>.08 (170)</td>
<td>.02 (170)</td>
<td>.07 (169)</td>
<td>.01 (170)</td>
<td>.05 (166)</td>
</tr>
<tr>
<td>Fatigue</td>
<td>.01 (175)</td>
<td>.13 (176)</td>
<td>.03 (176)</td>
<td>.06 (176)</td>
<td>-.02 (175)</td>
<td>.01 (176)</td>
<td>.09 (172)</td>
</tr>
<tr>
<td>Hopelessness</td>
<td>.06 (183)</td>
<td>.11 (184)</td>
<td>.05 (184)</td>
<td>.06 (184)</td>
<td>.00 (183)</td>
<td>.08 (184)</td>
<td>.10 (180)</td>
</tr>
<tr>
<td>Concentration</td>
<td>-.05 (184)</td>
<td>.20 (185)**</td>
<td>.00 (185)</td>
<td>-.02 (185)</td>
<td>.13 (184)</td>
<td>.09 (185)</td>
<td>.20 (181)**</td>
</tr>
<tr>
<td>Suicidal</td>
<td>.01 (160)</td>
<td>.10 (161)</td>
<td>.04 (161)</td>
<td>-.00 (161)</td>
<td>-.05 (159)</td>
<td>-.00 (161)</td>
<td>.05 (157)</td>
</tr>
<tr>
<td>Self-esteem</td>
<td>.05 (186)</td>
<td>.09 (187)</td>
<td>.04 (187)</td>
<td>.02 (187)</td>
<td>.00 (185)</td>
<td>.14 (187)*</td>
<td>.24 (184)**</td>
</tr>
<tr>
<td>Delusions</td>
<td>-.02 (124)</td>
<td>.27 (125)**</td>
<td>.09 (125)</td>
<td>.07 (125)</td>
<td>.18 (124)*</td>
<td>.07 (125)</td>
<td>.07 (121)</td>
</tr>
<tr>
<td>Aches</td>
<td>-.07 (165)</td>
<td>.17 (166)*</td>
<td>-.13 (166)</td>
<td>-.10 (166)</td>
<td>-.04 (164)</td>
<td>-.10 (166)</td>
<td>-.06 (162)</td>
</tr>
<tr>
<td>Withdrawal</td>
<td>.04 (186)</td>
<td>.01 (186)</td>
<td>.06 (186)</td>
<td>.11 (186)</td>
<td>.04 (185)</td>
<td>.04 (186)</td>
<td>.15 (182)*</td>
</tr>
</tbody>
</table>

*Note: Participants were allowed to answer "not observed/don't know" for the controllability attributions and these were excluded from analyses. N for each set of variables is in parentheses.*

* p < .05. ** p < .01.

When relative percentages were explored, a significant association was found between relative percentages of causality for two of the etiologies and global controllability.
ratings: biogenetic causes, \( r(176) = -0.14, p < .05 \), and intrapersonal variables, \( r(176) = .17, p < .05 \). Subjects who rated biogenetic causes high were more likely to believe that individuals have less control over their symptoms, while those who rated chance or intrapersonal variables high as a cause of depression were more likely to believe that individuals have more control over their symptoms. Correlations for all causal beliefs and individual controllability items are listed in Table 5.
Table 5

Correlations between Causal Percentage Ratings and Controllability Attributions for the Whole Sample.

<table>
<thead>
<tr>
<th>Attributions</th>
<th>Biogenetic</th>
<th>Chance</th>
<th>Family of origin conflict</th>
<th>Conflict w/ others</th>
<th>Higher power/evil</th>
<th>Environment</th>
<th>Intrapersonal</th>
</tr>
</thead>
<tbody>
<tr>
<td>Global controllability</td>
<td>-.18 (176)*</td>
<td>.12 (175)</td>
<td>-.01 (176)</td>
<td>.05 (176)</td>
<td>.14 (176)</td>
<td>-.02 (176)</td>
<td>.17 (176)*</td>
</tr>
<tr>
<td>Crying</td>
<td>-.04 (171)</td>
<td>.08 (170)</td>
<td>-.01 (171)</td>
<td>-.02 (171)</td>
<td>.15 (171)</td>
<td>-.08 (171)</td>
<td>.09 (171)</td>
</tr>
<tr>
<td>Irritable</td>
<td>-.12 (177)</td>
<td>.04 (176)</td>
<td>.08 (177)</td>
<td>.08 (177)</td>
<td>.16 (177)*</td>
<td>-.09 (177)</td>
<td>.24 (177)**</td>
</tr>
<tr>
<td>No interest</td>
<td>-.12 (178)</td>
<td>.15 (177)*</td>
<td>.04 (178)</td>
<td>.07 (178)</td>
<td>.11 (178)</td>
<td>-.06 (178)</td>
<td>.12 (178)</td>
</tr>
<tr>
<td>Appetite</td>
<td>-.07 (169)</td>
<td>.24 (168)*</td>
<td>-.06 (169)</td>
<td>.06 (169)</td>
<td>.10 (169)</td>
<td>-.02 (169)</td>
<td>.02 (169)</td>
</tr>
<tr>
<td>Insomnia</td>
<td>-.21 (157)*</td>
<td>.07 (156)</td>
<td>.04 (157)</td>
<td>-.07 (157)</td>
<td>.16 (157)*</td>
<td>.19 (157)*</td>
<td>.02 (157)</td>
</tr>
<tr>
<td>Sleeping</td>
<td>-.10 (166)</td>
<td>.11 (165)</td>
<td>.05 (166)</td>
<td>.00 (166)</td>
<td>.08 (166)</td>
<td>-.02 (166)</td>
<td>.09 (166)</td>
</tr>
<tr>
<td>Tired</td>
<td>-.19 (163)*</td>
<td>.18 (162)*</td>
<td>.11 (163)</td>
<td>.13 (163)</td>
<td>.12 (163)</td>
<td>.04 (163)</td>
<td>.15 (163)</td>
</tr>
<tr>
<td>Fatigue</td>
<td>-.17 (170)*</td>
<td>.17 (169)*</td>
<td>.01 (170)</td>
<td>.05 (170)</td>
<td>.15 (170)</td>
<td>.00 (170)</td>
<td>.18 (170)*</td>
</tr>
<tr>
<td>Hopelessness</td>
<td>-.12 (177)</td>
<td>.04 (176)</td>
<td>-.02 (177)</td>
<td>.04 (177)</td>
<td>.03 (177)</td>
<td>-.04 (177)</td>
<td>.13 (177)</td>
</tr>
<tr>
<td>Concentration</td>
<td>-.10 (178)</td>
<td>.03 (177)</td>
<td>-.08 (178)</td>
<td>-.01 (178)</td>
<td>.13 (178)</td>
<td>.01 (178)</td>
<td>.11 (178)</td>
</tr>
<tr>
<td>Suicidal</td>
<td>-.13 (155)</td>
<td>.06 (154)</td>
<td>.03 (155)</td>
<td>.09 (155)</td>
<td>.03 (155)</td>
<td>-.02 (155)</td>
<td>.04 (155)</td>
</tr>
<tr>
<td>Self-esteem</td>
<td>-.10 (180)</td>
<td>-.07 (179)</td>
<td>-.07 (180)</td>
<td>-.07 (180)</td>
<td>.00 (180)</td>
<td>-.02 (180)</td>
<td>.22 (180)**</td>
</tr>
<tr>
<td>Delusions</td>
<td>-.11 (121)</td>
<td>.04 (120)</td>
<td>.01 (121)</td>
<td>-.06 (121)</td>
<td>.11 (121)</td>
<td>-.02 (121)</td>
<td>.09 (121)</td>
</tr>
<tr>
<td>Aches</td>
<td>-.12 (160)</td>
<td>.05 (159)</td>
<td>-.10 (160)</td>
<td>.04 (160)</td>
<td>.21 (160)**</td>
<td>.00 (160)</td>
<td>.22 (160)**</td>
</tr>
<tr>
<td>Withdrawal</td>
<td>-.07 (179)</td>
<td>-.03 (178)</td>
<td>-.01 (179)</td>
<td>.04 (179)</td>
<td>.01 (179)</td>
<td>-.06 (179)</td>
<td>.08 (179)</td>
</tr>
</tbody>
</table>

Note: Participants were allowed to answer “not observed/don’t know” for the controllability attributions and these were excluded from analyses. N for each set of variables is in parentheses.

* p < .05. ** p < .01.
When only those who know someone with depression were included in the analyses, similar results were found. Chance, $r(122) = .21, p < .05$, and intrapersonal, $r(120) = .20, p < .05$, etiologies given as absolute ratings were associated with global controllability ratings. For relative percentages, biogenetic, $r(116) = -.24, p < .05$, higher power/evil, $r(116) = .20, p < .05$, and intrapersonal causes, $r(116) = .21, p < .05$, were significantly associated with global controllability when only those who knew someone were included in the analyses.

Correlations for the absolute and percentage ratings of causal attributions and global controllability ratings when only those who know someone with depression were analyzed are listed in Table 6.

**Table 6**

*Correlations between Causal Ratings and Global Controllability Attributions for Those Who Know Someone with Depression.*

<table>
<thead>
<tr>
<th></th>
<th>Absolute ratings</th>
<th>Percentage ratings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Biogenetic</td>
<td>-.03 (121)</td>
<td>-.24 (116)*</td>
</tr>
<tr>
<td>Chance</td>
<td>.21 (122)*</td>
<td>.13 (116)</td>
</tr>
<tr>
<td>Family of origin conflict</td>
<td>.06 (122)</td>
<td>-.03 (116)</td>
</tr>
<tr>
<td>Conflict w/ others</td>
<td>.08 (122)</td>
<td>.05 (116)</td>
</tr>
<tr>
<td>Higher power/evil</td>
<td>.15 (121)</td>
<td>.20 (116)*</td>
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<tr>
<td>Environment</td>
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<td>-.04 (116)</td>
</tr>
<tr>
<td>Intrapersonal</td>
<td>.20 (120)*</td>
<td>.21 (116)*</td>
</tr>
</tbody>
</table>

*Note: Participants were allowed to answer “not observed/don’t know” for the controllability attributions and these were excluded from analyses. N for each set of variables is in parentheses.*

* $p < .05$. 
Effects of Relationship Variables on Controllability Attributions

The type of relationship that a participant had with an individual with depression affected the ratings for controllability of symptoms. An analysis of variance, using four groups of relationship (spouse or parent, child or sibling, other type of relationship, do not know anyone with depression), showed significant differences in ratings for controllability of symptoms by type of relationship to a depressed individual, $F(3, 179) = 3.25, p < .05$. Tukey's post hoc testing indicated that those who evaluated a parent or partner ($M = 2.10, SD = .09$) had significantly higher ratings for controllability than those who evaluated a child or sibling, ($M = 1.80, SD = .12$).

Severity of episode and global controllability ratings were negatively correlated, $r(122) = -.18, p < .05$. In other words, when severity was rated higher, ratings of beliefs that individuals could control their symptoms were lower. This relationship was tested for curvilinearity. The results were nonsignificant, $p > .05$.

Effects of Other Participant Variables

Analyses were conducted to look at ratings of controllability attributions, absolute ratings of causal beliefs, and relative percentages of causal beliefs comparing across age, gender, and educational level. Because there were only six respondents between the ages of 17-21, these cases were collapsed into the category with those aged 22-30. There were no significant differences in global controllability attributions by age, $p > .05$. When analyzing absolute causal ratings, the belief that family of origin conflict was the cause of depression had significant differences by age, $F(3, 201) = 4.83, p < .01$, with participants aged 17-30...
(M = 4.06, SD = .75) and 41-50 (M = 3.98, SD = .84) having ratings significantly higher
than those aged 51 and over (M = 3.48, SD = .93). No differences by age were found when
exploring relative percentage of causal belief or controllability attributions, \( p > .05 \).

Analyses by age were repeated after removing those who did not personally know
anyone with depression. Again, family of origin conflict was significantly different by age,
\( F(3, 132) = 3.17, \ p < .05 \). Post hoc testing revealed that those aged 41-50 (M = 4.16,
SD = .76) gave a higher rating for causation attributed to family of origin conflict than the
group aged 51 and over (M = 3.66, SD = .88). There were no other significant differences by
age for causal or controllability attributions.

Differences in ratings for global controllability by sex were significant, \( F(1, 181) =
8.22, \ p < .01 \), with men (M = 2.30, SD = .68) rating the symptoms of depression as more
controllable than did women (M = 1.98, SD = .68). For only those who knew someone with
depression, the differences were not significant but continued to trend towards men
(M = 2.33, SD = .75) attributing more control than women (M = 2.01, SD = .74), \( F(1, 120) =
3.89, \ p = .051 \). For absolute ratings of causal attributions, there were significant differences
by sex for biogenetic causes, \( F(1, 202) = 5.44, \ p < .05 \), with women (M = 3.96, SD = .94)
being more likely to give higher percentages of biogenetic causation than were men
(M = 3.60, SD = 1.18). For relative percentages of causal attributions, there were significant
differences by sex for chance, \( F(1, 195) = 12.62, \ p < .001 \), and conflict with others,
\( F(1, 196) = 5.29, \ p < .05 \). Men (M = 3.99, SD = 6.64) were more likely to give higher
percentages of causation to chance than were women (M = 1.52, SD = 3.10) and more likely
to assign higher percentages of causation to conflict with others (M = 15.97, SD = 13.18;
M = 11.91, SD = 10.61, respectively).
After removing participants who did not know anyone with depression, analyses for
differences by sex were conducted. Measurable gender differences occurred solely within the
variable assessing the relative percentage of causation for chance, $F(1, 128) = 8.25, p < .01$,
with men ($M = 4.22, SD = 7.00$) assigning a higher percentage of causation to chance than
did women ($M = 1.62, SD = 3.22$).

Because only one case reported an educational level below high school and seven
participants had only a high school education, these cases were combined with those who had
some college education or an Associate’s degree. There were no differences in global
controllability attributions or relative percentages of causal attributions by level of education,
$p > .05$. Absolute ratings for causation showed significant differences by educational level
for conflict within the family of origin, $F(3, 201) = 3.95, p < .01$, and conflict with others,
$F(3, 200) = 3.11, p < .05$. Tukey’s post hoc testing for family of origin conflict revealed that
those with less than a high school education or some college ($M = 3.92, SD = .95$) were
significantly different than those with a graduate degree ($M = 3.49, SD = .97$). The lowest
educational group gave significantly more weight to family of origin conflict than did the
group with the most education. Post hoc testing for conflict with others revealed that
differences for the educational categories were not significant after removing the variance
accounted for by the other categories in the variable. After removing all cases in which
participants did not know anyone with depression, there were no significantly different
attributions by educational level.


**Conclusions**

The results support the hypothesized differences between causal and controllability attributions. It appears from the analyses that the relationship between causal attributions and controllability beliefs varies by type of causation. Although some causal beliefs were related to controllability beliefs, this relationship did not exist across all etiologies. In addition, results suggest that participant variables (type of relationship to a depressed individual and the severity of the depression of the person that they know) are related to respondents’ beliefs concerning the amount of control the individual can exert over the symptoms of depression. Contrary to predictions, no curvilinear relationships were found.

The data were examined for differences between those who know and those who do not know someone with depression. There were no significant differences between groups for beliefs in the overall amount of control that an individual exerts over depressive symptoms. Because causal attributions were rated in two different manners (absolute ratings and relative percentages), both sets of results were examined for differences. For absolute ratings, those who knew someone with depression were significantly more likely to believe biogenetic factors, family of origin conflict, and conflict with others caused the depression. For relative percentage ratings, those who knew someone were only more likely to rate family of origin conflict higher as a cause for depression.

The difference in these results suggests that the manner in which a question is asked influences the way that individuals report their beliefs about causation. For example, when exploring absolute ratings, there were significant differences between those who knew someone with depression and those who did not. However, some of these differences were
not apparent when studying relative percentages of causal beliefs. Large standard deviations among the causal percentage ratings suggest that there was great variability in responses, with somewhat more variability among those who knew someone with depression. However, we may conclude that conflict, particularly family of origin conflict, may be more salient to those who know someone with depression.

Certain types of causal attributions appear related to beliefs about the controllability of symptoms. The relationship between causal and controllability attributions had similarities whether analyzing the entire sample or only those who knew someone with depression. The belief in chance as the cause was correlated with ratings for controllability of symptoms when examining absolute causal ratings. When analyzing relative causal percentages, belief in biogenetic or intrapersonal variables were related to controllability beliefs. Subjects who rated intrapersonal causation higher were more likely to believe individuals can control depressive symptoms while those who rated biogenetic variables high as a cause of depression were more likely to believe individuals have less control over their symptoms. When analyzing only those who knew someone with depression, the relationship between absolute ratings and controllability included intrapersonal factors as well as chance, while percentage ratings added belief that a higher power/evil was the cause as well as biogenetic and intrapersonal variables.

These results suggest that the relationship varies between the causes to which people attribute depression and how much controllability they believe people have over the symptoms of depression. In addition, there were differences in the relationship based on how causal attributions were measured. These results provide some evidence that certain etiologies (chance, biogenetic, intrapersonal, higher power/evil) are likely to produce specific
beliefs about controllability; but the relationship between controllability and causal attributions does not exist across all causes. Therefore, we cannot make assumptions concerning controllability based on causal beliefs. This supports Brickman et al.'s attributional theory (Brickman, Rabinowitz, Karuza, Coates, Cohn, & Kidder, 1982), which suggests that there is a difference between responsibility for the origin of a problem and responsibility for the solution to a problem. This is not consistent with presuming that a belief in genetic factors is an indication that the person has no control over the outcome of their illness.

This study also provided evidence that beliefs concerning the controllability of depressive symptoms are associated with several aspects of an individual’s relationship with a depressed person. Those who had a parent/partner with depression believed this person had more control over depressive symptoms than did those who had a child/sibling. This may be a function of the type of relationship or due to the age differences of the depressed person.

In addition, severity of depression was associated with controllability beliefs. The higher a participant rated the severity of depression in the person that they knew, the lower were the beliefs that the person could control the depressive symptoms. This finding may help to explain varying results in previous attribution research (Casten, Rovner, Semuely-Dulitzki, Pasternak, Pelchat, & Ranen, 1999; Hooley & Licht, 1997; Licht, 2001). The study that linked controllability attributions to remission of depression used inpatients with more severe depression while the other studies were composed of individuals with less severe depression. However, caution is advised because severity ratings in this current study were subjective and may not be accurate ratings of the severity of depression. The ratings of
severity may have been affected by the respondent’s belief that individuals cannot control their symptoms.

In addition, because the length of time that a participant knew someone with depression and the number and length of episodes of depression were unrelated to controllability attributions, we may presume that attributions do not change over time. Although these results suggest that attributions do not change over time, this study utilized a cross-sectional design; a longitudinal analysis may obtain different results. It should also be noted that controllability ratings, in general, were quite low. Most participants do not believe that individuals have much control over depressive symptoms.

Further analyses examined differences in attributions by age, sex, and educational level. Differences by age when the whole sample was analyzed suggested that those 17-30 and 41-50 rated conflict within one’s family of origin as a cause for depression higher than did those aged 51 and over. After removing those who did not know anyone with depression, differences for family of origin conflict remained, with those aged 41-50 attributing higher causation to this factor than did those aged 51 and over. This suggests that conflict may be higher in families in this age group. Consistent with previous research (Hooley & Campbell, 2002), there was no relationship between age and controllability attributions.

An analysis of the entire sample revealed that men believed symptoms of depression are more controllable than did women. Contrary to previous research (Hooley & Campbell, 2002), there were no significant differences between sexes for only those who knew someone with depression, although the trend continued with men being more likely to attribute control than women. For absolute ratings of causality, women were more likely to rate biogenetic factors higher than men. Differences by sex also appeared when respondents assigned
relative percentages to the seven etiologies, with males being more likely to believe chance and conflict with others play a larger causal role than do women. However, after removing participants who did not know anyone with depression, the only measurable gender difference was for the causal variable chance, with men assigning a higher percentage of causation to chance than did women. It appears that, in general, men are more likely to expect individuals to be able to take control of their depression even if they are not responsible for the cause of the depression.

Finally, difference by educational level for the entire sample showed that those in the lowest educational group gave significantly more weight to family of origin conflict as a cause for depression than the group with the most education. There were no differences by level of education among only those who knew someone with depression.

*Implications for Future Research*

This research presents a new dimension from which to explore the relationship between attributions and relapse or remission of depression. A common assumption in previous research was that beliefs regarded as internal causation result in higher attributions for control, while beliefs regarded as external causation resulted in lower attributions for control. If this were true, we would expect to see correlations between all causal and controllability attributions. However, the results of this study suggest otherwise. Because there is no reliably significant relationship between all causal and controllability attributions, it may prove important to identify which types of attributions are being measured. Previous studies coded attributions extracted from interviews only if the statement contained a causal attribution. Attributions of control may not have been extracted if they were not related to a causal statement. In addition, many studies did not distinguish between control over the
cause of the illness or the resulting symptoms/behaviors. Previous work can be reassessed in light of the results of this study and may help to clarify the relationship between EE, attributions, and the course of mental illness.

In addition, the lack of a strong association between causal and controllability attributions points to the care that must be taken when measuring attributions. The measurement of different constructs may result in conflicting results for various studies. The discrepancy in results among the attribution literature may be due to variations in the attributions under study. For example, coding a remark in an interview as uncontrollable because the individual commented on an environmental etiology such as unemployment may be misleading. This study showed that there is no relationship between the belief in environmental causation and the amount of control depressed individuals are expected to exert over their symptoms.

Furthermore, the major findings concerning controllability attributions—the association with severity of depression and type of relationship—suggest that psychologists should consider these factors when researching the relationship between depression and attributions. This suggests that when studying attributions, the level of severity of depression should be considered. Using mean ratings across all participants regardless of severity may obscure results. Another factor that causes variability in ratings for attributions is the relationship between the rater and an individual with depression. This study suggests that those with a partner or parent with depression attribute more controllability to their relative than do those with a child or sibling with depression.

However, we still do not know whether controllability attributions are related to risk of relapse or remission of depression. It is suggested that future studies consider measuring
attributions for controllability of current symptoms separate from controllability of the origin of the problem to determine which are most salient to relapse or remission of depression. In addition, future studies may want to consider controlling for type of relationship, severity of depression, and the gender of the participants.

Limitations of the Study

In addition to factors noted above, this research was considered an exploratory study. Therefore, the results need to be interpreted with caution. Many of the participants in this study may have reported retrospectively since the survey did not request only responses from those who currently knew someone having a depressive episode. Responses concurrent with a known person's depressive episode may result in different responses. In addition, this sample was drawn from within a university setting. Cross-validation using a broader community sample may confirm or disconfirm the present study's findings.
References


Attributions Others Assign to Depressed Individuals


Appendix A

SAMPLE DEPRESSION ATTRIBUTION QUESTIONNAIRE

We all get sad from time to time, and many people say they are depressed. But clinical depression is a persistent depressed mood that interferes with daily functioning. Your answers on this survey can give us a better understanding of depression.

**Thank you for your help!**

Please answer the following questions on the scantron with a number 2 pencil.

1. What is your age?
   A) 17-21  B) 22-30  C) 31-40  D) 41-50  E) 51+

2. What is your race or ethnic group?
   A) White, non-Hispanic  B) Black  C) Latino/Hispanic  D) Asian
   E) Other, please specify: _______________________

3. What is your gender?
   A) Male  B) Female

4. What is the highest level of education you have completed?
   A) Less than high school
   B) High school
   C) Some college or Associate’s degree
   D) Bachelor’s degree
   E) Some graduate education or certificate
   F) Graduate degree (MS or above)

5. Have you been diagnosed with clinical depression, hospitalized or placed on antidepressants for depression (not for bipolar disorder or schizophrenia)?
   A) Yes  B) No

6. Do you know anyone who has been diagnosed by a doctor or mental health professional with clinical depression, hospitalized or placed on antidepressants for depression (not for bipolar disorder or schizophrenia)?
   A) Yes  B) No --If no, please skip to question 17.

   If you know more than one person, please think of the person whose depression is or was the most severe.

7. What is this person’s relationship to you?
   A) Parent
   B) Sibling
   C) Child
   D) Spouse/partner
   E) Other relative
   F) Friend
   G) Acquaintance/coworker
   H) Other, please specify: _______________________


8. What is this person's gender?
   A) Male    B) Female

9. How close do you feel to this individual?
   A) Not at all close
   B) Somewhat close
   C) Moderately close
   D) Very close
   E) Extremely close

10. How long have you known that this person suffers from depression?
    A) Less than 6 months
    B) 6 months - 1 year
    C) 1-2 years
    D) 2-5 years
    E) 6-10 years
    F) 10+ years

11. How many episodes of depression has this person had since you’ve known about their depression?
    A) 1 relatively brief, clearly defined episode
    B) 2 episodes
    C) 3
    D) 4
    E) 5+
    F) Continuous

12. What was the typical length of this person's depressive episodes?
    A) 2 weeks
    B) 2-4 weeks
    C) 1-2 months
    D) 3-6 months
    E) 7 months-1 year
    F) More than 1 year

13. What was the severity of this person's most recent episode?
    A) Very mild
    B) Mild
    C) Moderate
    D) Severe
    E) Very severe
14. Have you lived with this person while they had a depressive episode?
   A) Yes      B) No

15. Do you feel responsible for the depression?
   A) Not at all
   B) A little
   C) Moderately
   D) A lot
   E) Completely

16. Has this person ever had a suicide attempt?
   A) Yes      B) No

Listed below are some symptoms that are associated with depression. Rate how much control you believe the depressed person that you know has over each symptom. If you don't know anyone personally with depression, rate each statement as you believe it would apply to most individuals with depression.

Please answer on the scantron using a number 2 pencil. Also please make sure you begin with item 17 on the scantron if you did not answer questions 7 - 16.

   A - Not observed/don't know
   B - No control
   C - A little control
   D - Moderate control
   E - A lot of control
   F - Complete control

17. Crying a lot.
18. Irritability, complaining.
19. No interest in activities that were previously enjoyed.
20. Significant increase or decrease in appetite.
22. Sleeping all the time.
23. Being so tired, that he or she won't get out of bed.
24. Fatigue that keeps normal activities from being accomplished.
25. Talk of worthlessness, hopelessness, or inappropriate guilt.
26. Inability to concentrate or make decisions.
27. Talking of death or suicide.
29. Delusions or hallucinations (hears or sees things that others don't).
30. Complaining of body aches and pains.
31. Social withdrawal from others.
There are many theories that attempt to explain what causes depression. Please think about the following items and rate the extent to which you agree with each statement using the following scale. In particular, think about whether each statement applies to the person you know with depression. If you don't know anyone, rate each statement as you believe it would apply to most individuals with depression.

Please answer on the scantron using a number 2 pencil.

A - Strongly Disagree
B - Disagree
C - Neutral/undecided
D - Agree
E - Strongly Agree

Clinical depression is due to:

32. genetic/biological factors.
33. chance/bad luck.
34. family of origin conflicts (conflict within the family they grew up in).
35. conflicts with peers, romantic partners, children, etc.
36. a higher power/evil working in the world.
37. environmental difficulties (e.g. stress, unemployment, illness, death of a loved one, trauma, etc.)
38. intrapersonal problems- something in particular about the individual (e.g. personality, poor choices, maladaptive cognitions, insufficient effort, unfulfilled desires).

Please estimate the percentage to which each of the causes listed below is responsible for depression in the person that you know (or for most depressed individuals if you do not personally know anyone with depression). Some items may be rated as 0% if you believe that they are not at all responsible for causing depression. However, make sure you assign a percentage to each item and that the numbers total 100%. Write the percentages in the blanks provided on this sheet.

_____ 40. Chance/bad luck.
_____ 41. Family of origin conflicts (conflicts within the family they grew up in).
_____ 42. Conflicts with peers, romantic partners, children, etc.
_____ 43. A higher power/evil working in the world.
_____ 44. Environmental difficulties (e.g. stress, unemployment, illness, death of a loved one, trauma, etc.)
_____ 45. Intrapersonal problems- something in particular about the individual (e.g. personality, poor choices, maladaptive cognitions, insufficient effort, unfulfilled desires).

100% TOTAL

Thank you for your participation!
### Factor Analysis of Controllability Items

<table>
<thead>
<tr>
<th>Item</th>
<th>Component 1</th>
<th>Component 2</th>
</tr>
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<tr>
<td>Crying</td>
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<td>.12</td>
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<td>Irritable</td>
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<td>.30</td>
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<td>Appetite</td>
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<td>.51</td>
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<td>Insomnia</td>
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<td>.57</td>
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<td>Sleeping</td>
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<td>Concentration</td>
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<td>Aches</td>
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<td>Withdrawal</td>
<td>.58</td>
<td>.57</td>
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Table B2

Correlations between Relationship Variables and Controllability Attributions.

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<thead>
<tr>
<th>Symptom</th>
<th>Close</th>
<th>Time Known</th>
<th>Episodes</th>
<th>Length of Episode</th>
<th>Severity</th>
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<td>.07 (123)</td>
<td>.07 (122)</td>
<td>.01 (117)</td>
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<td>.02 (118)</td>
<td>.04 (117)</td>
<td>.03 (113)</td>
<td>-.20 (117)*</td>
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<td>.09 (125)</td>
<td>-.03 (124)</td>
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<td>.04 (124)</td>
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<td>Appetite</td>
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<td>-.16 (119)</td>
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<td>Insomnia</td>
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<td>-.01 (106)</td>
<td>-.10 (102)</td>
<td>-.26 (106)**</td>
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<td>Sleeping</td>
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<td>.10 (114)</td>
<td>.21 (113)*</td>
<td>.14 (110)</td>
<td>-.04 (113)</td>
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<td>Tired</td>
<td>.08 (111)</td>
<td>.16 (113)</td>
<td>.06 (112)</td>
<td>-.03 (108)</td>
<td>-.16 (112)</td>
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<tr>
<td>Fatigue</td>
<td>.03 (114)</td>
<td>.14 (116)</td>
<td>.13 (115)</td>
<td>.00 (111)</td>
<td>-.09 (115)</td>
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<td>Hopelessness</td>
<td>.06 (122)</td>
<td>.07 (124)</td>
<td>.05 (123)</td>
<td>.04 (118)</td>
<td>-.06 (123)</td>
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<td>Concentration</td>
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<td>.18 (125)*</td>
<td>.06 (124)</td>
<td>.06 (120)</td>
<td>-.18 (124)</td>
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<td>.02 (102)</td>
<td>.04 (98)</td>
<td>-.11 (102)</td>
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</tr>
<tr>
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<td>.00 (75)</td>
<td>.00 (71)</td>
<td>-.16 (75)</td>
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<td>Withdrawal</td>
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<td>.10 (127)</td>
<td>.09 (126)</td>
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</tbody>
</table>

Note: Participants were allowed to answer "not observed/don’t know" for the controllability attributions and these were excluded from analyses. N for each set of variables is in parentheses.

* p < .05. ** p < .01.
Table B3

*Mean Global Controllability Attribution Differences by Type of Relationship.*

<table>
<thead>
<tr>
<th>Relationship</th>
<th>Parent</th>
<th>Spouse/partner</th>
<th>Child</th>
<th>Sibling</th>
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<tbody>
<tr>
<td>Parent</td>
<td>--</td>
<td></td>
<td></td>
<td></td>
</tr>
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<td>Spouse/partner</td>
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<td>Child</td>
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<td>.96**</td>
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<td>Sibling</td>
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<td>.55*</td>
<td>.41</td>
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<td>.23</td>
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<td>.31</td>
<td>.64*</td>
<td>.24</td>
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<tr>
<td>Acquaintance/coworker</td>
<td>.31</td>
<td>.54</td>
<td>.42</td>
<td>.01</td>
</tr>
<tr>
<td>Other</td>
<td>.53</td>
<td>.75*</td>
<td>.21</td>
<td>.20</td>
</tr>
</tbody>
</table>

*Note.* Mean differences were analyzed using the Least Significant Difference comparison.

* *p < .05. ** p < .01.
Appendix C

HSRC Approval Form
Title of Proposal: Attributions others assign to depressed individuals and the relationship to severity of depressive symptoms, amount of contact, and familiarity with depressed individuals.

Principal Investigator: Joan McDowell
Faculty Sponsor: Ketl Freedman-Doan

Date Submitted: 1/20/04
New ☑ Renewal ☐ Modification ☐
Committee Action: Approved ☑ Provisionally Approved ☐ Disapproved ☐ Exempt ☑

Reason(s), if disapproved: n/a
Reason(s) if provisionally approved: n/a

Comments: This application was approved by the two assigned reviewers, both of whom saw no problems with your request. One reviewer noted that because you are not collecting identifying data and are appropriately requesting a waiver the process of signed informed consent (by the use of a very nice “Letter of Introduction” which contains all the usual consent elements, except signatures), your study is exempt from continuing review. This would, indeed appear to be the case. Good luck with your research!

Substitute or additional Comments:

Signature for the Committee:

Additional Comments:

Note:
1. Investigators are obligated to advise the Review Committee of any change in protocol which might bring into question the involvement of human subjects in a manner at variance with the considerations on which the prior approval was based.
2. For ongoing studies, every 12 months from the date of this approval -- or at shorter intervals when specified by the Committee -- the investigator must submit the protocol and a progress report to the Committee for re-review. (N/A for THIS EXEMPTED STUDY - KKS)
3. Investigators are required to immediately suspend any study in which he/she observes an unanticipated negative change in the health or behavior of a subject that may be attributable to the research. The investigator must report the circumstances promptly to the Review Committee for its further review and decision on continuation or termination of the project.