

ATTACHMENT PROCESSES, STRESS PROCESSES AND SOCIOCULTURAL
STANDARDS IN THE DEVELOPMENT OF EATING DISTURBANCES
IN COLLEGE WOMEN

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Minimal empirical research using longitudinal data to explore integrative models of eating disorder development exists. The purpose of this study was to further explore multidimensional models incorporating parental attachment, history of stress, appraisal/coping processes, internalization of the thin-ideal, negative affect, body image, and eating disordered behavior using prospective, longitudinal data. The models were evaluated using 238 participants who completed an initial series of self-report questionnaires during their first semester in college and completed follow-up questionnaires 6 months and 18 months later. Structural equation modeling was used to examine the relationships among the factors. Analyses confirmed that college freshman with insecure parental attachment relationships and those with a history of previous stressful experiences appraised the adjustment to college as more stressful and reported feeling less able to cope with the transition; these conditions predicted increased negative affect and increased eating disturbances. Women who reported experiencing negative affect and those that endorsed internalization of the thin-ideal also reported higher levels of body dissatisfaction; these women engaged in more disordered eating attitudes and behaviors. A second model investigating negative affect as mediating the relationship between the appraisal/coping process and eating disturbances also revealed that experiencing difficulties with the transition to college predicted later negative mood states. Further, women who reported increased negative affect also reported increased eating disturbances. Finally, cross-lagged and simultaneous effects between selected factors were evaluated. Results from these analyses are mixed, but they provide additional information about the predictive relationships among factors that play a role in the

development of eating disorders. The results of this study provide valuable information about the development of eating disorders that can be used to aid prevention and treatment. Examination of these models in a large independent sample might provide confirmation of these relationships, and investigation of the models during different developmental periods might also provide important information about the development of eating disturbances and those individuals who are most at risk.

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CHAPTER 1

INTRODUCTION

Definition, Epidemiology, and Onset

As recently as 25 years ago, it was difficult to define anorexia nervosa and bulimia nervosa (Costin, 1996). Now, college campuses and clinical settings treat these disorders on a regular basis (Kenny & Hart, 1992). Currently, anorexia nervosa is defined as the refusal to maintain body weight at a normal and healthy level based on age and height, an exaggerated fear of gaining weight, and a profound misinterpretation of body size and shape which influences self-evaluation. In women, the absence of a regular menstrual cycle, amenorrhea, is also seen (American Psychiatric Association [APA], 2000). Bulimia nervosa, on the other hand, is characterized by continual episodes of binge eating during which the individual consumes an excessive amount of food in a discrete time period and feels a lack of control over this behavior, engagement in recurrent and compensatory behaviors designed to prevent any weight gain, and self-evaluation based predominately on a disturbed perception of their body shape and weight (APA, 2000).

Since 1970, there has been a dramatic increase in eating disorders (Kenny & Hart, 1992), with more than 90% of the cases occurring in females. Additionally, it is estimated that 64% of college women show some symptoms of eating disordered behavior (Mintz & Betz, 1988). Recent prevalence studies have found that 0.5 – 1.0% of females meet the full criteria for anorexia nervosa (APA, 2000); whereas 5% of young women report sub-threshold symptoms of this disorder (Kaplan & Sadock, 1998). Bulimia nervosa has been shown to be more prevalent than anorexia nervosa, with estimates ranging from 1-3% of women (APA, 2000), yet occasional symptoms of this disorder have been reported in up to 40% of college women (Kaplan &

Sadock, 1998). Both disorders appear to be more prevalent in industrialized countries, and although incidences of bulimia do occur in other ethnic groups, it appears to be more prevalent in Caucasian women (APA, 2000).

The typical onset for anorexia nervosa occurs between the ages of 10 and 30, with 17 to 18 being the mean age of onset (APA, 2000; Kaplan & Sadock, 1998). It is estimated that 85% of individuals diagnosed with anorexia nervosa experience an onset of the disorder between the ages of 13 and 20 (Kaplan & Sadock, 1998). Additionally, the onset of anorexia nervosa is often associated with a stressful life event (APA, 2000). Bulimia nervosa usually begins in late adolescence or early adulthood, often with the binge eating coinciding with an episode of dieting (APA, 2000).

Etiology

From a developmental perspective, psychopathology is viewed as emerging from a dynamic relationship among social, emotional, and cognitive domains of functioning (Smolak, 1996). These domains are molded by behavioral, sociocultural, environmental, biological, and psychological variables. However, even with an abundance of research focused on advancing and understanding the development of eating disturbances, the exact pathogenesis of an eating disorder still remains unclear (Stice, 2001b). Currently, sociocultural standards of attractiveness, familial functioning, and stress are identified as important areas in the etiology of eating disorders (Bennett, Cooper, & Cooper, 1999; Zerbe, 1993).

The majority of approaches to understanding eating pathology tend to focus on single etiological factors, assuming a direct link between a specific predisposing factor and the onset of eating disordered symptomatology (O’Kearney, 1996; Stice, 2001b). Multidimensional models

of eating disorder development represent an improvement, because they attempt to understand biological, familial, sociocultural and individual variables in both etiology and maintenance. Unfortunately, these models are not integrative, and they often ignore the process in which the factors influence each other and culminate in eating pathology (Garfinkel & Garner, 1982; Stice, 2001b). Although it is likely that multiple factors motivate individuals to starvation, bingeing, and purging, the relevance of each variable must be examined to develop an inclusive model of the etiology of eating disturbances. Finally, a review of the current literature yielded minimal empirical research using longitudinal data to examine integrative models of eating disorder development (e.g., Ghaderi, 2003; Stice, 1998; Thompson, Coover, Richards, & Johnson, 1995). Thus, there is a need for more studies that investigate multidimensional risk factors for the development of eating disorders using longitudinal data (Ghaderi, 2003). The following sections cover theoretical frameworks and existing models as a basis for a hypothesized model integrating developmental, environmental, and sociocultural factors using prospective data.

Sociocultural Standards of Attractiveness and Internalization of the Thin-Ideal

Although the exact pathogenesis of an eating disorder remains unclear, sociocultural pressures are believed to influence both the development and the maintenance of eating disordered behavior (Garfinkel & Garner, 1982; Johnson & Connors, 1987; Stice & Shaw, 1994; Striegel-Moore, Silberstein, & Rodin, 1986). Some theorists even propose that the recent rise in eating disorders is mostly due to sociocultural influences (Bordo, 1993; Gordon, 1990; Wolf, 1991). The sociocultural model strives to connect eating disturbances with cultural trends that promote thinness as an essential characteristic of physical attractiveness.

Thinness is not simply equated with beauty. It has also become a symbol of success (Silverstein & Perdue, 1988). Describing the female body as a “message board” for cultural demands to be enacted as women fall victim to the ever-changing societal messages that communicate the preferred look and behaviors that give one status and esteem, Brumberg (1997) compared the diaries of adolescent females and found that women at the turn of the nineteenth century equated “goodness” with moral purity whereas adolescent women at the end of the 20th century seemed to equate “goodness” with thinness. Therefore, not only is society promoting a thin ideal, but it is also sending the message that being thin is important. The mechanism by which this idea affects the development of disturbed eating behaviors can be explained by Frederickson and Robert’s (1997) objectification theory. Based on the premise that men’s and women’s bodies are treated differently by the dominant culture, they suggest that the female body is “looked at, evaluated, and always potentially objectified (pp.175).” Yunker and Allison (1994) reported that females pay higher consequences than males do in terms of dating, education, and employment opportunities when they fail to meet societal ideals. Thus, as women are socialized to believe that others are observing and evaluating them, holding the power for social, educational, and occupational advancement, many women may see attractiveness as the key to success, choosing to pursue society’s thin-ideal body as a means of achieving the attractiveness they believe is associated with that success (Frederickson & Robert, 1997).

Family members and peers appear to be influential sources of the sociocultural message that body shape, slenderness, and perfectionistic achievement are important. Referring to Kandel’s socialization theory (Kandel, 1980; Kandel & Davies, 1992), Stice (1998) suggested that social reinforcement (criticism/teasing regarding weight or body shape, encouragement of weight control behaviors) and social modeling (performing weight control behaviors) may be

two processes by which family members and peers support and perpetuate sociocultural standards.

In support of this hypothesis, research suggests that parental reinforcement of the thin-ideal is related to eating pathology. Thelen and Cormier (1995) demonstrated a positive correlation between parental pressure to lose weight and eating disturbances. Similarly, family pressure to be thin was positively correlated with symptoms of bulimia (Irving, 1990; Stice, Nemeroff, & Shaw, 1996). Another study found that weight- and/or shape-related teasing by family members contributed to modest but significant increases in body dissatisfaction, weight management behaviors, investment in thinness, and eating disturbance (Levine, Smolak, & Hayden, 1994). Furthermore, mothers with daughters exhibiting high scores on measures of disordered eating were found to express more criticism of their daughter's weight and attractiveness than mothers of daughters who obtained low disordered eating scores (Moreno & Thelen, 1993; Pike & Rodin, 1991). In addition, adolescent girls reported being more likely to engage in dieting behaviors when encouraged by their parents (Paxton et al., 1991), and perception of parental investment in their daughter's weight was found to correlate positively with the adolescent girl's dieting behavior, fear of becoming fat, preoccupation with food, purging behavior, and interpersonal struggle with eating (Levine, Smolak, Moodey, Shuman, & Hessen, 1994).

Parental modeling of dieting attitudes and behaviors may also communicate the societal message that thinness is important, particularly for young women. Research indicates that high school girls who reported having a parent who diets were more likely to engage in dieting behaviors (Paxton et al., 1991). In addition, mothers with daughters reporting higher levels of eating disordered symptomatology reported more dieting behaviors and disordered eating

attitudes than control mothers (Pike & Rodin, 1991). However, some studies suggest conflicting results regarding parental modeling. Attie and Brooks-Gunn (1989) failed to find a relationship between mothers and daughters on measures of compulsive eating and body dissatisfaction, and another study reported no difference between control parents and parents of eating disordered adolescents on measures of dietary restraint and disturbed eating (Leon, Fulkerson, Perry, & Dube, 1994; Steiger, Stotland, Ghadirian, & Whitehead, 1995). Conflicting results regarding the association between parental modeling and eating disturbances suggest that although parental modeling of dieting behaviors may adversely affect some young women, parental reinforcement of sociocultural pressures may be a stronger risk factor in the development of eating disorders.

Research also suggests that peers reinforce and model sociocultural pressures to be thin. Compared to controls, Stice, Ziemba, Margolis, and Flick (1996) found that bulimic women perceived greater pressure to be thin from their peers, and bulimics indicated initiation of bingeing and purging behaviors following perceived peer pressure to lose weight (Mitchell, Hatsukami, Pyle, & Eckert, 1986). Teasing by peers is also strongly related to body dissatisfaction (Cattarin & Thompson, 1994; Levine, Smolak, & Hayden, 1994) and marginally associated with purging behaviors, changing eating patterns around peers, and attempts to emulate female actresses and models, although it is unknown if these were attempts to emulate body shape or fashion style (Field, Camargo, Taylor, Berkey, & Colditz., 1999). In addition, peers appear to be a significant source of information regarding sociocultural standards of attractiveness that may contribute to unhealthy weight and eating attitudes and behaviors (Chiodo & Latimer, 1983; Desmond, Price, Gray, & O'Connell, 1986; Levine & Smolak, 1992; Levine, Smolak, & Hayden 1994; Parker, Nichter, Nichter & Vuckovic, 1995).

In a comprehensive study, Stice (1998a) evaluated both family and peers as sources of social reinforcement and social modeling. He reported that family and peer reinforcement and modeling were associated with symptoms of bulimia. Furthermore, social reinforcement and modeling prospectively predicted bulimic pathology, suggesting that family members and peers are important influences of the sociocultural standard of thinness. Hamilton, Brooks-Gunn, and Warren (1985) suggest that pressure from these subcultures greatly increases the occurrence of pathological eating and weight management behaviors. Further, even a moderate level of pressure carries significant risks, physical and psychological, for adolescents (Rosen, Gross, & Vara, 1987).

Although family and peers are seen as influential in promoting the thin-ideal, research often focuses on the role the media plays in communication of this standard. With easy access and increased use of various media forms (Kilbourne, 1999; Roberts, Foehr, Rideout, & Brodie, 1999) to influence attitudes and teach behaviors to young women (Huston et al., 1992), the media, interacting with other sources of societal values, is seen as one of the most influential reinforcers of the sociocultural standard of thinness (Stice, 2001b, Stice and Shaw, 1994; Striegel-Moore et al., 1986).

Although American women have gotten heavier, popular media has presented a statistically significant thinning of the ideal body (Garner, Rockert, Olmstead, Johnson, & Corscina, 1985; Silverstein, Purdue, Peterson, & Kelly, 1986; Wiseman, Gray, Mosimann, & Ahrens, 1992). Researchers examining popular media have reported increases in the rates of anorexia and bulimia in the U.S., coinciding with media portrayal of decreasing ideal body weight over the past several decades (Garner, Garfinkel, Schwartz, & Thompson, 1980; Pyle, Halvorson, Neuman, & Mitchell, 1986; Szmukler, McCance, McCrone, & Hunter, 1986;

Wiseman et al., 1992). Additionally, the media has led women to believe that dieting, exercising, and other techniques to control weight will enable them to achieve this portrayed “ideal thinness” (Polivy & Herman, 2002). Like the increase of thin women in the media since 1950, research has also shown an increase in weight loss related advertisements (Snow & Harris, 1986). Garner et al. (1980) reported an increase in the number of articles on dieting and exercise programming that appeared in popular women’s magazines. Consistent with previous research (Paxton et al., 1991) and marketing surveys (Teen-Age Research Unlimited, 1989), Levine, Smolak, and Hayden (1994) reported that 60% of middle school females enjoyed regularly reading at least one magazine. More importantly, these young women considered the magazine an important source of information regarding body shape, dieting, fitness, and general beauty.

Jhally (2000) estimated that only 5% of females possess the body build necessary to pursue fashion modeling. Even fewer women have the approximate weight, height, and body shape necessary to meet the standards of the fashion modeling industry (Wolf, 1991). With few women having the capacity to obtain society’s ideal-body and society suggesting that this feat can and should be accomplished, many women resort to extreme methods to control and mold their bodies to conform to these ideals. Thus, eating disorders may be viewed as an overadaptation to cultural pressure to achieve a different weight and shape, an extreme condition on a continuum of dieting, exercise, and weight preoccupation (Pike & Rodin, 1991; Rodin, Silberstein, & Striegel-Moore, 1985; Stice & Shaw, 1994).

In support of this hypothesis, research has documented a relationship between exposure to sociocultural pressures to achieve the thin-ideal and eating disorder symptomatology. Stice, Shupak-Neuberg, Shaw, and Stein (1994) found that exposure to ideal body images was significantly correlated with measures of eating pathology. Additional research on media

modeling has found that bulimic women reported learning to binge and purge as methods of weight control via media sources (Chiodo & Latimer, 1983; Fairburn & Cooper, 1982). Despite the compelling evidence concerning the pervasive influence of sociocultural factors, only a proportion of women develop disturbed eating behaviors. Thus, it is likely that other factors are involved in the process.

Stice and Shaw (1994) suggested several variables that may mediate the relationship between exposure to ideal body images and the development of disturbed eating. One way sociocultural pressures to achieve a thin-ideal may promote the development of eating pathology is via negative affect. Theory suggests a relationship between emotional distress and bulimic symptomatology, hypothesizing that women use binge-eating as a method of comforting themselves (Humphrey, 1986). Similarly, purging behavior might be used to reduce feelings of shame, guilt, and depression that may result from the binge-eating episodes (Johnson & Larson, 1982). Previous research has documented that bulimics report more depression, more anxiety, and lower self-esteem when compared to non-bulimics (Bulik, Beidel, Duchmann, Weltzin, & Kaye, 1992; Dykens & Gerard, 1986; Mintz & Betz, 1988; Parmer, 1991; Shisslak, Pazda, & Crago, 1990; Vitousek & Manke, 1994; Weiss & Ebert, 1983). In addition, bulimic women reported periods of increased depression, anxiety, and feelings of inadequacy prior to engaging in binge-eating behaviors (Davis, Freeman, & Garner, 1988; Johnson & Larson, 1982; Lingswiler, Crowther, & Stephens, 1989; Steinberg, Tobin, & Johnson, 1990). Similarly, negative affect has been shown to predict the onset of bulimia (Killen et al., 1996; Stice & Argas, 1998) and increases in bulimic behaviors (Stice, 2001a). Most importantly, Stice and Shaw (1994) were able to extend the research confirming a relationship between negative affect and eating pathology, connecting negative affect to exposure to the thin-ideal. Using magazine

pictures of ultra-thin models, average-sized models, and pictures without the presence of models, Stice and Shaw found that exposure to the ultra-thin models resulted in depression, guilt, shame, insecurity, and stress. In sum, exposure to societal pressures to be thin appears to be related to the development of negative affect, a predictor of eating disorder symptomatology.

Exposure to thin-ideal images also may result in body dissatisfaction and subsequent eating pathology. Research has demonstrated a strong association between body dissatisfaction and eating pathology (Leon, Fulkerson, Perry, & Cudeck, 1993; Mintz & Betz, 1988, Stice, 1994) and increased dieting, a risk factor for the development of eating disorders (Stice, Mazzotti, et al., 2000; Stice, 2001b). More specifically, longitudinal research has shown that body dissatisfaction resulted in subsequent eating pathology (Attie & Brooks-Gunn, 1989; Striegel-Moore, Silberstein, Frensch, & Rodin, 1988). In addition, Silberstein, Striegel-Moore, Timko, and Rodin (1988) proposed that body image dissatisfaction results from the discrepancy between the perceived self and the ideal self, stating that one concept of ideal self results from society's portrayal of the ideal body. Irving's (1990) study provides empirical evidence for this proposal. Women exposed to slides of thin models displayed significantly lower body satisfaction compared to women who were exposed to slides of average or overweight models. Stice and Shaw (1994) obtained similar results using magazine pictures, and a recent study (Ogden & Munday, 1996) observed that acute exposure to media images of stereotypically attractive individuals (thin models) resulted in greater body dissatisfaction, whereas exposure to images of overweight models resulted in improved body satisfaction. Unfortunately research has not been able to confirm that exposure to the thin-ideal results in long-term effects of body dissatisfaction (Stice, Spangler, & Argas, 2001). Although exposure to the thin-ideal appears to

result in acute body dissatisfaction, additional factors may promote long term body dissatisfaction.

Stice and Shaw (1994) addressed this issue, proposing a final factor that may mediate the relationship between exposure to the thin-ideal body shape and the development of subsequent eating pathology. Sociocultural pressures to be thin may lead women to internalize a thin-ideal stereotype, which is thought to produce eating disorder symptomatology by fostering body dissatisfaction, negative affect, and dieting behavior (Stice & Argas, 1998; Thompson, Heinberg, Altabe, & Tantleff-Dunn, 1999). A small group of studies have provided empirical support for this hypothesis. Harrison and Cantor (1997) reported that media exposure using thin-ideal body content was correlated with thin-ideal internalization, body dissatisfaction, and symptoms of eating disorders. In two recent studies, Griffiths and colleagues, (Griffiths, Beaumont, et al., 1999; Griffiths, Mallia-Blanco, et al., 2000) reported that awareness and internalization of sociocultural attitudes regarding attractiveness was significantly and positively related to measures of eating pathology. Stice and Shaw (1994) found a strong positive relationship between internalization of the thin-ideal and eating pathology. In addition, multiple regression indicated that internalization of the thin-ideal, body dissatisfaction, and negative affect predicted bulimic symptoms. More importantly, experimental reduction of the thin-ideal internalization resulted in improved body satisfaction, less dieting behavior, improved affect, and reduction in bulimic symptoms (Stice, Mazotti, et al., 2000; Stice, Chase, Stormer, & Appel, 2001).

Based on support for the above risk factors, Stice (2001a; Stice, Ziemba, et al., 1996) proposed a dual pathway approach to the development of bulimia. Specifically, internalization of the thin ideal and body dissatisfaction are suggested pathways that may mediate the relationship between sociocultural pressures and eating disorders. In this model, Stice suggested that repeated

exposure to sociocultural pressures to be thin foster body dissatisfaction and internalization of the thin-ideal. Recall that research has already demonstrated an association between body dissatisfaction and eating disorder symptomatology (Attie & Brooks-Gunn, 1989; Leon et al., 1993; Mintz & Betz, 1988, Stice, 1994; Striegel-Moore et al., 1988). However, because achieving the thin-ideal is an unrealistic goal for many women, internalization also results in dissatisfaction with one's body, leading to negative affect and risky weight control behaviors (restrained eating), risk factors for development of bulimic pathology (Stice, 2001a; Stice, Ziemba, et al., 1996).

In support of the dual pathway model, Stice and Argas (1998) and Stice, Killen, Hayward, and Taylor (1998) found that sociocultural pressures to be thin, thin-ideal internalization, body dissatisfaction, dieting, and negative affect predicted the onset of bulimic symptoms. Similarly, Twamley and Davis (1999) used multiple regression analysis, establishing that internalization of the thin ideal and dissatisfaction with one's body mediated a relationship between sociocultural pressures to be thin and eating pathology. Further, in an attempt to evaluate this Model and document temporal precedence for the hypothesized mediational relations among the risk factors, Stice (2001a) conducted a study using random regression growth curve models and data from three different time periods. Results indicated that sociocultural pressures to be thin and internalization of the thin-ideal predicted increased body dissatisfaction. Initial body dissatisfaction predicted growth in dieting behaviors and negative affect, which led to increases in bulimic pathology.

Collectively, research suggests that family members, peers, and especially the media serve as influential messengers of sociocultural standards, encouraging thinness for women as a standard of both beauty and success. In addition, exposure to these messages, internalization of a

thin-ideal, body dissatisfaction, and negative affect appear to be risk factors in the development of eating disordered attitudes and behaviors. Further, it seems likely that exposure to sociocultural pressures to be thin foster an internalization of the thin-ideal, leading to a path of body dissatisfaction, negative affect, and unhealthy weight control behaviors.

Although empirical research supports a link between eating disordered behaviors and sociocultural influences, it is unlikely it is a single causal factor. The media, family, and peer pressures to be thin may provide the opportunity for a woman to develop negative beliefs regarding their body, yet the development of an eating disorder is multidimensional and other factors must be present for women to consider extreme levels of eating behaviors, exercising and other weight control techniques such that other facets of their lives are compromised. Current research has gone beyond linking exposure to the thin-ideal standard directly to the development of eating disorders, proposing that internalization of the thin-ideal, body dissatisfaction, and negative affect may mediate the relationship between exposure to the thin-ideal and the development of symptoms. Unfortunately, all women are still exposed to the sociocultural pressures to achieve an ideal body shape and have the potential to develop the mediating factors that may lead to an eating disorder. Consequently, one problem with the suggestion that sociocultural influences may be at the root of eating pathology is that they cannot predict which women will actually develop an eating disorder. Therefore, examining factors that may predispose women who are exposed to the thin-ideal to then internalize these messages is also important.

Stress Processes and the Development of Eating Disturbances

Clinical observations and empirical findings, suggest a connection between stress occurrences and the development of eating disorders. Specifically, an individual's experience of stress is thought to precede the development of anorexia (Garner & Garfinkel, 1980; Slade, 1982). Similarly, other researchers (Leitenberg & Rosen, 1988; Loro & Orleans, 1981; McManus & Waller, 1995; Muuss, 1986) believe that stress contributes to binge eating and purging behaviors. Folkman (1984) defines psychological stress as a dynamic and bi-directional relationship that exists between an individual and that individual's environment, which the individual views as taxing or exceeding his or her available resources, consequently endangering the well-being of the individual. Currently, stress is viewed as a process that includes not only a precipitating event and the response to that event, but several intervening variables. In one of the models that contributes to this view, Pearlin, Menaghan, Lieberman, and Mullan (1981) conceptualized the stress process as an interaction between sources of stress, mediators of stress, and manifestations of stress.

Keeping with Pearlin et al.'s (1981) conceptualization, Elliot and Eisdorfer (1982) outlined three categories for sources of stress: acute stressors, stress-event sequences, and chronic and chronic-intermittent stressors. Acute stressors are viewed as minor life events or time-limited experiences (e.g., a minor illness or injury), and stress-event sequences tend to be seen as major life events or those that extend over a lengthy period of time (e.g., a developmental transition). In comparison, chronic stressors have a more permanent and enduring nature, whereas chronic-intermittent stressors are analogous to daily hassles; they are not continuous, but they recur over time. Recent research examining the relationship between psychological stress and disturbed eating has primarily focused on life events (acute stressors and stress-event

sequences). Ball and Lee (2000) and Cattanach and Rodin (1988) have reviewed the literature discussing the stress process and its role in the development and the maintenance of eating disorders, noting associations between the development of eating disorder symptomatology and exposure to potentially stressful events, appraisal of the events as stressful, and coping strategies.

One hypothesis suggests that women with eating disorders have been exposed to more potentially stressful events than non-eating disordered individuals. Hawkins and Clement (1980) evaluated life events experienced by binge-eating undergraduate women in the past month and concluded that incidences of binge eating were positively correlated with the number of recently experienced life events. Greenberg (1986) and Soukoup, Beiler, and Terrell (1990) found similar results, indicating that bulimics reported a greater number of life events compared to non-bulimic women. Unfortunately, these studies were unable to distinguish whether or not the life event preceded the disordered eating behavior. In addition, they failed to reveal any information regarding stress and the development of anorexia. Studies examining precipitating events, though, shed more light on the association between anorexia and life event experience. Hamburg and Herzog (1985) reported that a traumatic event often preceded the development of anorexia or bulimia. In addition, they discovered that academic stress was linked to a worsening of the illness. Looking specifically at patients with anorexia, an early study by Crisp, Hsu, Harding, and Hartshorn (1980) reported that patients had experienced one or more stressful events prior to the onset of the disorder. However, a more consistent report is that approximately two-thirds of anorexic patients experience an environmental change or adverse life event prior to development of anorexia (Kay & Leigh, 1954; Margo, 1985; Morgan & Russell, 1975). Studies evaluating bulimic symptomatology and binge eating behavior revealed that bulimic patients also reported experiencing traumatic events and/or life changes, including sexual conflict, separation, or loss

prior to the onset of the bulimic symptoms (Lacey, Coker, & Birtchnell, 1986; Pyle, Mitchell, Eckert, 1981). Thus, many studies appear to support the contention that experience of a life event may precede or contribute to eating disordered behavior.

Additional studies report only partial support for an association between the occurrence of a potentially stressful life event and eating pathology (Gowers, North, Byram, Weaver, 1996; Horesh et al, 1995; Johnson, Stuckey, Lewis, & Schwartz, 1982; Mynors, Wallis, Treasure, & Chee, 1992; Schmidt, Tiller, & Treasure, 1993), and others lend no support for this hypotheses. For example, Gomez and Dally (1980) found that the only difference between anorexic patients and controls was an increase in family arguing. Weiss and Ebert (1983) added that the mean frequencies of life change events were not significantly different for bulimics than for controls. Thus, although many studies investigating the relationship between life events and disturbed eating have assumed that the occurrence of life events may play a role in the development of eating disorders, it is likely that other contributing factors mediate this relationship.

Some individuals react negatively to potential sources of stress, whereas others may experience positive or neutral reactions to the same event. Keeping with Pearlin et al.'s (1981) conceptualization of stress as a process, mediators such as the appraisal of the event or situation and perceptions of control may explain these individual differences as well as offer an explanation for the inconsistencies in research evaluating experience of life events and the development of adverse reactions to those events. According to Folkman, (1984) appraisal is the process by which an individual assigns meaning to the events they experience. There are two types of cognitive appraisal: primary appraisal and secondary appraisal. Primary appraisal refers to the individual's evaluation of the environment or situation, relevant to the impact it will have on their well-being, whereas secondary appraisal refers to an individual's evaluation of their

abilities and resources that might be used to manage the demands of the current situation or event (Lazarus & Folkman, 1984).

An alternative hypothesis is that an individual's perception of the life event and their perceived ability to manage the life event, rather than the actual occurrence or number of stressors, may be an important contributing factor. In their evaluation of the stress process in bulimia, Cattanaach and Rodin (1988) proposed that bulimic women appraise life events and potential stressors differently compared to other individuals, possibly viewing them as more taxing, less controllable, and less predictable, thus influencing dysfunctional coping in the form of eating pathology. This hypothesis can probably also be applied to the anorexic woman's experience of life events and potential stressors.

Consistent with this hypothesis, Soukoup et al. (1990) reported that both anorexics and bulimics scored significantly higher than controls on a measure of current perceived stress. More specifically, women at risk for developing eating disorders currently perceived higher levels of stress in their social life, home environment, school activities, and financial situations (Johnson Sabine, Wood, Patton, Mann, & Waekely, 1988; Patton, Johnson Sabine, Wood, Mann, & Waekely, 1990). In addition, high stress reactivity was found to be a strong predictor of concurrent eating disturbances in females (Leon et al., 1993).

Looking retrospectively at perceptions of stress, research shows that women displaying anorexic profiles also reported higher levels of stress than controls (Heilbrun & Flodin, 1989). Studies investigating bulimic and binge-eating symptomatology found that bulimics and severe binge-eaters had perceived significantly more stress than controls over the past year (Crowther & Chernyk, 1986). In addition, amount of stress perceived during the previous year was found to be a significant predictor of binge-eating behavior (Wolf & Crowther, 1983) and high perceptions

of stress reported at the end of one year were associated with worsening symptoms during the following year (Striegel-Moore et al., 1989). One longitudinal study (Haslam, Stevens, & Haslam, 1989) also found that the perception of stress was associated with a later increase in abnormal eating as measured by the EAT-26.

One study (Leon, Fulkerson, Perry, & Early-Zald, 1995) did not find perceived level of stress to be a significant predictor of disordered eating. However, this study assessed negative emotionality as a measure of perceived stress. It is likely that negative emotionality reflects mood or a reaction to stress, rather than actual perception of stress, and may not be as good a predictor. On the other hand, Ball and Lee (2000) add that the experience of life events and the perceptions that these events are stressful is a common experience for many young women (Cattanach & Rodin, 1988; Gomez & Dally, 1980), yet not all women experiencing these events and stressors develop eating disorders. In addition, the occurrence of life events and the experience of stress have also been linked to other forms of psychopathology and general psychological distress (Markush & Favero, 1974; Moore & Burrows, 1996). Therefore, other factors may be important in influencing the role of stress on subsequent eating pathology.

In support of the idea that coping strategies may mediate the relationship between stress occurrence, perception of stress, and the development of disturbed eating, research shows that women at risk for developing eating disorders as well as those with diagnosed eating disorders perceive themselves as less able to cope, less able to tolerate stress, and unable to effectively solve problems (Soukoup et al., 1990; Lehman & Rodin, 1989).

One possible explanation is that individuals who develop eating disorders use impaired coping strategies that prevent them from effectively managing life events and stress. Research has shown that bulimic women use more coping strategies than control women, suggesting that

they may appraise an adequate arsenal of coping resources for managing stressful situations (Katzman & Wolchik, 1984). However, Hawkins and Clement (1984) proposed that these women lack a full array of coping responses. Consequently, they may rely on only a few strategies or apply ineffective strategies. Research has shown that these women demonstrate more emotion-focused coping and avoidance-focused coping responses compared to controls (Neckowitz & Morrison, 1991; Shatford & Evans, 1986; Soukoup et al., 1990; Troop, Holbrey, Trowler, & Treasure, 1994). In addition, Katzman and Wolchik (1984) and Johnson and Larson (1982) reported that bulimic and binge-eating women displayed a passive coping style and an inability to express their feelings, characteristics associated with poor adjustment (Billings & Moos, 1981)

In summary, it appears that the occurrence of potentially stressful situations and life events may play an important role in the development of eating disorders. However, some research demonstrates a stronger connection between the perception of stressful events and appraisal of impaired tolerance for managing stress. In addition, faulty coping strategies and an impaired repertoire of coping responses may function as a mediator between stress and disturbed eating. However, little has been done to link the processes that lead from faulty coping mechanisms to the development of eating disorders. Further, it is unlikely that faulty coping mechanisms and/or an impaired repertoire of coping strategies is the only mediating link between stress and the development of eating pathology.

Attachment Processes and the Development of Eating Disturbances

Many clinicians have suggested that dysfunctional family relationships play a key role in the development and maintenance of eating disorders. Minuchin, Rosman, and Baker (1978)

along with other clinicians (Slevini-Palazzoli, 1974) described the anorectic family as rigid, enmeshed, overprotective, and characterized by poor conflict resolution, whereas the bulimic family is seen as chaotic, conflictual, and lacking in expressive communication (Head & Williamson, 1990). In a similar view, Bruch (1973) speculated that eating disturbances were related to family dynamics, including overprotective parents and a lack of responsiveness, warmth, and support for the child. Bruch suggested that the anorexic individual is torn between her emotional needs and nutritional ones, a conflict that arises from the faulty parent-child interaction in which the caregiver has demonstrated inadequate responses to the child's inner needs. Learner (1983) has added that low parental care resulting in dysfunctional working models may also contribute to the development of bulimia nervosa. One hypothesis is that food, a focus of some of the earliest and most enduring parent-child interactions, remains a symbol of nurturance throughout the child's development. Thus, individuals suffering from eating disorders who feel that their parents are less nurturing and empathetic toward them deprive and mistreat themselves as they feel that their parents have done. Bulimics turn to food as an externalized substitute for the familial sources of nurturance, whereas individuals with anorexia deprive their bodies of nutritional intake just as emotional support and nurturance is limited (Humphrey, 1986). Attachment theory may offer one perspective for examining familial factors that contribute to the development of eating disorders.

Bowlby (1969) and Ainsworth (1973) defined attachment as a long lasting emotional tie between an infant and primary caretaker. Attachment theory focuses on the nature of affectional bonds and the importance the infant-caregiver relationship has in the child's personality and social development (Bowlby, 1969). Bowlby's thoughts on attachment, loss, and separation

(Bowlby, 1969; 1973) form the foundations of the theory, whereas Ainsworth's research provides empirical support.

Drawing on ethology, control systems theory, and psychoanalytic thought, Bowlby (1988) posited that human beings seek out and form attachments with others as part of an innate growing process. The attachment process is crucial for survival of the infant, in that the function of attachment behavior is to protect the child from external sources of danger. Bowlby (1973) proposes that the attachment system serves a homeostatic function by keeping a state of equilibrium between the child and the environment. Therefore, the attachment figure serves as a secure base for the child to engage in and master his or her environment by maintaining a balance between exploratory behaviors and proximity seeking behaviors (Bowlby, 1973; Feeny & Noller, 1996).

A central idea in attachment theory is the development of the child's personality and view of the world. According to Bowlby (1969), as infants progress through their first year of life, they develop mental representations or internal working models of themselves and others based on their experiences with their attachment figures. When the attachment figure displays consistent and responsive behavior toward the child, the child develops an internal working model of self that is worthy of love and support and a belief that others are reliable and trustworthy. If the attachment figure is seen as either consistently unresponsive or inconsistently responsive, the infant is likely to form a negative working model of self and a belief that others are not to be trusted.

Building on Bowlby's theory, Ainsworth's naturalistic observations of mother-infant interactions and the examination of infant responses in a "strange situation" allowed for the development and classification of three attachment patterns: secure, avoidant, and anxious-

ambivalent (Ainsworth, Blehar, Waters, & Wall, 1978). According to Ainsworth et al., the secure attachment pattern finds the primary caregiver both accessible and responsive. The attachment relationship is seen as a secure base from which the infant can explore, yet still utilize for anxiety-reducing functions. Typically, a securely attached infant will actively seek reunion with the caregiver following separation, but will explore his or her environment confidently. For a secure infant, this quality of caregiving permits the formation of an internal working model of self as both worthy and competent and a model of others as responsible and dependable. According to Bowlby and Ainsworth, these representations are ideal for the development of a healthy personality.

Early experiences where the primary caregiver consistently rejects the infant's effort to solicit protection, support, and caring, form the avoidant pattern of attachment. These infants experience stress upon separation from the caregiver, but reject or ignore the caregiver upon their return. This relationship forms an internal working model of self as alone and unwanted and a model of others as rejecting and untrustworthy. Additionally, these conditions cause the likely result of undesirable outcomes in personality and behavior (Lopez, 1995).

The third pattern of attachment, anxious-ambivalent, views the infant experiencing the caregiver as inconsistently responsive. The relationship fosters the formulation of an uncertain and fearful sense of self and a model of others as potentially affirming yet unreliable. These infant's are ambivalent about the caregiver's return, but they stay in close proximity to the caregiver. The child's sense of confidence and motivation for exploring his or her environment is dependent upon the presence, approval, and support of the attachment figure and this dependency on others leaves the individual vulnerable to stress and emotional instability as it fails to promote affective self-regulation (Ainsworth et al., 1978).

Although working models of self and others are shaped by additional outside experiences, parent-child attachment relationships have been shown to be relatively stable throughout childhood and adolescence (Crandall, 1972; Hunt & Eischorn, 1972). Working models of these attachment relationships appear to be relatively stable across time (Bowlby, 1988; Rothbard & Shaver, 1994), and the models that are formed during early childhood experiences are likely to influence later interactions (Collins & Read, 1994). As children grow older, the attachment behaviors directed toward the caregiver become less intense and less frequent. However, continued parental attachment has been shown to remain significant throughout adolescence and the college experience (Armsden & Greenberg, 1987; Berman & Sperling, 1991). At the same time, though, Weiss (1982) suggests that attachment behaviors are often being directed toward non-caretaking individuals. Although the adolescent may view the parent as wiser, stronger, and more stable, peer relationships may be considered a type of attachment relationship on a situational or temporary basis. Adding support to this hypothesis, Burke and Weir (1978) found that adolescents who reported satisfaction with the support they received from both their peers and their parents also reported greater levels of psychological well-being. In another study, Greenberg, Siegal, and Leitch (1984) reported that both peer and parental attachments were related to self-esteem and life satisfaction in adolescents, suggesting that both of these attachment relationships may be important in determining an adolescent's well-being. Similar results, adding empirical support to this hypothesis, were obtained by Armsden and Greenberg (1987) who found that the perceived quality of both peer and parental attachment relationships was significantly and positively associated with well-being, particularly with measures of self-esteem and life satisfaction. In addition, Armsden and Greenberg reported

that the quality of both peer and parental relationships in late adolescence meaningfully predicted scores on measures of depression, anxiety, resentment, and alienation.

In addition, these mental representations are believed to influence personality development (Erikson, 1950; Blatt & Shichman, 1982; Bowlby, 1969, 1973, 1980). Research shows that insecure attachment relationships leave an individual vulnerable to the development of a personality structure marked by an inability regulate affect, maintain self-esteem, and engage in mutually satisfying interpersonal relationships (Blatt & Hoffman, 1992; Fishler, Sperling, & Carr, 1990; Salzman, 1996). Eventually these personality disturbances may lead to psychological disorders such as depression, anxiety, personality disorders, or eating disorders (Alden, Bieling, & Wallace, 1994; Blatt & Hoffman, 1992; Livesley, Jackson, & Schroeder, 1992; O’Kearney, 1996; Parker, Tupling, & Brown, 1979; West & Keller, 1994; West, Sheldon, & Reiffer, 1987).

Several studies have demonstrated an association between attachment difficulties and eating disorders (See Ward, Ramsey, & Treasure, 2000 for a review). Using a non-clinical sample, Becker, Bell, and Billington (1987) reported that women displaying bulimic symptomatology scored significantly higher than asymptomatic women on a measure of insecure attachment. Similar results were found by Evans and Wertheim (1988) and Sharpe et al., (1998), who discovered that insecurely attached women in a non-clinical sample reported greater weight concerns, body dissatisfaction, and eating disordered attitudes. The results of Armstrong and Roth’s (1989) study indicated significant impairments in the attachment relationships of hospitalized eating disorder patients. Specifically, 96% of the sample displayed characteristics associated with an anxious attachment style, and 85% of the women displayed symptoms of depression upon separation from their attachment figure. Additionally, Kenny and Hart (1992)

found that secure parental attachments characterized by emotional support and parental fostering of autonomy were inversely associated with dieting preoccupation, feelings of ineffectiveness, and bulimic behavior. Studies have also found similarities between eating disordered women and insecurely attached individuals, reporting lower levels of self-esteem and higher levels of social incompetence and personal ineffectiveness (Armstrong & Roth, 1989; Heesacker & Niemeier, 1990; Kenny & Hart, 1992; Sharpe et al., 1998).

A majority of the early work examining the relationship between attachment and eating disorders used the Parental Bonding Instrument (PBI; Parker et al., 1979). Calam, Waller, Slade, and Newton (1990) investigated perceptions of parental care and control in women diagnosed with anorexia and bulimia. Compared to a control group, anorexics and bulimics rated both parents lower on parental care. These findings confirm early research done by Humphrey (1986), who reported that eating disordered women perceived their parents to be rejecting and neglecting. Calam et al. also confirmed earlier work using the PBI in which women seeking treatment for eating disorders recalled less maternal empathy and perceived their fathers to be significantly less caring (Palmer, Oppenheimer, & Marshall, 1988; Pole, Waller, Stewart, & Parkin-Feigenbaum; 1988; Steiger, Van der Feen, Goldstein, & Leichner, 1989). In addition, Calam et al. reported that women diagnosed with an eating disorder perceived their fathers (but not their mothers) to be more overprotective. Later research (Lavik, Calusen, & Pedersen, 1991; Rhodes & Kroger, 1992) also found significantly higher perceptions of maternal overprotection, separation anxiety, and lower healthy separation scores among women displaying eating disordered symptoms.

Consistent with an insecure style of attachment, it appears that eating disordered women view their parents as emotionally unavailable and less supportive of their autonomous

functioning. Thus, they are likely to experience anxiety and believe their parents will not provide comfort and support when needed. It has been suggested that the symptoms of anorexia can be utilized to generate concerns and manipulate support from sources that might otherwise be unavailable, whereas the binge-eating and purging of bulimia provide a internal sense of comfort (Strober & Humphrey, 1987).

More recent studies have attempted to determine the style of the attachment relationships associated with eating disorders, using structured interview formats. In a sample of 14 psychiatric inpatients diagnosed with an eating disorder, Fonagy et al. (1996) classified one patient as securely attached. Nine patients displayed characteristics associated with a preoccupied (anxious) pattern of attachment, and four were characterized by a dismissive (avoidant) pattern of attachment. Similar results were found by Candelori and Ciocca (1998), who also reported that adolescent women diagnosed with restricting anorexia nervosa were more likely to display dismissive attachment patterns, and women diagnosed with bulimia and binge-purging anorexia were more likely to be classified by the preoccupied attachment pattern. Although the Adult Attachment Interview (AAI; George, Kaplan, & Main, 1985) is regarded as the “gold standard” among researchers interested in examining adult and adolescent attachment, the format is cumbersome and impractical for use on large samples. Thus, little empirical evidence using this format exists beyond a few studies that investigate the relationships among eating disorders and attachment.

Taken together, the above findings suggest that attachment relationships with both parents serve as influential factors that may provoke or prevent the development of eating disturbances (O’Kearney, 1996). Although only a few empirical studies demonstrate a connection between specific patterns of attachment that may contribute to eating disturbances, a

general conclusion can be drawn that insecure patterns of attachment lead to a vulnerability for development of later eating pathology. Additionally, although early bonds formed with caregivers are important in the developmental process, much of the literature linking attachment and eating disorders has failed to consider later environmental experiences, cultural factors, and interpersonal relationships that may exacerbate or mitigate the vulnerabilities created by insecure attachments.

Integration of Theories and Existing Models

Stress Processes and Sociocultural Standards of Attractiveness

The onset of an eating disorder for most women begins during the period of adolescence (APA, 2000), suggesting that this developmental period may be a risk factor for the onset of eating pathology (Halmi, Casper, Eckert, Goldberd, & Davis, 1979). More specifically, Smolak and Levine (1996) suggested that the developmental transitions, periods between the end of one stage and the beginning of another, may be times of special risk because changes in social roles and cultural expectations as well as significant reorganization of an individual's personality and cognitive and relationship structures occurs during this time. Extending the stress-appraisal-coping model, Levine and Smolak (1992; Smolak & Levine, 1996) focused on the developmental transitions of early adolescence, proposing several hypotheses that might explain the individual differences among adolescent girls regarding body dissatisfaction, dieting, and eating disordered symptomatology.

One hypothesis examines the relative timing of puberty. Research documents an association between the onset of puberty and increased body dissatisfaction and dieting behaviors. In addition, Brooks-Gunn (1987) and Crisp et al. (1980) suggested that later maturing

girls, compared to earlier maturing girls, feel more positively about their bodies following the onset of puberty. Thus, early maturing girls may display negative attitudes regarding body image and eating earlier than their peers, allowing those attitudes and behaviors to become more salient (Attie & Brooks-Gunn, 1989; Polivy & Herman, 1987). Smolak, Levine, and Gralen (1993) also suggested that stress associated with physically developing earlier than their peers may leave early maturing girls more vulnerable to psychological distress.

In an additional model, Smolak, Levine, and Gralen (1993) suggested that early dating may be connected to body dissatisfaction and dieting behaviors. Previous research suggests that early dating may result in early exposure to and adoption of adult-like behaviors such as dieting (Magnusson, Stattin, & Allen, 1986) and in believing that attractiveness that is based on thinness will make them more successful in the dating process (Paxton et al., 1991). Unfortunately, early research did not support the hypothesis that either dating or onset of puberty was related to an increase in eating disturbance in adolescent girls; however, research did show that a synchronous onset of menstruation and dating resulted in higher scores on an adolescent measure of disturbed eating attitudes and behaviors (Smolak, Levine et al., 1993).

Integrating these two hypotheses with the developmental transition of adolescence, Levine and Smolak (1992) proposed a cumulative stress model of eating disturbance. Specifically, the model suggests that developmental changes associated with adolescence, namely onset of dating, weight/fat gain resulting from puberty, and increased academic demands, interact with the thin-ideal body image to produce eating disturbances.

In a test of the model, Levine, Smolak, Moodey, et al. (1994) found an association between recent menstruation, recent dating, and engagement in extensive weight management behaviors. In addition, middle school girls who began menstruation and dating at the same time

reported significantly more weight management behaviors than girls who had experienced only one of these changes. Finally, adherence to the slender body-ideal in addition to these developmental changes may distinguish non-pathological dieting from disturbed eating attitudes and behaviors.

In their examination of late adolescent transition, Smolak and Levine (1996) proposed a similar developmental model, including predisposing factors, stress triggers, and developmental tasks. Specifically, the late adolescent period marks the end of high school and the transition to new environments such as going to college. Stress triggers during this period may include different standards of attractiveness, temporary reductions in social support, new dating and sexual expectations, increased academic demands, and financial concerns. Typically, these triggers may result in modifications to the self, cognitive and social structures as the adolescent examines the adequacy of her personal and social environment and her ability to cope with the demands of the transition (Smolak & Levine, 1996).

Although the developmental challenge of attending college is generally viewed as a positive stressor, Striegel-Moore, Silberstein, and Rodin (1993) suggested that certain predisposing factors may make this developmental adjustment period more difficult. Specifically, socially anxious women or those who require social approval may be more vulnerable to developing psychological disturbances, including eating disorders. Lowe (1993) added that socially anxious and/or socially needy women may also be hypersensitive to the sociocultural pressures emphasizing thinness during this period due to a need to make a good impression on others. Thus, the development of an eating disorder is a specific concern. In addition, an early history of dieting and internalization of societal standards of attractiveness has been shown to place women at greater risk for the development of eating disturbances (Lowe,

1993; Striegel-Moore & Marcus, 1995; Timko, Striegel-Moore, Silberstein, & Rodin, 1987; Tobin, Johnson, Steinberg, Staats, & Dennis, 1991). Smolak and Levine (1996) suggested that the risk is even greater during the late adolescent transition period because attitudes and behaviors become more consolidated.

Finally, research has demonstrated that college, rather than high school, is the time during which establishment of identity is completed, and the separation-individuation process that occurs during this developmental period plays a key role in that identity development. Separation involves a decreased dependency on caregivers, whereas individuation refers to the increase in self-responsibility. For some women, negotiating this task may be more difficult.

According to Gilligan (1982) women are socialized to value connection over independence, and many young women maintain strong connections to their caregivers as they leave home to attend college (Smolak, 1996). At the same time, society communicates a “superwoman model,” suggesting that women must maintain interpersonal connections by placing family first. However, society also dictates that women should possess careers that create a financial independence, emphasizing individuation (Smolak & Levine, 1996). Thus, women are often confused and frustrated by the separation-individuation process. In addition, a young woman’s family may have difficulties with the separation-individuation of their daughter. In particular, enmeshed, overprotective, and emotionally distant families may make it difficult for a young woman to establish autonomy, creating additional stress for the individual (Strober & Humphrey, 1987).

In summary, Smolak and Levine (1996) outlined a developmental transition model, suggesting that the developmental task of separation-individuation combined with the stress triggers of leaving home and attending college may interact with predisposing factors such as

social anxiety, neediness, susceptibility to sociocultural standards of attractiveness, and early history of dieting, leaving adolescent women who are navigating the late adolescent transition vulnerable to the development of eating disturbances.

Using structural equation modeling, Tripp (2002) conducted an examination of the direct and indirect links connecting the stress process and disordered eating. Providing support for Smolak and Levine's (1996) developmental transition model, Tripp's statistical findings suggested that women who endorsed previous occurrences of stressful events were likely to perceive difficulty coping with the demands associated with the developmental transition of attending college and experience increased levels of psychological distress. In addition, women who reported increased levels of psychological distress and greater internalization of the thin-ideal also displayed increased body dissatisfaction, a risk factor related to higher levels of eating disordered symptomatology.

Attachment and Stress Processes

Bowlby (1969; 1973; 1980) and Ainsworth (1973) have proposed that periods of stress often activate attachment behaviors, allowing individuals to react to their environment in an adaptive fashion. Research examining this hypothesis has demonstrated that a secure relationship between parent and child can serve as a buffer during stressful events because these relationships provide the individual with a sense of security (Peterson, Sarigian, & Kennedy, 1991). In addition, interpersonal theory suggests that individuals who are insecurely attached may lack the ability to regulate affective experiences, particularly when they are exposed to stress, leaving these individuals vulnerable to the development of psychological disturbances (Striegel-Moore & Smolak, 1996).

Examining attachment patterns and eating disordered behavior in college women may be particularly relevant because of the developmental and environmental stressors associated with this period. Specifically, leaving home and starting college has been regarded as a naturally occurring “strange situation” (Ainsworth et al., 1978; Bloom, 1980; Kaplan, 1991; Kenny, 1987) because adolescents separate from their familiar parental and family attachments and enter a new environment. This separation activates the attachment behavioral system, producing behaviors designed to regain and maintain optimal proximity to the caregiver (Bowlby, 1969, 1980). Because late adolescence and early adulthood is also a key period during which many individuals develop symptoms of psychopathology, blame for many of the behavioral and emotional difficulties of late adolescence and early adulthood is often placed on an individual’s negotiation of this transitional period (Bloom, 1987).

Since internal working models serve as patterns for subsequent attachment behaviors, the college student with a secure working model of both self and others should be able to anticipate that others will be trustworthy and responsive. Consequently, this individual should be better able to form supportive relationships with others and rely on their own abilities to moderate the stress associated with the separation from home and beginning of college. Similarly, negative or insecure working models of self and/or others may make the individual vulnerable to the effects of stress (Kenny & Rice, 1995). Thus, in keeping with attachment theory, it should be expected that individuals who experience secure attachment during their childhood would be better able to handle stressful situations by believing they are capable of managing their environment and believing that support is available if needed. On the other hand, individuals whose attachment figures are rejecting or inconsistent in their responses are likely to appraise a stressful situation

as unmanageable due to their negative beliefs about their own abilities to cope with the situation and their belief that others cannot be trusted or depended upon to provide support.

Kenny (2000) proposed that investigations into the way people experience and cope with stressful events begin with an examination into the earliest social and relational interactions the individual has with the world. With this idea, she suggested a model for understanding the development of coping and the management of stressful life experiences arguing that parental attachment is a key factor necessary for the successful management of stressful life experiences because early parent-child interactions permit the development of internal working models of relationships. When presented with life experiences, the internal working models formed in the parent-child interaction influence the individual's appraisal of the experiences and their coping repertoire. As individuals develop healthy working models of themselves and others, they learn to utilize other resources in their environment and develop less of a dependence on the attachment figure for support. Thus, if initial attachment relationships are faulty, the individuals will develop unhealthy working models, leading to poor social competence and vulnerability to stressful occurrences.

Borrowing Baltes (1987) conceptualization, Kenny (2000) proposed that development is an interaction of gains and losses across the life span, suggesting that developmental processes are characterized by the addition or losses of challenges to an individual's functioning as well as increasing development of resources and skills to meet those challenges. Specifically looking at the cognitive development that occurs during adolescence as individuals shift from concrete-operational to formal operational thinking (Piaget, 1970), Kenny proposed that the interaction of gains and losses may lead to maladaptive coping processes. Combined with the gains of this developmental period, including academic skills, problem solving abilities, and the development

of functional coping strategies that may protect adolescents from stressful experiences (Werner, 1995), Elkind (1967) reported that the adolescent also develops a form of egocentrism that causes them to mistakenly believe that the outside world is as concerned with their thoughts and appearances as they are, leading to the development of anxiety and self-criticism that can create a faulty use of their social environment in times of need. Thus, the adolescent may experience the developmental period as a stressful event.

Building on Garnezy and Masten's (1986) argument that attachment influences an individual's resiliency to stress by encouraging the development of social competence, Kenny (2000) cited three possible links between parental attachment and the development of social competence. First, a secure attachment relationship allows the individual to use the attachment figure as a secure base from which to explore their environment (Bowlby, 1973). In addition, exploration, problem-solving, and the development of mastery over their environment, has been linked to the development of social competence (Field, 1996; Jacobson, Edelstein, & Hoffman, 1994; Lyons-Ruth, 1996; Maccoby, 1983). Thus, an individual with a secure attachment style also is likely to develop social competence, leaving them more resilient during times of stress.

Second, early experiences with stressful events may influence subsequent development of social competence. Very young children have a limited repertoire of coping behaviors, so their typical reaction to a stressful event is to return to the attachment figure. Compared to securely attached infants, Lyons-Ruth (1996) reported that infants with disorganized patterns of attachment displayed lower mental development scores. Additional research suggests that maternal depression may lead to insecure patterns of attachment and subsequent deficits in social competence (Cox, Puckering, Pound, & Mills, 1987; Mills, Puckering, Pound, & Cox, 1985).

Third, Kenny (2000) cited a direct association between the development of social competence and secure attachment relationships. Bretherton (1985) reported securely attached infants displayed more prosocial behavior than insecurely attached infants. Conversely, children with insecure patterns of attachment displayed aggression, passive withdrawal, hyperactivity, helplessness, and depression (Main & Solomon, 1990; Renken, Egeland, Marvinney, Mangelsdorf, & Sroufe, 1989). In addition, longitudinal studies indicate that securely attached children are more independent, more socially competent, show higher self-esteem, and appear more resilient to stress than insecurely attached children (Goodyear, 1990).

In summary, Kenny viewed the adolescent developmental period as an interaction of gains and losses that may be experienced as stressful. However, she also suggested that an individual's early attachment relationships provide working models that influence the development of appraisal and coping processes. Specifically, secure patterns of attachment allow for the individual to form an internal working model of self as worthy and competent and a model of others as responsible and dependable. Therefore, the individual will be more likely to appraise stressful situations as manageable, feel competent in coping with the situation, and feel comfortable seeking help from others if needed. Second, Kenny proposed three means by which early attachment relationships may be related to the development of appropriate stress appraisal and coping processes. She presented an argument that secure attachments provide an infant a sense of safety that encourages exploration and development of social competence through successful experiences. These early experiences of success appear to be related to continuing cognitive development. Finally, Kenny cited a direct link between secure attachment and social competence. Thus, it appears that early attachment relationships might play an important role in

the development of eating pathology by impairing an individual's abilities to effectively appraise and cope with stressful situations.

Summary and Conclusions

Multiple etiological perspectives in the development of eating disturbances have been explored, and it is clear from the literature that the relationship among attachment, stress processes, sociocultural pressures, and eating disturbance is complex. Empirical findings support the role attachment relationships, history of experienced stress, appraisal/coping processes, psychological distress, internalization of sociocultural beliefs regarding appearance, and body image play in the development of eating pathology. However, to further understand the development of eating disturbances, conceptualizing the relationships among these variables is needed.

The first important connection to evaluate is the relationship between attachment and the stress process. As mentioned previously, research supports the importance of attachment in the development of eating pathology (See Ward et al., 2000 for a review). Attachment style influences an individual's working models of self and others, and affects the development of personality and self-experiences that culminate in a vulnerability to developing psychopathology, including eating disorders (Kenny, 2000; Kenny & Rice, 1995). Further, Kenny suggested that unhealthy internal working models resulting from insecure parental attachments lead to social incompetence, poor peer attachments, and vulnerability to stressful events, which may result in the use of maladaptive appraisal/coping processes and emotional distress. Current research supports a connection between parental attachment and appraisal/coping processes and confirms Kenny's hypothesis that secure attachments provide an infant a sense of safety that encourages

exploration and the development of social competence through successful experiences (Field, 1996; Jacobson, V, 1994; Lyons-Ruth, 1996; Maccoby, 1983). Secure parental attachment has also been shown to act as a buffer during the experience of stressful events (Petersen et al., 1991), suggesting that insecurely attached individuals may experience stressful events more negatively than securely attached individuals because they lack the competence to successfully manage stressful events.

In addition, the appraisal/coping process appears to be influenced by the history of experienced stressful events. Research findings suggest that increased incidence of prior stressful occurrences is directly and positively associated with stressful appraisal of current life events and less ability to cope with the event (Shatford & Evans, 1996; Tripp, 2002).

Empirical findings also support the importance of the history of experienced stress and the appraisal coping process in the development psychological disturbances, including eating disorders. Although somewhat mixed, findings generally support the hypothesis that eating disordered women have been exposed to more potentially stressful events than non-eating disordered women. In addition, research supports the idea that a woman's perception of the potentially stressful event and her appraisal/coping abilities may mediate the relationship between stress occurrence and the development of eating disorders. Additionally, empirical evidence also suggests a direct relationship between appraisal/coping processes and psychological distress, which may then lead to the development of eating disturbances (Shatford & Evans, 1986; Tripp, 2002). Thus, it appears that the experience of stressful events and/or deficits in the appraisal/coping process may leave an individual vulnerable to the development of psychological disturbances, and additional factors may be responsible for the development of specific disorders, including eating disorders.

Next, it is important to evaluate the link between negative affect and body dissatisfaction. Models tested by Stice (1994) and Striegel-Moore et al. (1986) propose that body dissatisfaction leads to psychological distress, such as negative affect, and other empirical evidence supports the placement of negative affect before body dissatisfaction (Kearney-Cooke & Striegel-Moore, 1994; Tripp, 2002; Tripp & Petrie, 2001).

Internalization of sociocultural beliefs also plays an important role in the development of eating disturbances. Stice's dual pathway model (Stice, 2001a; Stice, Ziemba, et al., 1996) suggests that internalization of the thin ideal fosters dissatisfaction with one's body and subsequent eating pathology. Further, Stice and colleagues (Stice, Mazotti, et al., 2000; Stice, Chase et al., 2001) demonstrated that experimental reduction of the thin-ideal internalization resulted in improved body satisfaction. However, research also has suggested that internalization of the thin-ideal promotes negative affect (Stice & Argas, 1998; Thompson et al., 1999). Thus, it appears that the empirical findings support a relationship between internalization of sociocultural beliefs regarding appearance and negative affect as well as between internalization of sociocultural beliefs regarding appearance and body dissatisfaction.

Finally, the relationship between body dissatisfaction and disordered eating must be evaluated. Stice's dual pathway model (Stice, 2001a; Stice, Ziemba et al., 1996) proposed that the relationship between body dissatisfaction and the development of eating disordered symptomatology is mediated by negative affect, but research also suggests a direct connection between dissatisfaction with one's body and eating pathology (Attie & Brooks-Gunn, 1989; Leon et al., 1993; Mintz & Betz, 1988, Stice, 1994; Striegel-Moore et al., 1988). In addition, the diagnostic criteria (APA, 2000) lists disturbed perception of body weight and shape as primary symptom of both anorexia and bulimia.

Investigating a multidimensional model of eating disorder development, Bradford (2004) demonstrated support for 2 models that explain the relationship among parental attachment, history of stress, appraisal/coping process, internalization of the thin-ideal, psychological distress, body image, and eating pathology in undergraduate women in their first semester in college. Using structural equation modeling, Bradford's study revealed that women who reported insecure attachments to their parents also reported insecure attachments to their peers. Further, women who reported insecure attachments to their parents and women who endorsed a higher incidence of stressful life experiences in the past six months were more likely to perceive the transition to college and the increased demands as being more stressful. In addition, these women felt less capable of their ability to cope with the transition and the new demands of college. Participants who perceived greater stress and less ability to cope with the transition and demands of being in college experienced greater levels of psychological distress (e.g., feeling stressed and anxious). Increased feelings of psychological distress and greater internalization of societal pressures to achieve the thin-ideal body type were related to increased body dissatisfaction. Finally, women who reported decreased body satisfaction and greater internalization of sociocultural pressures to be thin also reported more disturbed eating attitude and behaviors. Thus, this model supports linear pathways of eating disorder development in which direct paths exist from parental attachment and history of stress to appraisal/coping, from appraisal/coping to psychological distress, from psychological distress to body dissatisfaction, and from body dissatisfaction to eating pathology. Bradford's Model Also provided support for Tripp's (2002) evaluation of a model testing the relationships among the stress-coping process, negative affect, sociocultural influences, body disparagement, and eating disordered behaviors and Smolak and Levine's (1996) developmental transition model.

Results of Bradford's (2004) study also supported an equivalent model which fits the same data equally as well as the model discussed above. Similar to the above model, women who reported insecure attachments to their parents and women who endorsed a higher incidence of stressful life experiences in the past six months were more likely to perceive the transition to college and the increased demands as being more stressful. Like the previous model, these women felt less capable of their ability to cope with the transition and the new demands of college, and those who perceived greater stress and less ability to cope with the transition and demands of being in college experienced greater levels of psychological distress (e.g., feeling stressed and anxious). Greater internalization of the thin-ideal was also directly related to increased body dissatisfaction. In this model, though, increased levels of psychological distress, body image dissatisfaction, and internalization of the thin-ideal were all significantly related to the development of eating disturbances. Thus, this Model Advocates the influence of two separate paths in the development of eating disorders. In the first path, attachment processes and history of stress directly influence the appraisal coping process which directly influence psychological distress to lead to the development of eating disturbances. In the second path, sociocultural influences and body image also influence the development of eating disorders. This Model Also lends additional support to previous research in which both negative affect and body dissatisfaction were found to predict eating pathology (Killen et al., 1996; Stice & Argas, 1998; Stice, Mazotti, et al., 1998).

Bradford's (2004) work indicated that the explanation by which attachment processes, stress-coping processes, sociocultural pressures, and body image influence the development of eating disorders is complex and may occur along more than one pathway. Unfortunately, all data

in Bradford's study was collected at a single point in time. Thus, longitudinal research investigating these models is needed to provide further clarification.

Hypothesis

The purpose of this study is to expand on Bradford's (2004) study and examine an integration of multiple risk factors that contribute to the development of eating disorders using prospective, longitudinal data. The proposed Model Advocates a multidimensional view of the etiology of eating disorders, accounting for a hierarchical integration of the factors. The following correlates of disordered eating are incorporated into the proposed model: parental attachment, history of experienced stress, appraisal/coping processes, internalization of the thin-ideal, negative affect, body image, and eating disordered behavior. The hypothesized relationships are described below.

Model A

1. Parental attachment is hypothesized to have a direct positive effect on the appraisal/coping process and an indirect negative (inverse) effect on psychological distress, an indirect positive effect on body image, and an indirect negative (inverse) effect on eating disturbances. For example, positive parental attachment relationships will be related to positive appraisal/coping, decreased negative affect, positive body image, and less eating pathology.
2. History of stress is hypothesized to have a direct negative (inverse) effect on the appraisal/coping process and an indirect positive effect on negative affect, an indirect negative effect on body image, and an indirect positive effect on eating disturbances.

- For example, greater occurrences of past stress will be related to negative appraisal/coping, more negative affect, negative body image, and more eating pathology.
3. The appraisal/coping process is hypothesized to predict negative affect, having a direct negative (inverse) effect. For example, positive appraisal/coping will be related to less negative affect. In addition, it is hypothesized that the appraisal coping process will mediate the relationship between parental attachment and negative affect and between history of stress and negative affect.
 4. Negative affect is hypothesized to predict body image, having a direct negative (inverse) effect. For example, higher levels of negative affect will be related to negative body image. It is also hypothesized that negative affect will mediate the relationship between the appraisal/coping process and body image.
 5. Internalization of sociocultural beliefs regarding appearance is hypothesized to predict both body image and eating disturbance, having a direct negative (inverse) effects on body image and direct positive effects on eating disturbance. For example, greater internalization of the thin ideal body image will be related to negative body image and more eating pathology.
 6. Body image is hypothesized to predict eating disturbance, having a direct negative (inverse) effect. For example, positive body image will be related to less eating pathology. It is also hypothesized that body image will mediate the relationship between negative affect and eating disturbance and between internalization of the thin-ideal and eating disturbance.

Model B

1. Parental attachment is hypothesized to have a direct positive effect on the appraisal/coping process and an indirect negative (inverse) effect on negative affect and an indirect negative (inverse) effect on eating disturbances
2. History of stress is hypothesized to have a direct negative (inverse) effect on the appraisal/coping process and an indirect positive effect on negative affect and an indirect positive effect on eating disturbances.
3. The appraisal/coping process is hypothesized to predict negative affect, having a direct negative (inverse) effect. In addition, it is hypothesized that the appraisal coping process will mediate the relationship between parental attachment and negative affect and between history of stress and negative affect.
4. Negative affect is hypothesized to predict eating disturbance image, having a direct positive effect. For example, higher levels of negative affect will be related to more eating pathology. It is also hypothesized that negative affect will mediate the relationship between the appraisal/coping process and eating disturbance.
5. Internalization of sociocultural beliefs regarding appearance is hypothesized to predict both body image and eating disturbances, have direct negative (inverse) effects on body image and direct positive effects on eating disturbance. For example, greater internalization of the thin ideal body image will be related to negative body image and more eating pathology.

6. Body image is hypothesized to predict eating disturbance, having a direct negative (inverse) effect. For example, positive body image will be related to less eating pathology. It is also hypothesized that body image will mediate the relationship between internalization of the thin-ideal and eating disturbance.

Diagrams of the hypothesized models are presented in Figure 2 and Figure 3. Both figures present models using a fully cross-lagged design in which the variables are assessed at each time period and both the hypothesized relationships as well as alternative hypotheses are assessed. Dashed, single-headed arrows connecting like constructs (e.g., from Body Image Time 1 to Body Image Time 2) represent within construct paths, allowing for the examination of the cross-time stability of the construct. Solid, single-headed arrows represent all logical paths, including the hypothesized paths as well as the alternative paths (e.g., the hypothesized relationships that body image predicts eating disturbance as well as the contradictory hypothesis that eating disturbance predicts body image.) The double-headed arrows connecting the Time 1 constructs represent covariance among the variables. Disturbance errors for the indicated latent variables are indicated by the small error leading from the latent variables to the “D.” Disturbances within time are correlated; however, for ease of presentation these correlations are only represented between the Time 4 factors. Measurement errors for the measured variable are also correlated across time, but are not included in these figures.

CHAPTER 2

METHOD

Participants

Participants consisted of 480 females who volunteered to participate in an initial study during the fall 2003 semester and provided consent to be contacted for future data collections. The initial data collection consisted of 429 female freshmen participants in their first semester of college, who were enrolled in psychology courses, and 51 first semester freshman members of a women's social sorority from two large public universities located in the southwestern United States. Five of these participants did not sufficiently complete the data, and they were dropped from the initial study and any future participation in the study. All of the 475 participants who sufficiently completed the initial study were contacted and asked to participate in the second data collection. Out of the 475 initial participants, 351 completed the second data collection (26.89% total dropout), 173 completed the third data collection (63.96% total dropout), and 238 completed the fourth data collection (50.42% total dropout). Sixty-five participants completed the second data collection and the fourth data collection but did not complete the third data collection, so the data from the third collection was removed from the study. Thus, only the 238 participants who completed the first, second, and fourth data collections were included in this study. The study will refer to the first data collection as Time 1 (T1), the second data collection as Time 2 (T2), and the fourth data collection as Time 4 (T4).

Initial participation was limited to women at least 18 years of age at the time of the data collection and who were in their first semester of college. Participants ranged in age from 18 to 21 years at Time 1 ($M = 18.20$, $SD = .47$), from 18 to 21 years at Time 2 ($M = 18.69$, $SD = .58$), from 19 to 22 years at Time 4 ($M = 19.50$, $SD = .57$). Participants were predominantly

Caucasian (69.7%), but included African American (13.9%), Hispanic American (8.8%), American Indian (4.6%), Asian American/Pacific Islander (1.7%), and undisclosed origin (1.3%).

At Time 1, 237 (99.6%) of the participants indicated that they were single, while 1 participant (0.4%) reported being married. At Time 2, 232 (97.5%) of the participants indicated that they were single, while 4 participants (1.7%) reported being married and 2 participants (0.8%) declined to answer the question regarding marital status. At Time 4, 235 (98.7%) of the participants indicated that they were single, while 2 participants (0.8%) reported being married and 1 participant (0.4%) declined to answer the question regarding marital status. Body Mass Indices (BMI; kg/m^2) ranged from 16.64 to 42.97 at Time 1 ($M = 22.49$, $SD = 4.18$), from 16.14 to 42.97 at Time 2 ($M = 22.80$, $SD = 4.28$), and from 16.64 to 42.97 at Time 4 ($M = 23.06$, $SD = 4.33$). Ideal Body Mass Indices ranged from 16.74 to 31.96 at Time 1 ($M = 20.61$, $SD = 2.45$), from 16.64 to 34.97 at Time 2 ($M = 20.79$, $SD = 2.64$), and from 16.73 to 30.04 at Time 3 ($M = 20.88$, $SD = 2.55$).

All participants were enrolled in 9 to 18 hours of college coursework, with 97.9% of the women being enrolled in 9-16 hours of college course work at Time 1 ($M = 13.75$, $SD = 1.64$). Using a 100-point grading scale, high school grade point averages (GPA) for the participants ranged from 50 to 100 ($M = 85.0106$, $SD = 10.61$). Although initial participation was limited to women who were enrolled in their first semester of college, it was not a requirement that the participants maintain continued enrollment over the course of the study to continue to participate in the study. At Time 2, participants were enrolled in 0 to 18 hours of college coursework, with 89% of the women being enrolled in 9-16 hours of college course work ($M = 13.45$, $SD = 3.11$). College grade point averages for the participants during their first semester in college ranged

from 0.00 to 4.00 ($M = 3.02$, $SD = 0.67$). At Time 4, participants were enrolled in 0 to 19 hours of college coursework, with 88.2% of the women being enrolled in 9-16 hours of college course work ($M = 13.51$, $SD = 2.91$). College grade point averages for the participants ranged from 1.00 to 4.00 ($M = 3.01$, $SD = 0.57$).

The majority of the participants reported residing in a dormitory during their first semester in college (74.8%), but others reported living in an apartment/house with at least one roommate (8.84%), living alone in an apartment/house (1.3%), living with their parents (11.3%), and an undisclosed living situation (3.8%). In addition, 19.7% ($n = 47$) of the participants reported involvement in a sorority. At time 2, the majority of the participants again reported residing in a dormitory (60.1%), but others reported living in an apartment/house with at least one roommate (17.2%), living alone in an apartment/house (2.1%), living with their parents (18.1%), and an undisclosed living situation (2.5%). Forty-seven of the participants (18.9%) reported involvement in a sorority. At time 4, the participants reported residing in an apartment/house with at least one roommate (41.2%), and others reported living in a dormitory (18.9%), living alone in an apartment/house (7.6%), living with their parents (19.3%), and an undisclosed living situation (13.0%). Forty-eight of the participants (20.2%) reported involvement in a sorority.

Regarding annual income for their family of origin, 5.5% of the participants indicated that the annual income for their family was less than \$20,000, 17.2% reported a range from \$20,001 to 40,000, 15.1% reported a range from \$40,001 to 60,000, 14.7% reported a range from \$60,001 to 80,000, 22.3% reported a range from \$80,001 to 100,000, and 20.6% reported an annual income of greater than \$100,000; 4.6% ($n = 11$) of participants declined to answer the question regarding annual income in their family of origin.

Instruments

Demographics

A brief demographic survey designed by the researcher will be used to gather the following information: age, gender, marital status, race/ethnicity, academic rank, number of semesters attending an institute of higher education, high school GPA, socioeconomic status, current course load, height, present weight, ideal weight, sorority status, and current living arrangement.

Parental Attachment

The 25-item Parental Bonding Instrument (PBI; Parker et al., 1979) measures perceived parental bonding. Participants rated each biological parent or parent-figure separately, responding to questions based on recollections of their parents from the participant's first 16 years of life. The PBI is composed of two scales for each parent. The Care scale (12 items) measures the affective element of the parent-child relationship, with the positive end of the scale reflecting warmth, affection, empathy, and closeness and the negative end emotional coldness, indifference, and neglect. The Overprotection scale (13 items), measures the degree of control manifested by the parent, with encouragement of autonomy defining one end of the scale, and control, intrusion, excessive contact, and prevention of independent behavior reflecting the opposite end. Participants answered each question using a 4-point Likert scale, ranging from 1, *very like* to 4, *very unlike*. Scale scores were obtained by reverse scoring the appropriate items and summing across the scale. Total scale scores on the Care scale range from 12 to 48, with higher scores indicating affection and closeness. Total scores on the Overprotection scale range

from 13 to 52, with higher scores suggesting intrusiveness. A combination of high Care and low Overprotection defines optimal bonding.

Todd, Boyce, Heath, and Martin (1994) reported adequate internal consistency reliabilities for the Care (.94 to .95) and the Overprotection (.91 to .92) scales. Cronbach's alphas for the study were .90 for the Maternal Care and .93 for the Paternal Care scales and .82 for the Maternal Overprotection and .84 for the Paternal Overprotection scales. Parker and colleagues (1983; Parker et al., 1979) reported three-week test-retest reliabilities of .76 (Care) and .63 (Overprotection). Additionally, several studies have shown that scores on the PBI are independent of current mood states (Duggan, Sham, Minne, Lee, & Murray, 1998; Plantés, Prusoff, Brennan, & Parker, 1988).

History of Stress

The 49-item Inventory of College Students' Recent Life Experiences (ICSRLE; Kohn, Lafreniere, & Gurevich, 1990) measures the influence of everyday stressors across 7 subscales, including: (1) Developmental Challenge (influence and experience of stressors related to developmental and academic challenges); (2) Time Pressure (experience and influence of stressors related to demands on the individual's time); (3) Academic Alienation (experience and influence of stressors related to satisfaction in academic classes); (4) Romantic Problems (experience and influence of stressors related romantic relationships); (5) Assorted Annoyances (experience and influence of stressors that evoke anger rather than anxiety); (6) General Social Mistreatment (experience and influence of stressors related to social mistreatment by an unspecified source); (7) Friendship Problems (experience and influence of stressors related to peer relationships). For the purposes of this study, participants rated their experiences during the

previous 6 months. Participants answered each item using a 4-point Likert scale, ranging from 1, *not at all a part of my life* to 4, *very much a part of my life*. Subscale scores were obtained by summing across the items for each subscale and dividing by the number of items. Higher scores indicated greater experience of stress events.

Kohn et al. (1990) reported an internal consistency reliability of .89 for females for the entire scale. Cronbach's alpha for the current study was .92. Internal consistency reliabilities for the seven subscales ranged from .47 (Assorted Annoyances) to .80 (Time Pressure). For the current study, Cronbach's alphas were .76 (Academic Alienation), .57 (Assorted Annoyances), .81 (Developmental Challenge), .80 (Friendship Problems), .70 (General Social Mistreatment), .76 (Romantic Problems), and .82 (Time Pressure). In addition, items were selected for inclusion in the ICSRLE based on significant and positive correlations with the Perceived Stress Scale (PSS). Individual item correlations with the PSS ranged from .17 ($p < .05$) to .48 ($p < .0005$).

Appraisal/Coping

The 14-item Perceived Stress Scale (PSS; Cohen, Kamarch, & Mermelstein, 1983) assesses the degree to which participants appraise situations or events as stressful and evaluates how unpredictable, uncontrollable, and overloaded participants find their lives. Participants answered each question regarding stressful events from the past month, indicating the occurrence of negative thoughts and feelings. For the purposes of this study, participants rated the occurrence of negative thoughts and feelings regarding stressful events that have occurred in the last 6 months. Each item was rated using a 5-point scale, ranging from 0, *never* to 4, *very often*. Scores on the PSS range from 0 to 56, with higher scores indicating higher levels of stress.

Using a college student sample, Cohen et al. (1983) reported an internal consistency reliability of .84. Cronbach's alpha for the current study was .86. In addition, Cohen et al. found a two-day test-retest reliability of .85. Concerning construct validity, Cohen et al. reported small to moderate correlations between scores on the PSS and the number of life events ($r = .20, p < .01$) and the impact of life events ($r = .35, p < .01$).

Based on Folkman's (1984) appraisal/coping model, a 10-item questionnaire (Tripp, 2002) was used to assess participants' appraisal and coping in five critical life areas in college: concern regarding weight gain, academic demands, new friendship demands, romantic relationships demands, and novel living arrangement demands. The questionnaire assesses each participant's evaluation of the stress being experienced in each life area (primary appraisal) as well as their evaluation of their ability to handle each experience (secondary appraisal). Examples of items include, "Since starting college, I have been experiencing stress due to the academic demands in my classes" and "Coping with the academic demands in college has been difficult for me." Participants rated their level of agreement with each item using a 5-point Likert scale ranging from 1, *strongly disagree* to 5, *strongly agree*. A total primary appraisal score and a total secondary appraisal score were obtained by summing the ratings of the stressors.

Cronbach's alphas for the current study for the primary appraisal score and the secondary appraisal score were .52 and .48, respectively. Due to the poor internal consistency, the current study combined the primary and secondary appraisal scores for each critical life area. Using this approach, Cronbach's alphas for the current study were .79 (academic demands), .91 (new friendship demands), .93 (novel living arrangement demands), .93 (romantic relationship demands), and .94 (weight gain concerns).

Negative Affect

The 7-item Visual-Analogue Mood Scale (VAMS; Stice & Shaw, 1994) assesses participants' current affective states. The scale consists of items assessing depression, happiness, shame, guilt, confidence, anxiety, and stress. For each item, participants rated their emotional state along a 5-point scale ranging from 1, *not at all* to 5, *extremely*. For the purposes of this study, participants rated their emotional state since the beginning of the semester. Scores from individual items were used to assess emotional well-being in each area.

Previous studies (Folstein & Luria, 1973; Robinson, McHugh, & Folstein, 1975) have established the validity of visual-analogue scales. In addition, Stice and Shaw (1994) reported significant correlations between scores on the Beck Depression Inventory and the Depression item ($r = .35$), the Shame item ($r = .37$), the Guilt item ($r = .29$), and the Stress item ($r = .32$).

The 60-item Positive and Negative Affect Schedule – Expanded Form (PANAS-X; Watson & Lee, 1994) assesses participants' emotional states during the past few weeks. The scale consists of thirteen subscales assessing positive affect, negative affect, fear, hostility, guilt, sadness, joviality, self-assurance, attentiveness, shyness, fatigue, serenity, and surprise. Participants rated their agreement with each feeling or emotional state using a 5-point scale ranging from 1, *very slightly or not at all*, to 5, *extremely*. Subscale scores were obtained by summing across the items for each factor. For the purposes of this study, only the negative affect subscale was used.

The PANAS-X has been shown to have a high level of internal consistency. Watson and Lee (1994) reported coefficient alphas that ranged from .83 to .90. Cronbach's alpha for the negative affect scale in the current study was .87. In addition, two-month test-retest reliability ranged from .64 to .71. In terms of convergent validity, the PANAS-X negative affect scale was

correlated .56 with the Beck Depression Inventory and .51 with the STAI State Anxiety Scale. Further, a comparison of the convergence of self-ratings with the ratings of well-acquainted peers showed a self-peer convergence of .48 for positive affect and .36 for negative affect.

Internalization of Sociocultural Beliefs

The 19-item Beliefs About Attractiveness Scale-Revised (BAA-R; Petrie, Rogers, Johnson, & Diehl, 1996) measures women's endorsement of U.S. societal values concerning attractiveness and beauty. The BAA-R consists of two factors – Importance of Being Physically Fit (9 items) and Importance of Being Attractive and Thin (10 items). Participants rated their agreement with each item using a 7-point Likert scale ranging from 1, *Strongly Disagree* to 7, *Strongly Agree*. Factor scores were obtained by summing across the items for each factor and dividing by the number of items in the factor. Total factor scores range from 1, *low internalization* to 7, *high internalization*.

Petrie et al. (1996) reported internal consistency reliabilities of .85 for the Importance of Being Physically Fit factor and .85 for the Importance of Being Attractive and Thin factor. Cronbach's alphas for the current study ranged from .79 to .82 for the Importance of Being Physically Fit factor and from .84 to .87 for the Importance of Being Attractive and Thin. In addition, Petrie et al. provided evidence for the construct validity of the BAA-R. The Importance of Being Physically Fit and the Importance of Being Attractive and Thin factor were significantly correlated with concern for body shape as measured by the Body Shape Questionnaire (BSQ; $r = .44$; $r = .42$, respectively) as well as importance of appearance/grooming time and satisfaction with appearance as measured by the Multidimensional Body-Self

Relations Questionnaire, (MBSRQ; Appearance Orientation factor, $r = .24$; MBSRQ Appearance Evaluation factor, $r = -.26$, respectively).

Body Image

The 11-item Body Parts Satisfaction Scale-Revised (BPSSR; Petrie, Tripp, & Harvey, 2002) assesses individuals' satisfaction with their bodies, focusing on specific body parts that are typically associated with dissatisfaction in females. The BPSSR consists of two factors – Satisfaction with Body (7 items) and Satisfaction with Face (4 items). For each item, individuals rated their level of satisfaction using a 6-point Likert scale, ranging from 1, *extremely dissatisfied* to 6, *extremely satisfied*. Total factor scores were obtained by summing individual item ratings and dividing by the total number of items in the factor. Total factor scores range from 1 to 6, with higher scores indicating greater satisfaction.

Petrie et al. (2002) reported internal consistency scores of .90 (Satisfaction with Body) and .72 (Satisfaction with Face). Cronbach's alphas for the current study ranged from .85 to .88 (Satisfaction with Body) and from .64 to .69 (Satisfaction with Face). Concerning validity, they reported significant correlations between the Satisfaction With Body factor and Body Mass Index ($r = -.32$), the Multidimensional Body Self-Relations Questionnaire Appearance Evaluation subscale ($r = .75$), the Body Shape Questionnaire ($r = -.75$), and the Situational Inventory of Body Image Dysphoria ($r = -.73$). In addition, significant correlations were reported between the Satisfaction with Face factor and the Multidimensional Body Self-Relations Questionnaire Appearance Evaluation subscale ($r = .44$), the Body Shape Questionnaire ($r = -.39$), and the Situational Inventory of Body Image Dysphoria ($r = -.46$).

The Multidimensional Body Self-Relations Questionnaire (MBSRQ; Cash, Winstead, & Janda, 1986) assesses attitudes and behaviors regarding physical appearance, health, and fitness. The MBSRQ consists of 6 subscales (Appearance Evaluation, Appearance Orientation, Fitness Evaluation, Fitness Orientation, Health Evaluation, and Health Orientation), the Body Areas Satisfaction Scale (BASS), and the Weight Attitude Scale. For the purposes of this study, only the 7-item Appearance Evaluation subscale (MBSRQ-AE), which measures overall appearance satisfaction, was used. Participants responded to each item using a 5-point Likert scale ranging from 1, *definitely disagree* to 5, *definitely agree*. A total score was formed by summing across the item scores and dividing by the total number of items. Lower scores indicate negative body image.

Using a sample of females, Cash (1994) reported an internal consistency reliability of .85 and a test-retest reliability of .90 for the MBSRQ-AE. Cronbach's alpha for the current study ranged from .87 to .88. Regarding validity, Petrie et al. (1996) reported significant correlations between the MBSRQ-AE and bulimic symptoms ($r = -.49$), self-esteem ($r = .46$), depression ($r = -.29$), internalization of sociocultural values concerning attractiveness ($r = -.25$), and concern with body shape ($r = -.64$).

Eating Disturbances

The 36-item Bulimia Test-Revised (BULIT-R; Thelen, Mintz, & Vander Wal, 1996) assesses the symptoms of bulimia nervosa defined in the *DSM-IV* (APA, 1994). The BULIT-R consists of 28 scored items reflecting *DSM-IV* criteria and 8 unscored items relating to specific weight-control behaviors. Participants scored each item on a 5-point Likert scale, ranging from 1, absence of a disturbance to 5, extreme disturbance. Total scores range from 28 to 140, with

higher scores indicating greater endorsement of bulimic attitudes and behaviors such as binge eating, purging, perceived loss of control over eating, and a concern with body weight and shape.

Thelen et al. (1996) reported an internal consistency reliability of .98. Cronbach's alpha for the current study ranged from .94 to .96. Regarding validity, McCarthy, Simmons, Smith, Tomilson, and Hill (2002) reported significant correlations between BULIT-R scores and self-monitoring measures of bulimic symptoms. Further, the BULIT-R yielded a positive predictive value of .81, a negative predictive value of .98, a specificity of .96, and a sensitivity of .91 (Thelen et al., 1996).

The 26-item version of the Eating Attitudes Test (EAT-26; Garner, Olsted, Bohr, & Garfinkel, 1982) assesses different levels and types of eating disordered behavior (Argas, Schneider, Arnow, & Raeburn, 1989), including Dieting tendencies, Bulimia and Food Preoccupation, and Oral Control (self-control in the presence of food and perceived pressure to eat or gain weight). Participants responded to each item with one of six choices – never (0), rarely (0), sometimes (0), often (1), very often (2), or always (3) (Garner et al., 1982). Total scores on the EAT-26 range from 0 to 78, with higher scores indicating greater disturbances in eating attitudes and behaviors (Garner et al., 1982).

Garner et al. (1982) reported a high internal consistency reliability (.90) for the EAT-26. Cronbach's alpha for the current study ranged from .85 to .86. Concerning validity, Mintz and O'Halloran (2000) reported a significant correlation ($r = .79, p < .01$) between the EAT-26 scores and group membership (eating disorder or non-eating disorder). Further, Mintz and O'Halloran revealed a positive predictive value of .79, a negative predictive value of .94, a specificity of .94, and a sensitivity of .77 when the EAT-26 was used to differentially diagnose individuals with or without a *DSM-IV* eating disorder.

Social Desirability

The Marlowe-Crowne Social Desirability Scale (MCSD; Crowne & Marlowe, 1960) measures social desirability or “faking good,” a form of response bias encountered with self-report measures. A short version of the MCSD, Form B (Reynolds, 1982) consisting of 12 items was used. Participants were presented a list of culturally approved behaviors that occur infrequently and asked to respond to each item using a true-false format. Responses were scored dichotomously with total scores ranging from 0 to 12. Higher scores indicate greater social desirability.

Reynolds (1982) reported adequate reliability and validity for Form B of the MCSD. A reported reliability of .76 compares favorably with the reliability of the long version. Cronbach’s alpha for the current study was .77. Reynolds also observed a significant correlation between Form B and the Edwards Social Desirability Scale ($r = .38, p < .001$). Reynolds reported that this correlation was consistent with Crowne and Marlowe’s (1960) comparison between the MCSD and the Edwards scale. Additionally, Loo and Thorpe (2000) indicated that Form B was one of the best fitting short versions of the MCSD.

Procedures

For the initial data collection, individuals were solicited for participation between the eighth and the twelfth week of the fall 2003 semester. Participants completed the demographic survey, PBI, ISCRLE, PSS, 10-item Appraisal/Coping questionnaire, VAMS, BAA-R, BPSS-R, MBSRQ-AE, BULIT-R, EAT, and SDS individually and in small groups. After obtaining written consent for participation, each participant was given a numbered packet containing the self-report questionnaires to complete by hand. The participants were first asked to complete a brief

demographic questionnaire and then the remaining questionnaires, which were counterbalanced to control for ordering effects. Participants were requested to refrain from looking ahead at subsequent questionnaires in order to further control for ordering effects. Eligible participants were offered either research credit in their undergraduate psychology courses consistent with the guidelines established by the Department of Psychology or documentation of their participation to be used for community involvement credit.

Participants who sufficiently completed the initial data collection and who provided consent to be contacted for additional participation were contacted again during the 6th to 8th week of the spring 2004, fall 2004, and spring 2005 semesters and asked to complete subsequent questionnaires consisting of an abbreviated demographic questionnaire (age, marital status, living arrangements, current weight, ideal weight, GPA, credits enrolled, sorority involvement), PANAS-X, VAMS, BAA-R, MBSRQ, BPSS-R, BULIT-R, and EAT. Participants had the option of completing the subsequent questionnaires either by hand or by completing a secure online version of the questionnaire. For the spring 2004 data collection, 92.6% ($N = 325$) completed the questionnaire using the secure online version. For the subsequent data collections 100% of participants completed the questionnaire using the secure online version. All participants were entered into a drawing for one of several \$25 giftcards to be distributed following each data collection. In addition, eligible participants were offered research credit in their undergraduate psychology courses consistent with the guidelines established by the Department of Psychology or documentation of their participation to be used for community involvement credit.

Data Analysis

Structural Equation Modeling

Structural equation modeling (SEM) was used to test the model proposed in this study. SEM is a multivariate statistical procedure that is used to estimate, analyze and test models that specify causal relationships among a set of variables (Hoyle, 1995). The researcher proposes a theoretical Model and tests whether the observed data is consistent with the proposed hypothesis. SEM is used to estimate the unknown parameters of the Model and to test the goodness of fit of the hypothesized model to the sample data. The EQS Structural Equations Program (Bentler, 1995) was used to perform the SEM analyses in this study. EQS uses the maximum likelihood (ML) or the generalized least squares (GLS) procedure to estimate the model parameters. EQS utilizes both a mathematical and a statistical approach in the analysis of structural equation models, incorporating a variety of covariance models, utilizing matrix algebra, allowing for the estimation of parameter values, and testing the models using traditional multivariate normal theory as well as more general distribution theories (Bentler, 1995; Bentler, 1989; Bentler and Weeks, 1979; Browne, 1982; Chamberlain, 1982)

The primary index of fit is the chi-square (χ^2) goodness of fit test. The chi-square test assesses the degree to which the proposed model “fits” the observed data from the sample (Hoyle, 1995). Typically, non-significant chi-square values indicate good model fit, suggesting that the proposed model is a good representation of the causal structure of the observed data. In addition, the chi-square test provides a *p*-value indicating the likelihood of observing the sample data obtained in the study given that the model were correct in the population. However, the chi-square test has several limitations. Large sample sizes have strong statistical power, increasing the probability of detecting trivial differences between the reproduced model data and the

observed data, resulting in a significant chi-square value. This increases the likelihood of making a Type II error, since the model may still be a valid representation of the observed data despite the significant chi-square value. In addition, the chi-square distribution may not be as expected if the assumption of multivariate normality is violated. In this case, a model might be rejected because the distribution characteristics of the data are not normal even though the model is conceptually valid. Finally, the more complex the proposed model is, the more likely it is to produce a good fit. To address these problems, Bentler (1980) suggests using additional methods to evaluate goodness of fit.

EQS provides three relative fit indices: the normed fit index (NFI; Bentler & Bonnett, 1980), the non-normed fit index (NNFI; Bentler & Bonnett, 1980), and the comparative fit index (CFI; Bentler, 1990). All three indices compare the proposed model to a null model. The NFI uses the null model to represent the proportion of total covariance among the observed variables explained by the proposed model (Mulaik et al., 1989). NFI values range from 0 to 1, with values above .90 indicating a fit approaching a saturated model (complete fit with the observed data). Similar to the NFI, the NNFI has the advantage of being unaffected by sample size (Dunn, Everitt, & Pickles, 1993). In addition, it contains a penalty function based on the number of estimated parameters to control for model complexity (Hu & Bentler, 1995). Like the NFI, values above .90 indicate a good fit; however, Hu and Bentler (1999) suggested that values greater than or equal to .95 as the cutoff for good model fit. The NNFI values can exceed 1 and fall below 0. The CFI also avoids underestimation of fit due to sample variability. CFI values range from 0 to 1, with values above .90 indicating better fit. Both the NNFI and the CFI may perform better with non-normal data (Bentler, 1989).

EQS also provides two absolute fit indices: the standardized root mean square residual (SRMR) and the root mean square error of approximation (RMSEA). Both indices estimate the degree to which the tested model reproduces the observed data. The SRMR is the average difference between the predicted and the observed variances and covariances in the proposed model, based on standardized residuals (Ullman, 1996). The smaller the SRMR, the better the model fit. Values below .08 suggest that the model is correctly specified. A value of 0 indicates a perfect fit. The RMSEA is a measure of parsimony. The RMSEA corrects for model complexity by containing a penalty function based on the number of estimated parameters (Browne & Cudeck, 1993). In addition, EQS reports confidence intervals for the RMSEA. Hu and Bentler (1999) suggest that values less than or equal to .06 indicate good model fit.

Comparing parameter values with their standard errors (SE) allows for the assessment of the parameter values of each model. Large parameter values and small standard error indicates that a parameter is significant in a model. In addition, Bentler (1989) recommends a simple univariate test (a Z-test) to determine whether a parameter estimate is consistent with a population value. This is accomplished by dividing the parameter estimate by its SE.

When distributional assumptions of normality are violated, the chi-square estimates, estimates of parameters, and SEs may be affected (Anderson & Gerbing, 1988). Fortunately, EQS makes it possible to compute robust estimates of the SE's and test statistics. Chou, Bentler, and Satorra (1989) reported that these robust estimates may be more reliable when normality assumptions are violated. These robust estimates include the Satorra-Bentler chi-square (Satorra & Bentler, 1989) and the robust comparative fit index (RCFI). The Satorra-Bentler chi-square is an adjustment to chi-square, which penalizes chi-square for the amount of kurtosis in the data. It is an adjusted chi-square statistic that attempts to control for the bias that is introduced when data

are non-normal in distribution. Hu, Bentler, and Kano (1992) reported that the use of the Satorra-Bentler chi-square might provide a better estimate of model fit when the distributional assumptions are questionable. The RCFI is a variation of the CFI using robust parameter values. In addition, EQS uses Mardia's (1970) kurtosis test of multivariate normality to assess the effect of outlier and answer the question of normally distributed variables in the data. The Satorra-Bentler chi-square, the NFI, the NNFI, the CFI, the RCFI, the SRMR, and the RMSEA were used to assess the overall fit of the proposed model.

The structural equation modeling process centers around two steps: validating the measurement model and fitting the structural model (Anderson & Gerbing, 1988). Validating the measurement model is accomplished through confirmatory factor analysis (CFA). CFA is used to insure that the measured variables load on the latent constructs as specified by the researcher. Measured variables that fail to contribute unique variance to the latent construct are dropped from the measurement model. The proposed measurement model is displayed in Figure 1.

After the measurement model has been validated, the structural model is tested adding the structural paths to the confirmed measurement model. At this point, the researcher is testing the extent to which the covariance matrix predicted by the model corresponds to the observed covariances in the data. When a hypothesized and tested model results in unfavorable indicators of fit, post hoc tests can also be run to identify misspecified parameters in the model, and the model can be modified. EQS uses the Wald and Lagrange Multiplier (LM) tests (Bentler & Dijkstra, 1985; Lee, 1985) to provide information about the amount of χ^2 change that would result if one or more parameters were respecified. The Wald test indicates which free parameters may be dropped to improve model fit. The Wald test indicates which of the fixed or constrained

parameters could be freed to improve the fit of the model. Only changes that will contribute to significant changes in the fit of the Model and are consistent with theory are considered.

The EQS analysis produces a standardized solution for both the measurement model and the structural model. These solutions contain an equation with the structural coefficients and the measurement error. The structural coefficients are similar to the beta weights in regression analyses. They represent the direction and magnitude of the relationship among the latent variables. The measurement error is the proportion of variance of the observed variables that measures something other than the latent variable. The measurement error serves as a measure of reliability. Each observed variable has a measurement error associated with it, and each factor has an error variance referred to as a “disturbance term.”

Structural equation analysis rarely involves testing a single hypothesized model (Hoyle & Panter, 1995). Alternative models may be compared by examining the incremental improvements of the fit indices, or preferably, by conducting chi-square difference tests. A decrease in chi-square, SRMR, or RMSEA indicates an improvement in model fit of the alternative model over the originally specified model. Similarly, increases in the NFI, NNFI, CFI, or RCFI indicate improved fit. For the purposes of this study, two hypothesized models will be specified a priori and tested. The hypothesized models are displayed in Figure 2 and Figure 3.

CHAPTER 3

RESULTS

Descriptive Analysis

Initially, the raw data from the first data collection were examined for missing values. One hundred forty-two cases (29.3%) were missing individual values on at least one of the measured variables. In five of the cases, values were missing for the entire measured variable, and these cases were dropped from the raw data set. Structural equation modeling requires a complete data set for each case. To avoid losing the data from the remaining cases with missing values, the SPSS Missing Values Analysis™ (SPSS, Inc., Chicago, Illinois, www.spss.com) procedure using the estimated series means, was used to replace the missing values in these cases. Data from the second data collection were examined and added to the raw data set. The secure online questionnaire prevented participants from submitting their results with incomplete data and there were no missing data points from the questionnaires that were submitted by students who opted to complete a paper version. Thus, there was no need to replace any missing values. However, 129 participants who participated in the initial data collection did not participate in the second data collection, and these cases were dropped from the raw data set. Data from third and the fourth data collection were examined. All participants used the secure online questionnaire to submit data during the third and fourth data collections, so there were no missing data values. However, only 173 of the participants who participated in both the initial data collection and the second data collection participated in the third data collection. Two hundred thirty-eight of the participants who participated in both the initial data collection and the second data collection also completed the questionnaire during the fourth data collection. In order to maintain an adequate sample size of complete cases for structural equation modeling,

only the data from the fourth data collection were added to the raw data set. There were 112 participants who did not participate in the fourth data collection but had previously completed the questionnaires during both the first and second data collections, so these cases were dropped from the raw data set.

Measured variables were then totaled from the individual data values, and descriptive statistics were run to determine the means and standard deviations of all the variables. In addition, internal consistency reliability analyses were run for each of the measured variables, and the distributional properties were examined for normality for multivariate analysis. (See Table 1 for means, standard deviations, skewness, kurtosis, and Cronbach's alphas for all measured variables). With the exception of the Eating Attitudes Test-26 (EAT-26), skewness and kurtosis for all measured variables appeared to be in acceptable ranges. The EAT-26 was positively skewed in the sample. However, it was detected that cases 66 and 220 were outliers. With the removal of these cases from the structural equation modeling analysis, the skewness of all measured variables, including the EAT-26, fell within an acceptable range, and the kurtosis of the measured variables were not unusually large. Therefore, the data demonstrated adequate multivariate normality for the maximum likelihood procedure (Hoyle, 1995).

Social desirability in responding to the questionnaire was evaluated. Frequency reports indicated that 18.1% ($n = 43$) of the participants had responded to the items in a socially desirable manner (score of 10-12). However, internal consistency for the SDS was poor (.62). Correlations between the SDS and the other measured variables also were examined. Significant correlations did exist between the measured variables and the SDS (see Table 3). However, the correlations were small and the shared variances were less than 10%. Thus, social desirability does not appear to be a concern, and data from all participants were retained for data analysis.

Next, attrition analyses were conducted to determine whether or not any systematic dropout had occurred between Time 1 and Time 4. T-tests were conducted for all demographic variables and all measured variables, comparing the final sample of participants to those who did not full participate in the study beyond the Time 1 data collection. Those participants who dropped out of the study did not differ from those who remained on any of these factors (all p values $> .05$). Table 2a presents the means, standard deviations, and t-test statistics for each group for all continuous demographic variables and all measured variables. Table 2b presents the chi-square statistics for all categorical demographic variables.

Correlations were run to examine the relationships among the measured variables. Specifically, correlations were inspected to ensure that the measured variables selected to load on specific latent variables grouped together as expected. Only the Perceived Stress Scale (PSS) did not group with its associated measured variables. Initially, it was expected that the PSS would load on the History of Stress Factor and correlate with the subscales of the Inventory of College Students' Life Experiences (ICSRLE). The PSS failed to correlate significantly with the subscales of the ICSRLE; however, it did correlate significantly with the measures of Appraisal/Coping, suggesting that the PSS appeared to be a better indicator of one's appraisal of stressful events and their ability to cope with those events than an indicator of prior stressful occurrences.

Correlations also were run to identify and examine any unusual and/or unexpected relationships between demographic variables and measured variables. As expected, significant correlations existed between the participant's body mass index (BMI) scores (actual BMI and ideal BMI) and measures of body satisfaction and internalization of sociocultural beliefs regarding the thin-ideal. All correlations were in the expected directions. For example, Ideal BMI

scores were negatively correlated with measures of positive body image. However, significant correlations did not exist between the participant's BMI and measures of eating disturbance. The demographic variables income, race/ethnicity, and sorority status also displayed significant correlations with several of the measured variables, and these relationships were further examined. Income level in the family of origin was significantly, though only moderately, correlated with measures of parental attachment, measures of perceived stress, and measures of internalization of the thin-ideal and eating disturbances. Specifically, higher levels of annual income in the family of origin were associated with more secure parental attachment, perception that life events are less stressful, increased internalization of the thin-ideal, and more disordered eating behaviors and attitudes. Sorority status also was correlated significantly with parental attachment relationships, internalization of the thin-ideal, body image, eating disordered behavior and attitudes, and negative affect. Specifically, involvement in a sorority was associated with more secure parental attachment relationships, more disordered eating behaviors and attitudes, stronger belief in the thin-ideal, negative body image, and feelings of happiness. Significant, though small, negative correlations also existed between race/ethnicity and measures of secure parental attachment and measures of internalization of the thin-ideals. (See Table 3 for a correlation matrix of the measured variables and the demographic variables).

Frequency reports of body size and body satisfaction were examined. Both current and ideal body mass index (BMI) scores were calculated for each participant. According to the Center for Disease Control (2001), 16.8% ($n = 40$) of the participants at Time 1 were underweight (BMI <19), 62.2% ($n = 148$) were normal weight (BMI 19.01 to 25), 16% ($n = 38$) were overweight (BMI 25.01 to 30), and 5% ($n = 12$) were obese (BMI > 30). Furthermore, the participants' ideal BMI scores at Time 1 indicate that 26.9% ($n = 64$) desired a BMI score that

would classify them as underweight, whereas only 0.8% ($n = 2$) desired a BMI score that would classify them as overweight or obese. At time 2, 14.9% ($n = 35$) of the participants were underweight (BMI <19), 61.3% ($n = 146$) were normal weight (BMI 19.01 to 25), 16.2% ($n = 39$) were overweight (BMI 25.01 to 30), and 7.7% ($n = 18$) were obese (BMI > 30).

Furthermore, the participants' ideal BMI scores at Time 2 indicate that 24.3% ($n = 57$) desired a BMI score that would classify them as underweight, while only 0.8% ($n = 2$) desired a BMI score that would classify them as overweight or obese. At Time 4, 13.9% ($n = 33$) of the participants were underweight (BMI <19), 59.1% ($n = 141$) were normal weight (BMI 19.01 to 25), 20.20% ($n = 48$) were overweight (BMI 25.01 to 30), and 6.8% ($n = 16$) were obese (BMI > 30). Furthermore, the participants' ideal BMI scores at Time 4 indicate that 22.4% ($n = 53$) desired a BMI score that would classify them as underweight, while only 0.4% ($n = 1$) desired a BMI score that would classify them as overweight or obese.

Disordered eating attitudes and behaviors also were examined. At Time 1, 5% ($n = 12$) of the women exceeded the recommended cutoff score (104 or greater) for a diagnosis of bulimia nervosa on the BULIT-R. However, 8.8% ($n = 21$) scored above an 85, the cutoff score recommended for researchers who are using the BULIT-R as a screening tool (Thelen, Farmer, Wonderlich, & Smith, 1991). In addition 11.3% ($n = 27$) of the women exceeded the recommended cutoff score (≥ 20) for consideration of a diagnosis of an eating disorder (anorexia nervosa, bulimia nervosa, EDNOS) on the EAT-26. At Time 2, 1.3% ($n = 3$) of the women exceeded the recommended cutoff score (104 or greater) for a diagnosis of bulimia nervosa on the BULIT-R. However, 5% ($n = 12$) scored above an 85, the cutoff score recommended for researchers who are using the BULIT-R as a screening tool (Thelen et al., 1991). In addition 10.1% ($n = 24$) of the women exceeded the recommended cutoff score (≥ 20) for consideration

of a diagnosis of an eating disorder (anorexia nervosa, bulimia nervosa, EDNOS) on the EAT-26. At Time 4, 4.4% ($n = 8$) of the women exceeded the recommended cutoff score (104 or greater) for a diagnosis of bulimia nervosa on the BULIT-R. However, 8.0% ($n = 19$) scored above an 85, the cutoff score recommended for researchers who are using the BULIT-R as a screening tool (Thelen et al., 1991). In addition 10.9% ($n = 26$) of the women exceeded the recommended cutoff score (≥ 20) for consideration of a diagnosis of an eating disorder (anorexia nervosa, bulimia nervosa, EDNOS) on the EAT-26.

Frequency of occurrence of eating disordered behaviors was examined using 8 key items on the BULIT-R. At Time 1, to help control their weight, 3% ($n = 7$) and 2.1% ($n = 5$) of the women described using diuretics and using laxatives or suppositories at least 2-3 times a month, respectively. 5% ($n = 12$) of the women reported intentionally vomiting at least once a month. A greater percentage of the women reported exercising more than one hour a day in order to burn calories (20.1%; $n = 48$) and going on a diet at least once in the past year (47.1%; $n = 112$) as means by which to control their weight. In addition, 49.6% ($n = 118$) reported rapidly eating large amounts of food at least 2-3 times a month, and 16.4% ($n = 39$) described that eating as binge eating. Of the women that admitted to engaging in binge eating behavior, 13.9% ($n = 33$) reported that they had been binge eating for at least 3 months. (See Table 4 for frequency reports of eating disordered behaviors on the BULIT-R). At Time 2, to help control their weight, 3.3% ($n = 8$) and 1.6% ($n = 4$) of the women described using diuretics and using laxatives or suppositories at least 2-3 times a month, respectively. 5.5% ($n = 13$) of the women reported intentionally vomiting at least once a month. A greater percentage of the women reported exercising more than one hour a day in order to burn calories (23.1%; $n = 55$) and going on a diet at least once in the past year (42.0%; $n = 101$) as means by which to control their weight. In

addition, 41.6% ($n = 99$) reported rapidly eating large amounts of food at least 2-3 times a month, and 25.2% ($n = 60$) described that eating as binge eating. Of the women that admitted to engaging in binge eating behavior, 19% ($n = 46$) reported that they had been binge eating for at least 3 months. (See Table 4 for frequency reports of eating disordered behaviors on the BULIT-R). At Time 4, to help control their weight, 3% ($n = 7$) and 2.1% ($n = 5$) of the women described using diuretics and using laxatives or suppositories at least 2-3 times a month, respectively. 6.8% ($n = 16$) of the women reported intentionally vomiting at least once a month. A greater percentage of the women reported exercising more than one hour a day in order to burn calories (12.6%; $n = 30$) and going on a diet at least once in the past year (41.6%; $n = 99$) as means by which to control their weight. In addition, 35.7% ($n = 85$) reported rapidly eating large amounts of food at least 2-3 times a month, and 24.8% ($n = 59$) described that eating as binge eating. Of the women that admitted to engaging in binge eating behavior, 20.6% ($n = 49$) reported that they had been binge eating for at least 3 months. (See Table 4 for frequency reports of eating disordered behaviors on the BULIT-R).

The proportion of women experiencing stress related to the transition to college, difficulties related to coping with those stressors, also was examined. Agreement with the experienced stressors and difficulty coping with the stressors was based on a response of “agree” or “strongly agree” on the individual items of the Appraisal/Coping measure. Since starting school that semester, 60.9% ($n = 145$) of the participants agreed that they were experiencing stress due to academic demands, and 28.1% ($n = 67$) acknowledged difficulty coping with the academic demands. Regarding living situation, 39.5% ($n = 94$) of participants agreed that they had been experiencing stress since starting school, and 26.1% ($n = 62$) admitted to difficulty coping with their living situation. When asked about the demands associated with making new

friends, 32.8% ($n = 78$) agreed that they had experienced stress since the beginning of the semester, and 21.4% ($n = 51$) reported difficulty coping with the demands of making new friends. Nearly 43% ($n = 101$) of the participants reported experiencing stress associated with having or not having romantic relationships since the beginning of school, and 36.1% ($n = 86$) acknowledged difficulty coping with having or not having romantic relationships. When asked about weight gain since the beginning of the semester, 31.5% ($n = 75$) of the participants agreed that they had been experiencing stress, and 26.4% ($n = 63$) reported difficulty coping with weight gain. Further, almost half of the women ($n = 113$) reported feeling at least moderately sad or depressed and over 75% indicated feeling at least moderately stressed and anxious during their first semester.

Finally, parental attachment relationships also were examined. The recommended cutoff scores of ≥ 27 and ≥ 24 were used to define high maternal care and paternal care, respectively, and the recommended cutoff scores of ≥ 13.5 and ≥ 12.5 were used to define high maternal overprotection and paternal overprotection, respectively. With regards to the mother-child relationship, 50% ($n = 119$) of the relationships met the criteria for optimal bonding (high care, low overprotection) and were classified as secure attachment relationships. The remaining 50% ($n = 119$) of mother-child relationships were classified as insecure attachment relationships: (a) 53.8% ($n = 64$) fell in the affectionate constraint category (high care, high overprotection), (b) 34.9% ($n = 42$) met the criteria for the affectionless control category (low care, high overprotection), and (c) 10.9% ($n = 13$) were placed in the neglectful parenting category (low care, low overprotection). Looking at the father-child relationship, 43.3% ($n = 103$) of the relationships met the criteria for optimal bonding (high care, low overprotection) and were classified as secure attachment relationships. The remaining 56.7% ($n = 135$) father-child

relationships were classified as insecure attachment relationships: (a) 42.2% ($n = 57$) fell in the affectionate constraint category (high care, high overprotection), (b) 37% ($n = 50$) met the criteria for the affectionless control category (low care, high overprotection), and (c) 20.7% ($n = 28$) were placed in the neglectful parenting category (low care, low overprotection). Overall, 31.1% ($n = 74$) of participants reported secure parent-child relationships with both their mother and their father, whereas 37.8% ($n = 90$) described insecure parent-child relationships with both parents.

Analyses of Models with All Measured Factors

Measurement Model

The confirmatory analysis of the measurement model was begun on the construct of parental attachment. Initially, the Parental Bonding Instrument (PBI) Maternal Care, Paternal Care, Maternal Overprotectiveness, and Paternal Overprotectiveness scales were tested on the Parental Attachment Factor (Time 1 only). The Paternal Care scale appeared to be too highly associated with other measured variables, and it was dropped from the model due to an inability to contribute common variance to the latent construct it was intended to identify. In the measurement model, the Maternal Care scale loaded negatively on the Parental Attachment Factor, and the Maternal Overprotectiveness and Paternal Overprotectiveness scales loaded positively on the Parental Attachment Factor, designating the overall Parental Attachment as representative of insecure parental attachment.

Next, the history of stress construct was introduced to the measurement model. The Developmental Challenge subscale, Time Pressure subscale, Academic Alienation subscale, Romantic Problems subscale, Assorted Annoyances subscale, General Social Mistreatment

subscale, and Friendship Problems subscale of the Inventory of College Students' Recent Life Experiences (ICSRLE) were tested on the History of Stress Factor (Time 1 only). Both the Assorted Annoyances subscale and the General Social Mistreatment subscale failed to load on the History of Stress Factor due to an inability to contribute common variance to the latent construct. Furthermore, the Assorted Annoyances subscale had poor internal consistency reliability (.57), and both subscales were dropped from the measurement model. All remaining scales loaded appropriately on the History of Stress Factor. However, when the standardized solution was examined, the Romantic Problems subscale and the Friendship Problems subscale had very low loadings, and these also were dropped from the measurement model. As a result, the Developmental Challenge, Academic Alienation, and Time Pressure subscales of the ICSRLE best represented the History of Stress Factor, and these measures loaded positively on the construct, indicating that this factor measured greater experiences with stressful events.

The appraisal and coping process construct was then added to the existing measurement model. Initially, it was hypothesized that the total primary appraisal score (the sum of the five items assessing appraisal of stressors) and the total secondary appraisal score (the sum of the five items assessing coping with stressors) would load on the Appraisal/Coping Factor (Time 1 only). However, due to the poor internal consistency reliabilities of the primary appraisal score (.52) and secondary appraisal score (.48), the primary appraisal item and secondary appraisal item for each dimension (academic demands, friendship demands, living arrangement demands, romantic relationship demands, and weight gain concerns) were paired and tested, along with the Perceived Stress Scale (PSS), on the Appraisal/Coping Factor. All measures loaded appropriately on the Appraisal/Coping Factor. However, when the standardized solution was examined, the paired items measuring appraisal and coping of stressors related to weight gain concerns,

romantic relationship demands, living arrangements, and friendship demands had very low loadings, and these were dropped from the measurement model. Thus, the PSS and the paired items measuring appraisal and coping of stressors related to academic demands best represented the Appraisal/Coping Factor. These factors loaded positively onto the construct, designating it as representative of high levels of perceived stress and difficulty coping with stressors.

The three constructs representing body image (Body Image Time 1, Body Image Time 2, and Body Image Time 4) were then introduced to the existing measurement model, testing the Body Parts Satisfaction Scale (BPSS) Body Factor, BPSS Face Factor, and the Multidimensional Body Self Relations Questionnaire-Appearance Evaluation subscale (MBSRQ-AE) as measured at each of the three time points. Per Bollen's (1989) recommendation, the measurement errors for the corresponding indicators at the three points in time were correlated. Due to poor internal consistency (.64 to .69) and high associations with other measured variables, suggesting failure to contribute common variance to the Body Image Factor at any of the three time points, the BPSS Face Factor for each time point was dropped from the measurement model. The remaining variables for each time point loaded strongly on the corresponding Body Image Factor, reflecting positive body image.

Following the addition of the body image constructs, the constructs representing internalization of the thin-ideal (Internalization of Thin-Ideal Time 1, Internalization of Thin-Ideal Time 2, and Internalization of Thin-Ideal Time 4) were tested by adding the Beliefs About Attractiveness (BAAR) Physically Fit subscale and BAAR Attractive and Thin subscale as measured at each time point. The measurement errors for the corresponding indicators at the three points in time were correlated. For each time point, both scales loaded appropriately on the respective Internalization of Thin-Ideal Factor, so the eating disturbance constructs (Eating

Disturbance Time 1, Eating Disturbance Time 2, and Eating Disturbance Time 4) were then added to the model with the measurement errors for the corresponding indicators at the three points in time being correlated. Initial attempts to test the Bulimia Test – Revised (BULIT-R) and Eating Attitudes Test – 26 (EAT-26) on the respective Eating Disturbance Factors resulted in a non-significant error variance for the BULIT-R at Time 1 and at Time 2. Utilizing Bollen’s (1989) procedure for estimating measurement error, the error variance for each was set to 1 minus the scale reliability times the variance for the measure at each particular time point. This resulted in an estimated value of 19.95 for the error variance for the BULIT-R at Time 1 and 22.61 for the error variance for the BULIT-R at Time 2. This allowed the variables for each time point to load appropriately on the corresponding Eating Disturbance Factor, designating the factors as representative of greater eating pathology.

Finally, the negative affect constructs (Negative Affect Time 1, Negative Affect Time 2, Negative Affect Time 4) were added to the measurement model with the measurement errors for the corresponding indicators at the three points in time being correlated. Initial attempts to load all seven of the individual Visual Analogue Mood Scale (VAMS) items (Sad, Happy, Shame, Guilt, Confidence, Anxious, Stressed) for each of the three time points were unsuccessful and resulted in the Negative Affect Factors being constrained at the upper bound with the Appraisal/Coping Factor, indicating that the parameter estimates were not within the specified bounds of the program. Following modifications to the constructs, the VAMS Sad or Depressed, VAMS Happy, and VAMS Stressed items for each time point loaded appropriately on the corresponding latent construct and appeared to be the best representations of the Negative Affect Factors. The VAMS Sad or Depressed item and VAMS Stressed item loaded positively on each factor, whereas the VAMS Happy item loaded negatively on each factor. In addition, the Positive

and Negative Affect Scale – Extended Version (PANAS-X) Negative Affect Scale loaded positively on the Negative Affect Factor Time 4. Recall that this variable was only measured at Time 4. Thus each factor reflected negative affect.

Using the EQS program for structural equation modeling and the Maximum Likelihood (ML) estimation procedure to estimate the parameters of the model, the confirmatory factor analysis (CFA) supported the resulting measurement model, and the overall fit of the data was good (see Table 11). The standardized parameter estimates (factor loadings and error variances) for the measurement Model Are displayed in Table 5, and the final representation of the measurement model is displayed in Figure 4. Correlations among the factors are presented in Table 17.

Structural Model

After validating the measurement model, the model was modified to include the structural pathways. The pathways between the latent constructs were added to the measurement model, and all paths were tested simultaneously to identify the best fitting and most parsimonious causal model.

Investigating a multidimensional model of eating disorder development, Bradford (2004) demonstrated support for 2 models explaining the relationship among the constructs specified in this study at a single point in time. The first model supported a linear pathway of eating disorder development in which direct paths existed from parental attachment and history of stress to appraisal/coping, from appraisal/coping to negative affect, from negative affect to body dissatisfaction, and from body dissatisfaction to eating pathology. A path also was included from internalization of the thin-ideal to body image and to eating pathology. The second model

supported two separate paths in the development of eating disorders. In the first path, attachment processes and history of stress directly influenced the appraisal coping process, which directly influenced negative affect to lead to the development of eating disturbances. In the second path, sociocultural influences directly influenced body image and both sociocultural influences and body image influenced the development of eating disorders.

This study was intended to be an extension of this work, testing the relationships among these constructs over time to determine causality. Thus, the following relationships had been hypothesized a priori and then tested. In structural Model A (see Figure 5), parental attachment and history of stress were hypothesized to have a direct effect on the appraisal/coping process which was hypothesized to predict negative affect. It was then hypothesized that negative affect and internalization of the thin-ideal would predict body image. Internalization of the thin-ideal and body image were hypothesized to predict eating disturbance. In structural Model B (see Figure 6), parental attachment and history of stress were hypothesized to have a direct effect on the appraisal/coping process which was hypothesized to predict negative affect, and negative affect was hypothesized to predict eating disturbance. It was then hypothesized that internalization of the thin-ideal would predict body image, and both internalization of the thin-ideal and body image were hypothesized to predict eating disturbance.

To examine the predictive relationships, negative affect, body image, internalization of the thin-ideal, and eating disturbance were assessed at three time points, and a fully cross-lagged model was tested. This type of analysis included paths between the same construct measured at all times to evaluate the cross-time relative stability of the constructs. It also included both the hypothesized relationships among the variables as well as the competing hypothesis to accurately examine prediction.

Initial attempts to fit the specified structural models with all of the above mentioned pathways were unsuccessful, as the models failed to converge and produce a solution. Attempts to specify better start values for EQS were conducted by trying to fit the models in which only the hypothesized paths were tested and in which only the competing paths were tested. These models also exhibited significant problems and failed to produce results that enabled the use of the EQS output to correct any mis-specified paths.

Stevens (1996) recommends as least 15 cases per measured variable, and Bentler and Chou (1987) encourage no fewer than five cases per parameter estimate. Consequences of using smaller samples include more convergence failures (EQS is unable to reach a satisfactory solution), improper solutions, and decreased accuracy of parameter estimates. The hypothesized models include 36 measured variables, so 236 cases may result in an insufficient sample size.

In summary, the proposed measurement model, with modification, fit the data well. However, both of the proposed structural models evaluating an integration of multiple risk factors that contribute to the development of eating disorders using prospective, longitudinal data failed to converge on a solution. Attempts to employ modifications to each of the structural models also were unsuccessful.

Analysis of Models with Selected Factors

To further evaluate the models suggested by Bradford (2004) and reduce the number of measured variables and parameter estimates, the data from Time 2 and from Time 4 were tested by running each model with selected data from a single point in time. Recall that several of the constructs were only measured during the first data collection. To allow for the evaluation of the causal direct and indirect effects between these constructs and the remaining constructs in each

model, the Time 1 variables for these constructs were included in each model. These models and the results are described below.

Evaluation of Selected Factors at Time 1 and Time 2

To evaluate simplistic versions of the multidimensional models of risk factors that contribute to the development of eating disorders as measured at Time 1 and Time 2, the following factors were selected for the model: Parental Attachment, History of Stress, Appraisal/Coping, Negative Affect Time 2, Body Image Time 2, Internalization of Thin-Ideal Time 2, and Eating Disturbance Time 2. A confirmatory analysis of the measurement model was conducted and 2 structural models were tested. These models are consistent with the models presented in Bradford's (2004) examination of these same factors, but the variables measured at Time 2 have been substituted for the corresponding variables at Time 1.

Measurement model. The confirmatory analysis of the measurement model for selected factors at Time 1 and Time 2 was begun with the Parental Attachment Factor, the History of Stress Factor, and the Appraisal/Coping Factor. Recall that the measurement model presented above also began with the addition of each of these constructs. Thus, the measured variables that represent these latent constructs remain the same. The Parental Attachment Factor is representative of insecure parental attachment as measured by the Maternal Care scale, the Maternal Overprotectiveness scale, and the Paternal Overprotectiveness scales of the Parental Bonding Instrument (PBI). The History of Stress Factor is representative of greater experiences with stressful events as measured by the Developmental Challenge, Academic Alienation, and Time Pressure subscales of the Inventory of College Students' Recent Life Experiences

(ICSRLE). The Appraisal/Coping Factor is representative of high levels of perceived stress and difficulty coping with stressors as measured by the Perceived Stress Scale (PSS) and the paired items measuring appraisal and coping of stressors related to academic demands.

Next, the Body Image Time 2 Factor was introduced to the measurement model. Again, the Body Parts Satisfaction Scale (BPSS) Face Time 2 Factor failed to contribute common variance due to high associations with other measured variables. The internal consistency for this variable also was moderate (.68). The BPSS Body Time 2 Factor and the Multidimensional Body Self Relations Questionnaire-Appearance Evaluation (MBSRQ-AE) subscale Time 2 appropriately loaded on the Body Image Time 2 Factor and designated the construct as representative of positive body image.

The Internalization of Thin-Ideal Time 2 Factor was then added to the measurement model. Both the Beliefs About Attractiveness (BAAR) Physically Fit subscale Time 2 and BAAR Attractive and Thin subscale Time 2 loaded appropriately on the Internalization of Thin-Ideal Factor Time 2, designating the construct as representative of greater internalization of the thin-ideal.

The Eating Disturbance Time 2 Factor was added to the measurement model. Initial attempts to test the Bulimia Test – Revised (BULIT-R) and Eating Attitudes Test – 26 (EAT-26) on the Eating Disturbance Time 2 Factor resulted in non-significant error variance for the BAAR Attractive and Thin Subscale Time 2 and for the BULIT-R Time 2. Utilizing Bollen's (1989) procedure for estimating measurement error, the error variance for each was set to 1 minus the scale reliability times the variance for the measure. This resulted in an estimated value of .15 for the error variance for the BAAR Attractive and Thin subscale Time 2 and 19.95 for the error variance for the BULIT-R Time 2. The BULIT-R and the EAT-26 loaded appropriately on the

Eating Disturbance Time 2 Factor, designating it as representative of greater eating disordered symptomatology.

Finally, the Negative Affect Time 2 Factor was added to the measurement model. Only the Visual Analogue Mood Scale (VAMS) Sad or Depressed item, VAMS Happy item, and VAMS Stressed item loaded appropriately on the Negative Affect Time 2 Factor, designating the construct as representative of greater negative affect.

Using the EQS program for structural equation modeling and the Maximum Likelihood (ML) estimation procedure to estimate the parameters of the model, the confirmatory factor analysis (CFA) supported the resulting measurement model, and the overall fit of the data was good (see Table 11). The standardized parameter estimates (factor loadings and error variances) for the measurement Model Are displayed in Table 6, and the final representation of the measurement model is displayed in Figure 7.

Structural Model A. After validating the measurement model, the model was modified to include the structural model pathways. The pathways between the latent constructs were added to the measurement model, and all paths were tested simultaneously to identify the best fitting and most parsimonious causal model.

The following hypothesized relationships had been determined a priori and then tested in structural Model A. The Parental Attachment Factor was hypothesized to have a direct effect on the Appraisal/Coping Factor and thus an indirect effect on the Negative Affect Time 2 Factor, the Body Image Time 2 Factor, and the Eating Disturbance Time 2 Factor. The History of Stress Factor was hypothesized to have a direct effect on the Appraisal/Coping Factor and an indirect effect on the Negative Affect Time 2 Factor, the Body Image Time 2 Factor, and the Eating

Disturbance Time 2 Factor. The Appraisal/Coping Factor was hypothesized to have a direct effect on the Negative Affect Time 2 Factor and an indirect effect on the Body Image Time 2 Factor, and the Eating Disturbance Time 2 Factor. The Negative Affect Time 2 Factor was hypothesized to have a direct effect on the Body Image Time 2 Factor and an indirect effect on the Eating Disturbances Time 2 Factor. The Internalization of Thin-Ideal Time 2 Factor was hypothesized to have both a direct effect on the Eating Disturbance Time 2 Factor and an indirect effect on the Eating Disturbances Time 2 Factor through a direct effect on the Body Image Time 2 Factor. Finally, the Body Image Time 2 Factor was hypothesized to have a direct effect on the Eating Disturbance Time 2 Factor. The hypothesized structural Model A is displayed in Figure 8.

The initial attempt to fit the structural model with all of the above mentioned pathways was successful. No problems were encountered, and the testing of the structural paths supported the model. Although the chi-square statistic was significant, all other fit indices indicated a strong fit with the data (see Table 11).

As previously discussed, post hoc tests can be run to identify parameters in the model that can be modified to improve the fit of the model. EQS uses the Wald and Lagrange Multiplier (LM) tests (Bentler & Dijkstra, 1985; Lee, 1985) to provide information about the amount of χ^2 change that would result if one or more parameters were respecified. The Wald test indicates which free parameters may be dropped to improve model fit, and the Wald test indicates which of the fixed or constrained parameters could be freed to improve the fit of the model. Only modifications that will contribute to significant changes in the fit of the Model and are substantively justified are considered. Hoyle and Panter (1995) advise against the use of post hoc modifications unless there is a clear and compelling reason for the addition of the pathways. Thus, there must be a balance of theory and empiricism when model-generating statistics are

used in research. Thus, only logical (i.e., Time 2 Body Image cannot predict Time 1 Parental Attachment) and empirically-based or theoretically-based paths were considered.

The Wald test indicated that the addition of a pathway from the Appraisal/Coping Factor to the Eating Disturbances Time 2 Factor, LM $\chi^2(1, N = 236) = 15.620$ could significantly improve the fit of the model. The additional pathway from the Appraisal/Coping Factor loaded significantly on the Eating Disturbances Time 2 Factor and the fit indices indicated that this model was a good fit with the data (see Table 11).

To assess the extent to which this respecified model is an improvement over the original structural model, the difference in χ^2 between the two models was examined. The chi-square difference test ($\Delta\chi^2 = 13.69, df = 1$) was significant ($p < .001$), indicating that the respecified model produced a statistically significant and substantial improvement in model fit over the original structural model. In addition, increases were seen in the NFI, NNFI, CFI, and RCFI and decreases were observed in the SRMR and RMSEA, indicating improvement in model fit. Although the chi-square statistic for the final model was non-significant ($p < .001$), the model demonstrated good fit with the data according to all other fit indices.

Both the direct effects and the indirect effects (amount of mediation) were examined (see Table 8). With the exception of the indirect effects from the History of Stress Factor on both the Body Image Time 2 Factor and the Eating Disturbance Time 2 Factor, all effects were significant. In this respecified structural model, the significant effects explained 18.11% of the variance for the Appraisal/Coping Factor, with the direct effects from the Parental Attachment Factor accounting for 15.92% of the variance and the History of Stress Factor accounting for 2.19% of the variance. 34.56% of the variance for the Negative Affect Factor was explained by the significant effects, with the direct effect from the Appraisal/Coping Factor accounting for

29.27% of the variance and the indirect effects from the Parental Attachment Factor and the History of Stress Factor accounting for 4.67% and 0.64% of the variance, respectively. The significant indirect effects from the Parental Attachment Factor and the History of Stress Factor indicate that the relationship from each of these factors to the Negative Affect Factor is mediated by the Appraisal/Coping Factor. The significant effects also explained 27.03% of the variance of the Body Image Factor, with the direct effects from the Internalization of the Thin-Ideal Factor and the Negative Affect Factor accounting for 10.89% and 12.04% of the variance, respectively. The significant indirect effects from the Parental Attachment Factor and the Appraisal Coping Factor accounted for 0.56% and 3.53% of the variance in the Body Image Factor, respectively. Thus, the relationship between the Parental Attachment Factor and the Body Image Factor is mediated by the Appraisal/Coping Factor and the Negative Affect Factor, and the relationship between the Appraisal/Coping Factor and the Body Image Factor is mediated by the Negative Affect Factor. 38.36% of the variance of the Eating Disturbances Factor was explained by the significant direct and indirect effects. The significant direct effects from the Appraisal/Coping Factor, the Body Image Factor, and the Internalization of the Thin-Ideal Factor accounted for 5.86%, 19.10%, and 5.38% of the variance, respectively, and the significant indirect effects from the Parental Attachment Factor, the Appraisal/Coping Factor, the Negative Affect Factor, and the Internalization of the Thin-Ideal Factor accounted for 1.66%, 0.67%, 3.61%, and 2.07% of the variance, respectively. The significant indirect effects indicate that the relationship between the Parental Attachment Factor and the Eating Disturbance Factor is mediated by the Appraisal/Coping Factor, the Negative Affect Factor, and the Body Image Factor. The significant direct effect and indirect effect from the Appraisal/Coping Factor to the Eating Disturbance Factor indicates that there is a direct relationship between the two constructs and the

relationship is also mediated by the Negative Affect Factor and the Body Image Factor. Similarly, there is a direct relationship from the Internalization of the Thin-Ideal Factor to the Eating Disturbance Factor and the relationship is also mediated by the Body Image Factor. Finally, the significant indirect effect between the Negative Affect Factor and the Eating Disturbance Factor indicates that this relationship is mediated by the Body Image Factor. Figure 9 depicts the respecified structural model with all existing pathways, standardized parameter estimates, and measurement errors.

Structural Model B. As mentioned earlier, structural equation analysis rarely involves testing a single model (Hoyle & Panter, 1995). In order to evaluate how well a given model fits a set of data, it is necessary to determine whether or not other models exist that fit the same set of data equally well (Lee & Hershberger, 1991). Thus, a second structural model was tested.

The following hypothesized relationships had been determined a priori and then tested in structural Model B. The Parental Attachment Factor was hypothesized to have a direct effect on the Appraisal/Coping Factor and an indirect effect on the Negative Affect Time 2 Factor and the Eating Disturbance Time 2 Factor. The History of Stress Factor was hypothesized to have a direct effect on the Appraisal/Coping Factor and an indirect effect on the Negative Affect Time 2 Factor and the Eating Disturbance Time 2 Factor. The Appraisal/Coping Factor was hypothesized to have a direct effect on the Negative Affect Time 2 Factor and an indirect effect on the Eating Disturbance Time 2 Factor. The Negative Affect Time 2 Factor was hypothesized to have a direct effect on the Eating Disturbances Time 2 Factor. The Internalization of Thin-Ideal Time 2 Factor was hypothesized to have both a direct effect on the Eating Disturbance Time 2 Factor and an indirect effect on the Eating Disturbances Time 2 Factor through a direct

effect on the Body Image Time 2 Factor. Finally, the Body Image Time 2 Factor was hypothesized to have a direct effect on the Eating Disturbance Time 2 Factor. The hypothesized structural Model B is displayed in Figure 10.

The initial attempt to fit the structural model with all of the above mentioned pathways was successful. No problems were encountered, and the testing of the structural paths supported the model. Although the chi-square statistic was significant, all other fit indices indicated a strong fit with the data (see Table 11).

Post hoc tests also were run on Model B to identify parameters in the model that can be modified to improve the fit of the model. The Wald test indicated that the addition of a pathway from the Negative Affect Time 2 Factor to the Body Image Time 2 Factor, $LM \chi^2(1, N = 236) = 16.275$ and a pathway from the Appraisal/Coping Factor to the Eating Disturbance Factor Time 2, $LM \chi^2(1, N = 236) = 15.937$ could significantly improve the fit of the model. The addition of the path from the Negative Affect Time 2 Factor to the Body Image Time 2 Factor resulted in a significant decreased in chi-square ($\Delta\chi^2 = 18.31, df = 1$) and improvement in model fit. The addition of the path from the Appraisal/Coping Factor to the Eating Disturbance Factor Time 2 resulted in the path from Negative Affect Time 2 to Eating Disturbance Time 2 being non-significant and model fit being poor. The removal of the path from Negative Affect Time 2 to Eating Disturbance Time 2 resulted in the previously present model (Model A). Thus, Model A appears to be the best representation of the data for the structural path among the selected factors at Time 1 and Time 2 (see Figure 9).

Evaluation of Selected Factors at Time 1 and Time 4

To evaluate simplistic versions of the multidimensional models of risk factors that contribute to the development of eating disorders as measured at Time 1 and Time 4, the following factors were selected for the model: Parental Attachment, History of Stress, Appraisal/Coping, Negative Affect Time 4, Body Image Time 4, Internalization of Thin-Ideal Time 4, and Eating Disturbance Time 4. A confirmatory analysis of the measurement model was conducted and 2 structural models were tested. These models are consistent with the models presented in Bradford's (2004) examination of these same factors, but the variables measured at Time 4 have been substituted for the corresponding variables at Time 1.

Measurement model. The confirmatory analysis of the measurement model for selected factors at Time 1 and Time 4 was begun with the Parental Attachment Factor, the History of Stress Factor, and the Appraisal/Coping Factor. Recall that the measurement model presented above also began with the addition of each of these constructs. Again, the measured variables that represent these latent constructs remain the same. The Parental Attachment Factor is representative of insecure parental attachment as measured by the Maternal Care scale, the Maternal Overprotectiveness scale, and the Paternal Overprotectiveness scales of the Parental Bonding Instrument (PBI). The History of Stress Factor is representative of greater experiences with stressful events as measured by the Developmental Challenge, Academic Alienation, and Time Pressure subscales of the Inventory of College Students' Recent Life Experiences (ICSRLE). The Appraisal/Coping Factor is representative of high levels of perceived stress and difficulty coping with stressors as measured by the Perceived Stress Scale (PSS) and the paired items measuring appraisal and coping of stressors related to academic demands.

Next, the Body Image Time 4 Factor was introduced to the measurement Model. Again, the Body Parts Satisfaction Scale (BPSS) Face Time 4 Factor failed to contribute common variance due to high associations with other measured variables. The internal consistency for this variable also was moderate (.64). The BPSS Body Time 4 Factor and the Multidimensional Body Self Relations Questionnaire-Appearance Evaluation (MBSRQ-AE) subscale Time 4 appropriately loaded on the Body Image Time 4 Factor and designated the construct as representative of positive body image.

The Internalization of Thin-Ideal Time 4 Factor was then added to the measurement model. Both the Beliefs About Attractiveness (BAAR) Physically Fit subscale Time 4 and BAAR Attractive and Thin subscale Time 4 loaded appropriately on the Internalization of Thin-Ideal Time 4 Factor, designating the construct as representative of greater internalization of the thin-ideal.

The Eating Disturbance Time 4 Factor also was added to the measurement model. Initial attempts to test the Bulimia Test – Revised (BULIT-R) and Eating Attitudes Test – 26 (EAT-26) on the Eating Disturbance Time 4 Factor resulted in a non-significant error variance for the BULIT-R Time 4. Utilizing Bollen’s (1989) procedure for estimating measurement error, the error variance for each was set to 1 minus the scale reliability times the variance for the measure. This resulted in an estimated value of 20.99 for the error variance for the BULIT-R Time 4. The BULIT-R and the EAT-26 loaded appropriately on the Eating Disturbance Time 4 Factor, designating it as representative of greater eating disordered symptomatology.

Finally, the Negative Affect Time 4 Factor was added to the measurement model. To maintain consistency in the models examining selected factors at Time 1 and Time 2 and those examining selected factors at Time 1 and Time 4, the PANAS-X Negative Affect Scale was not

tested on the Negative Affect Time 4 Factor. Only the Visual Analogue Mood Scale (VAMS) Sad or Depressed item, VAMS Happy item, and VAMS Stressed item loaded appropriately on the Negative Affect Time 4 Factor, designating the construct as representative of greater negative affect.

Using the EQS program for structural equation modeling and the Maximum Likelihood (ML) estimation procedure to estimate the parameters of the model, the confirmatory factor analysis (CFA) supported the resulting measurement model, and the overall fit of the data was good (see Table 11). The standardized parameter estimates (factor loadings and error variances) for the measurement Model Are displayed in Table 7, and the final representation of the measurement model is displayed in Figure 11.

Structural Model A. After validating the measurement model, the model was modified to include the structural model pathways. The pathways between the latent constructs were added to the measurement model, and all paths were tested simultaneously to identify the best fitting and most parsimonious causal model.

The following hypothesized relationships had been determined a priori and then tested in structural Model A. The Parental Attachment Factor was hypothesized to have a direct effect on the Appraisal/Coping Factor and an indirect effect on the Negative Affect Time 4 Factor, the Body Image Time 4 Factor, and the Eating Disturbance Time 4 Factor. The History of Stress Factor was hypothesized to have a direct effect on the Appraisal/Coping Factor and an indirect effect on the Negative Affect Time 4 Factor, the Body Image Time 4 Factor, and the Eating Disturbance Time 4 Factor. The Appraisal/Coping Factor was hypothesized to have a direct effect on the Negative Affect Time 4 Factor and an indirect effect on the Body Image Time 4

Factor, and the Eating Disturbance Time 4 Factor. The Negative Affect Time 4 Factor was hypothesized to have a direct effect on the Body Image Time 4 Factor and an indirect effect on the Eating Disturbances Time 4 Factor. The Internalization of Thin-Ideal Time 4 Factor was hypothesized to have both a direct effect on the Eating Disturbance Time 4 Factor and an indirect effect on the Eating Disturbances Time 4 Factor through a direct effect on the Body Image Time 4 Factor. Finally, the Body Image Time 4 Factor was hypothesized to have a direct effect on the Eating Disturbance Time 4 Factor. The hypothesized structural Model A is displayed in Figure 12.

The initial attempt to fit the structural model with all of the above mentioned pathways was successful. No problems were encountered, and the testing of the structural paths supported the model. Although the chi-square statistic was significant, all other fit indices indicated a strong fit with the data (see Table 11).

As previously discussed, post hoc tests can be run to identify parameters in the model that can be modified to improve the fit of the model. The Wald test indicated that the addition of a pathway from the Appraisal/Coping Factor to the Eating Disturbances Time 4 Factor, $LM \chi^2(1, N = 236) = 15.994$, could significantly improve the fit of the model. The additional pathway from the Appraisal/Coping Factor loaded significantly on the Eating Disturbances Time 4 Factor and the fit indices indicated that this model was a good fit with the data (see Table 11).

To assess the extent to which this respecified model is an improvement over the original structural model, the difference in χ^2 between the two models was examined. The chi-square difference test ($\Delta\chi^2 = 19.40, df = 1$) was significant ($p < .001$), indicating that the respecified model produced a statistically significant and substantial improvement in model fit over the original structural model. In addition, increases were seen in the NFI, NNFI, CFI, and RCFI and

decreases were observed in the SRMR and RMSEA, indicating improvement in model fit. Although the chi-square statistic for final model was non-significant ($p < .001$), the model demonstrated good fit with the data according to all other fit indices.

Both the direct effects and the indirect effects (amount of mediation) were examined (see Table 9). With the exception of the indirect effects from the History of Stress Factor on the Body Image Factor, all effects were significant. In this respecified structural model, the significant effects explained 19.68% of the variance for the Appraisal/Coping Factor, with the direct effects from the Parental Attachment Factor accounting for 17.06% of the variance and the History of Stress Factor accounting for 2.62% of the variance. 25.33% of the variance for the Negative Affect Factor was explained by the significant effects, with the direct effect from the Appraisal/Coping Factor accounting for 21.16% of the variance and the indirect effects from the Parental Attachment Factor and the History of Stress Factor accounting for 3.61% and 0.56% of the variance, respectively. The significant indirect effects from the Parental Attachment Factor and the History of Stress Factor indicate that the relationship from each of these factors to the Negative Affect Factor is mediated by the Appraisal/Coping Factor. The significant effects also explained 27.17% of the variance of the Body Image Factor, with the direct effects from the Internalization of the Thin-Ideal Factor and the Negative Affect Factor accounting for 19.62% and 6.05% of the variance, respectively. The significant indirect effects from the Parental Attachment Factor and the Appraisal Coping Factor accounted for 0.22% and 1.28% of the variance in the Body Image Factor, respectively. Thus, the relationship between the Parental Attachment Factor and the Body Image Factor is mediated by the Appraisal/Coping Factor and the Negative Affect Factor, and the relationship between the Appraisal/Coping Factor and the Body Image Factor is mediated by the Negative Affect Factor. 39.23% of the variance of the

Eating Disturbances Factor was explained by the significant direct and indirect effects. The significant direct effects from the Appraisal/Coping Factor, the Body Image Factor, and the Internalization of the Thin-Ideal Factor accounted for 8.29%, 19.89%, and 3.24% of the variance, respectively, and the significant indirect effects from the Parental Attachment Factor, the History of Stress Factor, the Appraisal/Coping Factor, the Negative Affect Factor, and the Internalization of the Thin-Ideal Factor accounted for 1.96%, 0.10%, 0.28%, 1.32%, and 4.24% of the variance, respectively. The significant indirect effects indicate that the relationship between the Parental Attachment Factor and the Eating Disturbance Factor and between the History of Stress Factor and the Eating Disturbance Factor is mediated by the Appraisal/Coping Factor, the Negative Affect Factor, and the Body Image Factor. The direct effect and indirect effect from the Appraisal/Coping Factor to the Eating Disturbance Factor indicate that there is a direct relationship between the two constructs and the relationship is also mediated by the Negative Affect Factor and the Body Image Factor. Similarly, there is a direct relationship from the Internalization of the Thin-Ideal Factor to the Eating Disturbance Factor and the relationship is also mediated by the Body Image Factor. Finally, the indirect effect between the Negative Affect Factor and the Eating Disturbance Factor indicates that this relationship is mediated by the Body Image Factor. Figure 13 depicts the respecified structural model with all existing pathways, standardized parameter estimates, and measurement errors.

Structural Model B. As mentioned earlier, structural equation analysis rarely involves testing a single model (Hoyle & Panter, 1995). In order to evaluate how well a given model fits a set of data, it is necessary to determine whether or not other models exist that fit the same set of data equally well (Lee & Hershberger, 1991). Thus, a second structural model was tested.

The following hypothesized relationships had been determined a priori and then tested in structural Model B. The Parental Attachment Factor was hypothesized to have a direct effect on the Appraisal/Coping Factor and an indirect effect on the Negative Affect Time 4 Factor and the Eating Disturbance Time 4 Factor. The History of Stress Factor was hypothesized to have a direct effect on the Appraisal/Coping Factor and an indirect effect on the Negative Affect Time 4 Factor and the Eating Disturbance Time 4 Factor. The Appraisal/Coping Factor was hypothesized to have a direct effect on the Negative Affect Time 4 Factor and an indirect effect on the Eating Disturbance Time 4 Factor. The Negative Affect Time 4 Factor was hypothesized to have a direct effect on the Eating Disturbances Time 4 Factor. The Internalization of Thin-Ideal Time 4 Factor was hypothesized to have both a direct effect on the Eating Disturbance Time 4 Factor and an indirect effect on the Eating Disturbances Time 4 Factor through a direct effect on the Body Image Time 4 Factor. Finally, the Body Image Time 4 Factor was hypothesized to have a direct effect on the Eating Disturbance Time 4 Factor. The hypothesized structural Model B is displayed in Figure 14.

The initial attempt to fit the structural model with all of the above mentioned pathways was successful. No problems were encountered, and the testing of the structural paths supported the model. Although the chi-square statistic was significant, all other fit indices indicated a strong fit with the data (see Table 11).

Post hoc tests were also run on Model B to identify parameters in the model that can be modified to improve the fit of the model. The Wald test indicated that the addition of a pathway from the Negative Affect Time 4 Factor to the Body Image Time 4 Factor, $LM \chi^2(1, N = 236) = 9.226$ could significantly improve the fit of the model. When the path from the Negative Affect Time 4 Factor to the Body Image Time 4 Factor was added to the model, the model failed to

converge due to the Negative Affect Time 4 Factor and the Body Image Time 4 Factor being linearly dependent. Thus, the originally specified model was retained.

Both the direct effects and the indirect effects (amount of mediation) were examined (see Table 10). With the exception of the indirect effects from the History of Stress Factor on both the Negative Affect Factor and the Eating Disturbance Factor, all effects were significant. In this respecified structural model, the significant effects explained 17.74% of the variance for the Appraisal/Coping Factor, with the direct effects from the Parental Attachment Factor accounting for 15.52% of the variance and the History of Stress Factor accounting for 2.22% of the variance. 21.84% of the variance for the Negative Affect Factor was explained by the significant effects, with the direct effect from the Appraisal/Coping Factor accounting for 18.92% of the variance and the indirect effect from the Parental Attachment Factor accounting for 2.92% of the variance. The significant indirect effect from the Parental Attachment Factor to the Negative Affect Factor indicates that this relationship is mediated by the Appraisal/Coping Factor. The significant direct effect from the Internalization of the Thin-Ideal Factor accounted for 23.33% of the variance of the Body Image Factor. Finally, 39.07% of the variance of the Eating Disturbances Factor was explained by the significant direct and indirect effects. The significant direct effects from the Negative Affect Factor, the Body Image Factor, and the Internalization of the Thin-Ideal Factor accounted for 3.28%, 26.11%, and 2.86% of the variance, respectively, and the significant indirect effects from the Parental Attachment Factor, the Appraisal/Coping Factor, and the Internalization of the Thin-Ideal Factor accounted for 0.10%, 0.62%, and 6.10% of the variance, respectively. The significant indirect effects indicate that the relationship between the Parental Attachment Factor and the Eating Disturbance Factor is mediated by the Appraisal/Coping Factor, and the Negative Affect Factor. The significant indirect effect from the Appraisal/Coping

Factor to the Eating Disturbance Factor indicates that the relationship is mediated by the Negative Affect Factor. The significant direct effect and indirect effect from the Internalization of the Thin-Ideal Factor to the Eating Disturbance Factor indicates that there is a direct relationship between the two constructs and the relationship is also mediated by the Body Image Factor. Figure 15 depicts the respecified structural model with all existing pathways, standardized parameter estimates, and measurement errors.

In summary, simplistic versions of Model A and Model B using select factors from Time 1 and Time 2 were tested. Model A with the addition of a direct path from the Appraisal/Coping Factor Time 1 to the Eating Disturbance Factor Time 2 fit the data well. Model B also fit the data well, but recommended modifications resulted in a model identical to Model A. Simplistic versions of Model A and Model B using select factors from Time 1 and Time 4 were also tested. Model A with the addition of a direct path from the Appraisal/Coping Factor Time 1 to the Eating Disturbance Factor Time 4 fit the data well. Model B also fit the data well.

Analysis of Individual Cross-Lagged and Simultaneous Effects

The above models allow for the evaluation of some, but not all, of the causal relationships among the constructs at all 3 points in time. Further, the sample size is only meeting liberally recommended minimum requirements. Without a sufficient sample size, statistical power is greatly reduced and results should be carefully interpreted. One purpose of this study was to further evaluate the relationships among factors that contribute to the development of eating disorders using prospective longitudinal data to be able to make statements about causality among the constructs. The models previously presented are integrative but causal statements can only be made about the relationships in which the measurement of the

variables for one construct preceded the measurement of the variables for another construct in time. To further evaluate the predictive relationships among factors that contribute to the development of eating disorders, paired constructs were selected to test both cross-lagged and simultaneous effects. The following selected pairs are consistent with theory and previous empirical support suggesting that the paired constructs are connected to one another in the development of eating disorder symptomatology. The selected constructs are also consistent with the larger models presented above.

Negative Affect and Body Image at Time 1 and Time 2

Consistent with previous research (Bradford, 2004; Kearney-Cooke & Striegel-Moore, 1994; Tripp, 2002; Tripp & Petrie, 2001), the models presented earlier in this study demonstrated relationships in which negative affect was hypothesized to influence body image dissatisfaction. However, models tested by Stice (1994) and Striegel-Moore et al. (1986) propose that body dissatisfaction leads to psychological distress, such as negative affect. It is also possible that these constructs are reciprocally related. Thus, it was important to further evaluate and clarify the relationships between these constructs using prospective data.

Measurement model. Examination of the relationship between Time 1 and Time 2 for the constructs of negative affect and the constructs of body image was begun by conducting a confirmatory test of the measurement model. Consistent with the previously presented measurement models for these factors, it was hypothesized a priori that the individual items on the Visual Analog Mood Scale (VAMS) would represent the Negative Affect Factors, and the Body Parts Satisfaction Scale (BPSS) Body Factor, BPSS Face Factor, and the Multidimensional

Body Self Relations Questionnaire-Appearance Evaluation subscale (MBSRQ-AE) would represent the Body Image Factors. For the Negative Affect Factors, only the VAMS Sad or Depressed item, the VAMS Happy item, and the VAMS Stressed item for each time point loaded appropriately on the corresponding Negative Affect Factors. The remaining VAMS items were removed from the measurement model. For the Body Image Factors, the BPSS Face Factor for each time point failed to load on the Body Image Factor. These variables were also removed from the measurement model. At this point, error variances for the MBSRQ-AE Time 1 and the VAMS Sad or Depressed Time 2 were constrained at the lower bound. Following Bollen's (1989) recommendations, the error variances for these variables were set to 0.0757 and 0.0674, respectively.

Using the EQS program for structural equation modeling and the Maximum Likelihood (ML) estimation procedure to estimate the parameters of the model, the confirmatory factor analysis (CFA) supported the resulting measurement model, and the overall fit of the data was good (see Table 12). In the measurement model, the VAMS Sad or Depressed items and the VAMS Stressed items loaded positively on the corresponding constructs, and the VAMS Happy item loaded negatively on the corresponding constructs, designating the Negative Affect Factors as representative of negative affect. The measured variables for the Body Image Factors loaded positively on the corresponding constructs, designating the overall constructs as representative of positive body image. All factors were allowed to intercorrelate and the error variance for each measured variable at Time 1 was correlated with the error variance for the same measured variable at Time 2 (i.e., error variance for MBSRQ-AE Time 1 was correlated with error variance for MBSRQ-AE Time 2). The measurement model with standardized parameter estimates (factor loadings and error variances) is displayed in Figure 16.

Examination of the relationships among the factors in the measurement model (see Figure 16) revealed positive and significant correlations between the Negative Affect Factor at Time 1 and at Time 2 ($r = .56, p < .05$) and between the Body Image Factor at Time 1 and at Time 2 ($r = .90, p < .05$), suggesting that the rank ordering for these constructs was relatively temporally stable, although body image was highly stable over time, whereas negative affect was only moderately stable over time. The measurement model also showed negative and significant correlations between the within-time constructs ($r = -.35, p < .05$ for Time 1; $r = -.30, p < .05$ for Time 2) as well as negative and significant cross-time correlations between the Negative Affect Time 1 Factor and the Body Image Time 2 Factor ($r = -.25, p < .05$) and between the Body Image Time 1 Factor and the Negative Affect Time 2 Factor ($r = -.32, p < .05$). Temporal precedence is suggested in the cross-time correlations, because the Time 1 measures precede the Time 2 measures. Additionally, both the within-time correlations and the cross-time correlations between the constructs are significant, and the cross-time correlations are generally slightly weaker than the within-time correlations, suggesting that negative affect also may be reciprocally related to body image. To properly evaluate these relationships among the constructs, it is recommended that a predictor variable should account for variance in a criterion variable while controlling for the temporal stability of the criterion variable (Newcomb, 1994). Thus, a baseline stability model, 3 alternative models representing cross-lagged effects, and 3 models representing simultaneous effects were constructed to further examine the structural relationships between negative affect and body image at Time 1 and Time 2. These models are described below and presented in Figure 17.

Structural models. To compare competing models, a baseline stability model was created. Only the paths from Time 1 latent factors to the corresponding Time 2 latent factors were included in this baseline model. In addition, latent factors at Time 1 were correlated and Time 2 disturbances for the latent factors were correlated. The measurement errors for measured variables at Time 1 were correlated with the corresponding measurement error at Time 2 to prevent the stability paths from being inflated as a result of shared measurement error (Bollen, 1989). The baseline model fit the data well (see Table 12).

The empirically supported path from the Negative Affect Time 1 Factor to the Body Image Time 2 Factor was specified in Model 1 (see Figure 17). This model showed an acceptable fit to the data, but the chi-square change was non-significant (see Table 12). Thus, Model 1 did not represent an improvement over the baseline model. In addition, the specified path from the Negative Affect Time 1 Factor to the Body Image Time 2 Factor was not significant, indicating a lack of support for the expected relationship in which negative affect would predict body disparagement.

Testing the competing hypothesis, Model 2 specified a path from the Body Image Time 1 Factor to the Negative Affect Time 2 Factor (see Figure 17). Again, this model showed an acceptable fit to the data, but the non-significant chi-square change indicated that the baseline model still better represented the data (see Table 12). Further, the specified path from the Body Image Time 1 Factor to the Negative Affect Time 2 Factor was not significant, demonstrating a lack of support for the competing model in which body disparagement would predict negative affect (see Figure 17).

Model 3 tested an alternate hypothesis in which negative affect and body image would be reciprocally related between Time 1 and Time 2. Paths were included from the Negative Affect

Time 1 Factor to the Body Image Time 2 Factor and from the Body Image Time 1 Factor to the Negative Affect Time 2 Factor (see Figure 17). Model 3 also showed an acceptable fit to the data, but the non-significant chi-square change indicated that the baseline model still better represented the data (see Table 12). Consistent with Model 1 and Model 2, the specified cross-time paths were not significant (see Figure 17). Thus, these results did not provide evidence for the reciprocal relationship.

The time lag between negative affect and body image as measured at Time 1 and Time 2 was approximately 6 months. However, it is possible that the causal time lag is less than 6 months, and the relationship between negative affect and body image may be more immediate. Compared to the correlations between Body Image Time 1 and Body Image Time 2 ($r = .90$), the correlations between Negative Affect Time 1 and Negative Affect Time 2 ($r = .56$) indicate variation in mood over the 6-month time period. A shorter causal time lag or simultaneous analyses reduces the fluctuations in mood. Models 4, 5, and 6 examined the simultaneous effects between the Negative Affect Factor and the Body Image Factor at Time 2. The correlation between the disturbances for the Time 2 factors was removed from these models. Thus, Models 4, 5, and 6 represent reparameterizations of the baseline model and are not nested within the baseline model.

Model 4 tested the empirically supported path from the Negative Affect Time 2 Factor to the Body Image Time 2 Factor (see Figure 17). This model showed an acceptable fit with the data (see Table 12). However, the path from the Negative Affect Time 2 Factor to the Body Image Time 2 Factor was not significant (see Figure 17). Thus, these results did not support the expected relationship in which negative affect would predict body disparagement.

Model 5 tested the competing simultaneous hypothesis in which body disparagement would predict negative affect. This model specified a path from the Body Image Time 2 Factor to the Negative Affect Time 2 Factor (see Figure 17). This model showed an acceptable fit with the data (see Table 12), and the path from the Body Image Time 2 Factor to the Negative Affect Time 2 Factor was significant (see Figure 17). These results support the competing model, indicating that body disparagement leads to negative affect. However, it should be noted that this effect occurs within the same time frame. Thus, it is not a predictive relationship.

Model 6 tested the contemporaneous reciprocal relationship between negative affect and body disparagement. This model specified a path from the Negative Affect Time 2 Factor to the Body Image Time 2 Factor and from the Body Image Time 2 Factor to the Negative Affect Time 2 Factor (see Figure 17). The model demonstrated acceptable fit with the data (see Table 12), but only the path from the Body Image Time 2 Factor to the Negative Affect Time 2 Factor was significant (see Figure 17). Model 6 also was run with the effects constrained to be equal, but this resulted in a significant decrement in model fit ($\Delta\chi^2 = 107.74, df=1, p < .001$). These results indicate that the path from the Body Image Time 2 Factor to the Negative Affect Time 2 Factor is significantly larger than the path from the Negative Affect Time 2 Factor to the Body Image Time 2 Factor. These results are consistent with the results from Model 4 and Model 5, and they indicate that body disparagement leads to negative affect within a relatively short time frame. Model 6 does represent a significant improvement in fit over Model 4 ($\Delta\chi^2 = 5.57, df=1, p < .05$), but Model 6 does not represent a significant improvement over Model 5 ($\Delta\chi^2 = 0.57, df=1, p > .05$). Thus, Model 5 is the best representation of the simultaneous effects between the Negative Affect Time 2 Factor and the Body Image Time 2 Factor.

Negative Affect and Body Image at Time 2 and Time 4

Measurement model. Examination of the relationship between Time 2 and Time 4 for the constructs of negative affect and the constructs of body image also was begun by conducting a confirmatory test of the measurement model. Again, it was hypothesized a priori that the individual items VAMS would represent the Negative Affect Factors and the BPSS Body Factor, BPSS Face Factor, and the MBSRQ-AE would represent the Body Image Factors. For the Negative Affect Factors, only the VAMS Sad or Depressed item, the VAMS Happy item, and the VAMS Stressed item for each time loaded appropriately on the corresponding Negative Affect Factor. For the Body Image Factors, the BPSS Face Factor for each time failed to load on the Body Image Factor. Again, the remaining VAMS items and the BPSS Body Factors were removed from the measurement model.

Using the EQS program for structural equation modeling and the Maximum Likelihood (ML) estimation procedure to estimate the parameters of the model, the confirmatory factor analysis (CFA) supported the resulting measurement model, and the overall fit of the data was good (see Table 12). In the measurement model, the VAMS Sad or Depressed items and the VAMS Stressed items loaded positively on the corresponding constructs, and the VAMS Happy item loaded negatively on the corresponding constructs, designating the Negative Affect Factors as representative of negative affect. The measured variables for the Body Image Factors loaded positively on the corresponding constructs, designating the overall constructs as representative of positive body image. Again, all factors were allowed to intercorrelate and the error variance for each measured variable at Time 2 was correlated with the error variance for the same measured variable at Time 4. The measurement model with standardized parameter estimates (factor loadings and error variances) is displayed in Figure 18.

Examination of the relationships among the factors in the measurement model (see Figure 18) revealed positive and significant correlations between the Negative Affect Factor at Time 2 and at Time 4 ($r = .57, p < .05$) and between the Body Image Factor at Time 2 and at Time 4 ($r = .84, p < .05$), suggesting that the rank ordering for these constructs was relatively temporally stable, although body image was highly stable over time, whereas negative affect was only moderately stable over time. The measurement model also showed positive and significant correlations between the within-time constructs ($r = -.31, p < .05$ for Time 2; $r = -.22, p < .05$ for Time 4) as well as positive and significant cross-time correlations between the Negative Affect Time 2 Factor and the Body Image Time 4 Factor ($r = -.30, p < .16$) and between the Body Image Time 2 Factor and the Negative Affect Time 4 Factor ($r = .39, p < .05$). The Time 2 measures precede the Time 4 measures, suggesting temporal precedence in the cross-time correlations. Further, both the within-time correlations and the cross-time correlations between the constructs are significant, and the cross-time correlations are either similar or generally slightly weaker than the within-time correlations, suggesting that negative affect also may be reciprocally related to body image. A baseline stability model, 3 alternative models representing cross-lagged effects, and 3 models representing simultaneous effects were also constructed to further examine the structural relationships between negative affect and body image at Time 2 and Time 4. These models are described below and presented in Figure 19.

Structural models. Only the paths from Time 2 latent factors to the corresponding Time 4 latent factors were included in this baseline model. In addition, latent factors at Time 2 were correlated, Time 4 disturbances for the latent factors were correlated, and measurement errors for

measured variables at Time 2 were correlated with the corresponding measurement error at Time 4. The baseline model fit the data well (see Table 12).

The empirically supported path from the Negative Affect Time 2 Factor to the Body Image Time 4 Factor was specified in Model 1 (see Figure 19). This model showed an acceptable fit to the data, but the chi-square change was non-significant (see Table 12). Thus, Model 1 did not represent an improvement over the baseline model. In addition, the specified path from the Negative Affect Time 2 Factor to the Body Image Time 4 Factor was not significant, indicating a lack of support for the expected relationship in which negative affect would predict body disparagement.

Testing the competing hypothesis, Model 2 specified a path from the Body Image Time 2 Factor to the Negative Affect Time 4 Factor (see Figure 19). Again, this model showed an acceptable fit to the data, but the non-significant chi-square change indicated that the baseline model still better represented the data (see Table 12). Further, the specified path from the Body Image Time 2 Factor to the Negative Affect Time 4 Factor was not significant, demonstrating a lack of support for the competing model in which body disparagement would predict negative affect (see Figure 19).

Model 3 tested an alternate hypothesis in which negative affect and body image would be reciprocally related between Time 2 and Time 4. Paths were included from the Negative Affect Time 2 Factor to the Body Image Time 4 Factor and from the Body Image Time 2 Factor to the Negative Affect Time 4 Factor (see Figure 19). Model 3 also showed an acceptable fit to the data, but the non-significant chi-square change indicated that the baseline model still better represented the data (see Table 12). Consistent with Model 1 and Model 2, the specified cross-

time paths were not significant (see Figure 19). Thus, these results did not provide evidence for the reciprocal relationship.

The time lag between negative affect and body image as measured at Time 2 and Time 4 was approximately 12 months. However, it is possible that the causal time lag is less than 12 months, and the relationship between negative affect and body image may be more immediate. Compared to the correlations between Body Image Time 2 and Body Image Time 4 ($r = .84$), the correlations between Negative Affect Time 2 and Negative Affect Time 4 ($r = .57$) indicate variation in mood over the 12-month time period. A shorter causal time lag or simultaneous analyses may reduce the fluctuations in mood. Models 4, 5, and 6 examined the simultaneous effects between the Negative Affect Factor and the Body Image Factor at Time 4. The correlation between the disturbances for the Time 4 factors was removed from these models. Thus, Models 4, 5, and 6 represent reparameterizations of the baseline model and are not nested within the baseline model.

Model 4 tested the empirically supported path from the Negative Affect Time 4 Factor to the Body Image Time 4 Factor (see Figure 19). This model showed an acceptable fit with the data (see Table 12). However, the path from the Negative Affect Time 4 Factor to the Body Image Time 4 Factor was not significant (see Figure 19). Thus, these results did not support the expected relationship in which negative affect would predict body disparagement.

Model 5 tested the competing simultaneous hypothesis in which body disparagement would predict negative affect. This model specified a path from the Body Image Time 4 Factor to the Negative Affect Time 4 Factor (see Figure 19). This model showed an acceptable fit with the data (see Table 12), but the path from the Body Image Time 4 Factor to the Negative Affect

Time 4 Factor was not significant (see Figure 19). Thus, no support was provided for the competing model.

Model 6 tested the contemporaneous reciprocal relationship between negative affect and body disparagement. This model specified a path from the Negative Affect Time 4 Factor to the Body Image Time 4 Factor and from the Body Image Time 4 Factor to the Negative Affect Time 4 Factor (see Figure 19). The model demonstrated acceptable fit with the data (see Table 12), but neither of the paths between the Body Image Time 4 Factor and the Negative Affect Time 4 Factor were significant (see Figure 19). These results do not support the hypothesis that negative affect predicts body disparagement and that body disparagement predicts negative affect in return within relatively short time lag.

Negative affect and Body image at Time 1 and Time 4

Measurement model. Examination of the relationship between Time 1 and Time 4 for the constructs of negative affect and the constructs of body image was begun by conducting a confirmatory test of the measurement model. Again, it was hypothesized a priori that the individual items VAMS would represent the Negative Affect Factors and the BPSS Body Factor, BPSS Face Factor, and the MBSRQ-AE would represent the Body Image Factors. For the Negative Affect Factors, only the VAMS Sad or Depressed item, the VAMS Happy item, and the VAMS Stressed item for each time loaded appropriately on the corresponding Negative Affect Factor. For the Body Image Factors, the BPSS Face Factor for each time failed to load on the Body Image Factor. Again, the remaining VAMS items and the BPSS Body Factors were removed from the measurement model.

Using the EQS program for structural equation modeling and the Maximum Likelihood (ML) estimation procedure to estimate the parameters of the model, the confirmatory factor analysis (CFA) supported the resulting measurement model, and the overall fit of the data was good (see Table 12). In the measurement model, the VAMS Sad or Depressed items and the VAMS Stressed items loaded positively on the corresponding constructs, and the VAMS Happy item loaded negatively on the corresponding constructs, designating the Negative Affect Factors as representative of negative affect. The measured variables for the Body Image Factors loaded positively on the corresponding constructs, designating the overall constructs as representative of positive body image. Again, all factors were allowed to intercorrelate and the error variance for each measured variable at Time 1 was correlated with the error variance for the same measured variable at Time 4. The measurement model with standardized parameter estimates (factor loadings and error variances) is displayed in Figure 20.

Examination of the relationships among the factors in the measurement model (see Figure 20) revealed positive and significant correlations between the Internalization of Thin-ideal Factor at Time 1 and at Time 4 ($r = .56, p < .05$) and between the Negative Affect Factor at Time 1 and at Time 4 ($r = .90, p < .05$), suggesting that the rank ordering for body image was highly stable over time, whereas negative affect was only moderately stable over time. The measurement model also showed positive and significant correlations between the within-time constructs ($r = -.35, p < .05$ for Time 1; $r = -.30, p < .05$ for Time 4) as well as positive and significant cross-time correlations between the Negative Affect Time 1 Factor and the Body Image Time 4 Factor ($r = -.25, p < .05$) and between the Body Image Time 1 Factor and the Negative Affect Time 4 Factor ($r = -.32, p < .05$). Time 1 measures preceded Time 4 measures, suggesting temporal precedence in the cross-time correlations. Further both the within-time correlations and the cross-time

correlations between the constructs are significant, and the cross-time correlations are generally slightly weaker than the within-time correlations, suggesting that negative affect may also be reciprocally related to body image. A baseline stability model, 3 alternative models representing cross-lagged effects, and 3 models representing simultaneous effects were also constructed to further examine the structural relationships between negative affect and body image at Time 1 and Time 4. These models are described below and presented in Figure 21.

Structural models. Only the paths from Time 1 latent factors to the corresponding Time 4 latent factors were included in this baseline model. In addition, latent factors at Time 1 were correlated, Time 4 disturbances for the latent factors were correlated, and measurement errors for measured variables at Time 1 were correlated with the corresponding measurement error at Time 4. The baseline model fit the data well (see Table 12).

The empirically supported path from the Negative Affect Time 1 Factor to the Body Image Time 4 Factor was specified in Model 1 (see Figure 21). This model showed an acceptable fit to the data, but the chi-square change was non-significant (see Table 12). Thus, Model 1 did not represent an improvement over the baseline model. In addition, the specified path from the Negative Affect Time 1 Factor to the Body Image Time 4 Factor was not significant, indicating a lack of support for the expected relationship in which negative affect would predict body disparagement.

Testing the competing hypothesis, Model 2 specified a path from the Body Image Time 1 Factor to the Negative Affect Time 4 Factor (see Figure 21). Again, this model showed an acceptable fit to the data, but the non-significant chi-square change indicated that the baseline model still better represented the data (see Table 12). Further, the specified path from the Body

Image Time 1 Factor to the Negative Affect Time 4 Factor was not significant, demonstrating a lack of support for the competing model in which body disparagement would predict negative affect (see Figure 21).

Model 3 tested an alternate hypothesis in which negative affect and body image would be reciprocally related between Time 1 and Time 4. Paths were included from the Negative Affect Time 1 Factor to the Body Image Time 4 Factor and from the Body Image Time 1 Factor to the Negative Affect Time 4 Factor (see Figure 21). Model 3 also showed an acceptable fit to the data, but the non-significant chi-square change indicated that the baseline model still better represented the data (see Table 12). Consistent with Model 1 and Model 2, the specified cross-time paths were not significant (see Figure 21). Thus, these results did not provide evidence for the reciprocal relationship.

The time lag between negative affect and body image as measured at Time 1 and Time 4 was approximately 18 months. However, it is possible that the causal time lag is less than 18 months, and the relationship between negative affect and body image may be more immediate. Compared to the correlations between Body Image Time 1 and Body Image Time 4 ($r = .90$), the correlations between Negative Affect Time 1 and Negative Affect Time 4 ($r = .56$) indicate variation in mood over the 18-month time period. A shorter causal time lag or simultaneous analyses may reduce the fluctuations in mood. Models 4, 5, and 6 examined the simultaneous effects between the Negative Affect Factor and the Body Image Factor at Time 4. The correlation between the disturbances for the Time 4 factors was removed from these models. Thus, Models 4, 5, and 6 represent reparameterizations of the baseline model and are not nested within the baseline model.

Model 4 tested the empirically supported path from the Negative Affect Time 4 Factor to the Body Image Time 4 Factor (see Figure 21). This model showed an acceptable fit with the data (see Table 12). However, the path from the Negative Affect Time 4 Factor to the Body Image Time 4 Factor was not significant (see Figure 21). Thus, these results did not support the expected relationship in which negative affect would predict body disparagement.

Model 5 tested the competing simultaneous hypothesis in which body disparagement would predict negative affect. This model specified a path from the Body Image Time 4 Factor to the Negative Affect Time 4 Factor (see Figure 21). This model showed an acceptable fit with the data (see Table 12), but the path from the Body Image Time 4 Factor to the Negative Affect Time 4 Factor was not significant (see Figure 21). Thus, no support was provided for the competing model.

Model 6 tested the contemporaneous reciprocal relationship between negative affect and body disparagement. This model specified a path from the Negative Affect Time 4 Factor to the Body Image Time 4 Factor and from the Body Image Time 4 Factor to the Negative Affect Time 4 Factor (see Figure 21). The model demonstrated acceptable fit with the data (see Table 12), but neither of the paths between the Body Image Time 4 Factor and the Negative Affect Time 4 Factor were significant (see Figure 21). These results do not support the hypothesis that negative affect predicts body disparagement and that body disparagement predicts negative affect in return within relatively short time lag.

Internalization of Thin-Ideal and Body Image at Time 1 and Time 2

Researchers have suggested that the pervasive presence of messages from media, peers and family promote unrealistic ideals for women and body types that require drastic means to

obtain and maintain, resulting in body dissatisfaction (Smolak & Levine, 1996) The models previously presented in this study supported a direct pathway from the Internalization of the Thin-Ideal Factor to the Body Image Factor, confirming prior research in which women who acknowledged awareness and internalization of societal ideals possessed more negative body image (Bradford, 2004; Tripp, 2002). However, previous research examining the direct relationship between these constructs used data collected during the same time period. Thus, two alternative hypothesis the relationship between internalization of the thin-ideal and body image are also possible. It is possible that negative body image leads to increased internalization of the thin-ideal or that internalization of the thin-ideal and body image are reciprocally related. To further evaluate the relationships between these constructs, prospective data was used to test cross-lagged and simultaneous effects between internalization of the thin-ideal and body image.

Measurement model. Examination of the relationship between Time 1 and Time 2 for the constructs of internalization of thin-ideal and the constructs of body image was begun by conducting a confirmatory test of the measurement model. Consistent with the previously presented measurement models for these factors, it was hypothesized a priori that the Beliefs About Attractiveness (BAAR) Physically Fit subscale and Attractive and Thin subscale would represent the Internalization of Thin-Ideal Factors, and the Body Parts Satisfaction Scale (BPSS) Body Factor, BPSS Face Factor, and the Multidimensional Body Self Relations Questionnaire-Appearance Evaluation subscale (MBSRQ-AE) would represent the Body Image Factors. The BPSS Face Factor for each time failed to load on the Body Image Factor, and these variables were removed from the measurement model.

Using the EQS program for structural equation modeling and the Maximum Likelihood (ML) estimation procedure to estimate the parameters of the model, the confirmatory factor analysis (CFA) supported the resulting measurement model, and the overall fit of the data was good (see Table 13). All measured variables loaded positively on the corresponding constructs. Thus, the Internalization of Thin-Ideal Factors represented greater internalization of the thin-ideal, and the Body Image Factors represented positive body image. All factors were allowed to intercorrelate and the error variance for each measured variable at Time 1 was correlated with the error variance for the same measured variable at Time 2. The measurement model with standardized parameter estimates (factor loadings and error variances) is displayed in Figure 22.

Examination of the relationships among the factors in the measurement model (see Figure 22) revealed positive and significant correlations between the Internalization of Thin-ideal Factor at Time 1 and at Time 2 ($r = .73, p < .05$) and between the Body Image Factor at Time 1 and at Time 2 ($r = .82, p < .05$), suggesting that the rank ordering for these constructs was relatively temporally stable. The measurement model showed positive and significant correlations between the within-time constructs ($r = .43, p < .05$ for Time 1; $r = .44, p < .05$ for Time 2) as well as positive and significant cross-time correlations between the Internalization of Thin-Ideal Time 1 Factor and the Body Image Time 2 Factor ($r = .37, p < .05$) and between the Body Image Time 1 Factor and the Internalization of Thin-Ideal Time 2 Factor ($r = .36, p < .05$). Because the Time 1 measures preceded the Time 2 measures, the cross-time correlations suggest temporal precedence. Additionally, both the within-time correlations and the cross-time correlations between the constructs are significant, and the cross-time correlations are generally slightly weaker than the within-time correlations, suggesting that internalization of thin-ideal may also be reciprocally related to body image. To properly evaluate the relationships among the constructs,

it is recommended that a predictor variable should account for variance in a criterion variable while controlling for the temporal stability of the criterion variable (Newcomb, 1994). Thus, a baseline stability model, 3 alternative models representing cross-lagged effects, and 3 models representing simultaneous effects were constructed to further examine the structural relationships between internalization of thin-ideal and body image at Time 1 and Time 2. These models are described below and presented in Figure 23.

Structural models. To compare competing models, a baseline stability model was created. Only the paths from Time 1 latent factors to the corresponding Time 2 latent factors were included in this baseline model. In addition, latent factors at Time 1 were correlated and Time 2 disturbances for the latent factors were correlated. The measurement errors for measured variables at Time 1 were correlated with the corresponding measurement error at Time 2 to prevent the stability paths from being inflated as a result of shared measurement error (Bollen, 1989). The baseline model fit the data well (see Table 13).

The empirically supported path from the Internalization of Thin-Ideal Time 1 Factor to the Body Image Time 2 Factor was specified in Model 1 (see Figure 13). This model showed an acceptable fit to the data, and the chi-square change was significant (see Table 13). Model 1 represents an improvement over the baseline model. Additionally, the specified path from the Internalization of Thin-Ideal Time 1 Factor to the Body Image Time 2 Factor was significant, indicating that internalization of the thin-ideal predicts body disparagement over time (see Figure 23).

Testing the competing hypothesis, Model 2 specified a path from the Body Image Time 1 Factor to the Internalization of Thin-Ideal Time 2 Factor (see Figure 23). Again, this model

showed an acceptable fit to the data, and the significant chi-square change indicated that Model 2 represented an improvement over the baseline model (see Table 13). Further, the specified path from the Body Image Time 1 Factor to the Internalization of Thin-Ideal Time 2 Factor was significant, demonstrating support for the competing model in which body disparagement predicts greater internalization of the thin-ideal over time (see Figure 23).

Model 3 tested an alternate hypothesis in which internalization of the thin-ideal and body image are reciprocally related between Time 1 and Time 2. Paths were included from the Internalization of Thin-Ideal Time 1 Factor to the Body Image Time 2 Factor and from the Body Image Time 1 Factor to the Internalization of Thin-Ideal Time 2 Factor (see Figure 23). Model 3 also showed an acceptable fit to the data, and the significant chi-square change indicated a better representation of the data compared to the baseline model (see Table 13). Further, Model 3 represented a significant improvement over Model 1 ($\Delta\chi^2 = 4.68, df=1, p < .05$) and Model 2 ($\Delta\chi^2 = 5.79, df=1, p < .05$). Consistent with Model 1 and Model 2, the specified cross-time paths also were significant (see Figure 23). Thus, the hypothesis that internalization of the thin-ideal and body image are reciprocally related over time was supported. The results indicate that internalization of the thin-ideal predicts body disparagement over time and body disparagement predicts greater internalization of the thin-ideal over time.

The time lag between body image and eating disturbance as measured at Time 1 and Time 2 was approximately 6 months. However, it is possible that the causal time lag is less than 6 months, and the relationship between internalization of the thin-ideal and body image may be more immediate. Models 4, 5, and 6 examined the simultaneous effects between the Internalization of Thin-Ideal Factor and the Body Image Factor at Time 2. The correlation between the disturbances for the Time 2 factors was removed from these models. Thus, Models

4, 5, and 6 represent reparameterizations of the baseline model and are not nested within the baseline model.

Model 4 tested the empirically supported path from the Internalization of Thin-Ideal Time 2 Factor to the Body Image Time 2 Factor (see Figure 23). This model showed an acceptable fit with the data (see Table 13). Further, the path from the Internalization of Thin-Ideal Time 2 Factor to the Body Image Time 2 Factor was significant (see Figure 23). These results indicate that internalization of the thin-ideal predicts body disparagement. However, it should be noted that this effect has a relatively short time lag.

Model 5 tested the competing simultaneous hypothesis in which body disparagement predicts internalization of the thin-ideal. This model specified a path from the Body Image Time 2 Factor to the Internalization of Thin-Ideal Time 2 Factor (see Figure 23). This model showed an acceptable fit with the data (see Table 13), and the path from the Body Image Time 2 Factor to the Internalization of Thin-Ideal Time 2 Factor was significant (see Figure 23). Thus, support also was provided for the competing model, indicating that body disparagement leads to internalization of the thin-ideal within a relatively short time lag. However, Hoyle and Panter (1995) noted that simply switching the direction of the path between two constructs may not result in any change in the parameter estimate or the overall fit of the model. Thus, it is difficult to establish directionality.

Model 6 tested the contemporaneous reciprocal relationship between internalization of the thin-ideal and body image. This model specified a path from the Internalization of Thin-Ideal Time 2 Factor to the Body Image Time 2 Factor and from the Body Image Time 2 Factor to the Internalization of Thin-Ideal Time 2 Factor (see Figure 23). The model demonstrated acceptable fit with the data (see Table 13), but only the path from the Internalization of Thin-Ideal Time 2

Factor to the Body Image Time 2 Factor was significant (see Figure 23). Model 6 also was run with the effects constrained to be equal, but this resulted in a significant decrement in model fit ($\Delta\chi^2 = 119.70$, $df=1$, $p < .001$). EQS indicates that the path from the Internalization of Thin-Ideal Time 2 Factor to the Body Image Time 2 Factor is significantly larger than the path from the Body Image Time 2 Factor to the Internalization of Thin-Ideal Time 2 Factor. These results are consistent with the results from Model 4, and they indicate that internalization of the thin-ideal leads to body disparagement within a relatively short time frame. Further, Model 6 represents a significant improvement in fit over Model 5 ($\Delta\chi^2 = 5.73$, $df=1$, $p < .05$). However, Model 6 does not represent a significant improvement over Model 4 ($\Delta\chi^2 = 3.83$, $df=1$, $p > .05$). Thus, Model 4 is the best representation of the simultaneous effects between the Internalization of Thin-Ideal Time 2 Factor and the Body Image Time 2 Factor.

Internalization of Thin-Ideal and Body Image at Time 2 and Time 4

Measurement model. Examination of the relationship between Time 2 and Time 4 for the constructs of internalization of thin-ideal and the constructs of body image was begun by conducting a confirmatory test of the measurement model. Again, it was hypothesized a priori that the BAAR Physically Fit subscale and BAAR Attractive and Thin subscale would represent the Internalization of Thin-Ideal Factors, and the BPSS Body Factor, BPSS Face Factor, and the MBSRQ-AE would represent the Body Image Factors. The BPSS Face Factor for each time failed to load on the Body Image Factor, and these variables were removed from the measurement model.

Using the EQS program for structural equation modeling and the Maximum Likelihood (ML) estimation procedure to estimate the parameters of the model, the confirmatory factor

analysis (CFA) supported the resulting measurement model, and the overall fit of the data was good (see Table 13). All measured variables loaded positively on the corresponding constructs. Thus, the Internalization of Thin-Ideal Factors represented greater internalization of the thin-ideal, and the Body Image Factors represented positive body image. Again, all factors were allowed to intercorrelate and the error variance for each measured variable at Time 2 was correlated with the error variance for the same measured variable at Time 4. The measurement model with standardized parameter estimates (factor loadings and error variances) is displayed in Figure 24.

Examination of the relationships among the factors in the measurement model (see Figure 24) revealed positive and significant correlations between the Internalization of Thin-ideal Factor at Time 2 and at Time 4 ($r = .84, p < .05$) and between the Body Image Factor at Time 2 and at Time 4 ($r = .86, p < .05$), suggesting that the rank ordering for these constructs was relatively temporally stable. The measurement model also showed negative and significant correlations between the within-time constructs ($r = -.39, p < .05$ for Time 2; $r = -.49, p < .05$ for Time 4) as well as negative and significant cross-time correlations between the Internalization of Thin-Ideal Time 2 Factor and the Body Image Time 4 Factor ($r = -.38, p < .05$) and between the Body Image Time 2 Factor and the Internalization of Thin-Ideal Time 4 Factor ($r = -.44, p < .05$). Again, the Time 2 measures preceded the Time 4 measures, suggesting temporal precedence in the cross-time correlation. Further, both the within-time correlations and the cross-time correlations between the constructs are significant, and the cross-time correlations are generally slightly weaker than the within-time correlations, suggesting that internalization of thin-ideal also may be reciprocally related to body image. A baseline stability model, 3 alternative models representing cross-lagged effects, and 3 models representing simultaneous effects were also

constructed to further examine the structural relationships between internalization of thin-ideal and body image at Time 2 and Time 4. These models are described below and presented in Figure 25.

Structural models. Only the paths from Time 2 latent factors to the corresponding Time 4 latent factors were included in this baseline model. In addition, latent factors at Time 2 were correlated, Time 4 disturbances for the latent factors were correlated, and measurement errors for measured variables at Time 2 were correlated with the corresponding measurement error at Time 4. The baseline model fit the data well (see Table 13).

The empirically supported path from the Internalization of Thin-Ideal Time 2 Factor to the Body Image Time 4 Factor was specified in Model 1 (see Figure 25). This model showed an acceptable fit to the data, and the chi-square change was significant (see Table 13). Model 1 represents an improvement over the baseline model. Additionally, the specified path from the Internalization of Thin-Ideal Time 2 Factor to the Body Image Time 4 Factor was significant, indicating that internalization of the thin-ideal predicts body disparagement over time (see Figure 25).

Testing the competing hypothesis, Model 2 specified a path from the Body Image Time 2 Factor to the Internalization of Thin-Ideal Time 4 Factor (see Figure 25). Again, this model showed an acceptable fit to the data, but the non-significant chi-square change indicated that the baseline model still better represented the data (see Table 13). Further, the specified path from the Body Image Time 2 Factor to the Internalization of Thin-Ideal Time 4 Factor was not significant, demonstrating a lack of support for the competing model in which body disparagement predicts internalization of the thin-ideal over time (see Figure 25).

Model 3 tested an alternate hypothesis in which internalization of the thin-ideal and body image are reciprocally related between Time 2 and Time 4. Paths were included from the Internalization of Thin-Ideal Time 2 Factor to the Body Image Time 4 Factor and from the Body Image Time 2 Factor to the Internalization of Thin-Ideal Time 4 Factor (see Figure 25). Model 3 also showed an acceptable fit to the data (see Table 13). Consistent with Model 1 and Model 2, the path from the Internalization of Thin-Ideal Time 2 Factor to the Body Image Time 4 Factor was significant, but the path from the Body Image Time 2 Factor to the Internalization of Thin-Ideal Time 4 Factor was not significant (see Figure 25). Model 3 did represent a significant improvement over Model 2 ($\Delta\chi^2 = 4.98$, $df=1$, $p < .05$), but not over Model 1 ($\Delta\chi^2 = 0.26$, $df=1$, $p > .05$) or over the Baseline Model (see Table 13). Although this model indicates that internalization of the thin-ideal predicts body disparagement over time, Model 3 is not a meaningful improvement over the baseline model, and the baseline model would be preferred for the sake of parsimony. Thus, Model 1 appears to be the best representation of the cross-lagged effects between internalization of the thin-ideal and body image.

The time lag between internalization of the thin-ideal and body image as measured at Time 2 and Time 4 was approximately 12 months, and the relationship between internalization of the thin-ideal and body image may be more immediate. However, it is possible that the causal time lag is less than 12 months. Models 4, 5, and 6 examined the simultaneous effects between the Internalization of Thin-Ideal Factor and the Body Image Factor at Time 4. The correlation between the disturbances for the Time 4 factors was removed from these models. Thus, Models 4, 5, and 6 represent reparameterizations of the baseline model and are not nested within the baseline model.

Model 4 tested the empirically supported path from the Internalization of Thin-Ideal Time 4 Factor to the Body Image Time 4 Factor (see Figure 25). This model showed an acceptable fit with the data (see Table 13). Further, the path from the Internalization of Thin-Ideal Time 4 Factor to the Body Image Time 4 Factor was significant (see Figure 25). These results indicate that internalization of the thin-ideal predicts body disparagement. However, it should be noted that this effect has a relatively short time lag.

Model 5 tested the competing simultaneous hypothesis in which body disparagement predicts internalization of the thin-ideal. This model specified a path from the Body Image Time 4 Factor to the Internalization of Thin-Ideal Time 4 Factor (see Figure 25). This model showed an acceptable fit with the data (see Table 13), and the path from the Body Image Time 4 Factor to the Internalization of Thin-Ideal Time 4 Factor was significant (see Figure 25). Thus, support also was provided for the competing model, indicating that body disparagement leads to internalization of the thin-ideal within a relatively short time lag. However, Hoyle and Panter (1995) noted that simply switching the direction of the path between two constructs may not result in any change in the parameter estimate or the overall fit of the model. Thus, it is difficult to establish directionality.

Model 6 tested the contemporaneous reciprocal relationship between internalization of the thin-ideal and body image. This model specified a path from the Internalization of Thin-Ideal Time 4 Factor to the Body Image Time 4 Factor and from the Body Image Time 4 Factor to the Internalization of Thin-Ideal Time 4 Factor (see Figure 25). This model demonstrated acceptable fit with the data (see Table 13), but only the path from the Internalization of Thin-Ideal Time 4 Factor to the Body Image Time 4 Factor was significant (see Figure 25). Model 6 also was run with the effects constrained to be equal, but this resulted in a significant decrement in model fit

($\Delta\chi^2 = 133.36, df=1, p < .001$). This indicates that the path from the Internalization of Thin-Ideal Time 4 Factor to the Body Image Time 4 Factor is significantly larger than the path from the Body Image Time 4 Factor to the Internalization of Thin-Ideal Time 4 Factor. Further, Model 6 represents a significant improvement in fit over Model 4 ($\Delta\chi^2 = 13.93, df=1, p < .001$) and over Model 5 ($\Delta\chi^2 = 6.64, df=1, p = .01$). Thus, Model 6 is the best representation of the simultaneous effects between the Internalization of Thin-Ideal Time 4 Factor and the Body Image Time 4 Factor. The results from Model 6 converge with the results from Model 4 examining the cross lagged effects and indicate that internalization of the thin-ideal results in body disparagement but body disparagement does not predict increased internalization of the thin-ideal.

Internalization of Thin-Ideal and Body Image at Time 1 and Time 4

Measurement model. Examination of the relationship between Time 1 and Time 4 for the constructs of internalization of thin-ideal and the constructs of body image also was begun by conducting a confirmatory test of the measurement model. Again, it was hypothesized a priori that the BAAR Physically Fit subscale and BAAR Attractive and Thin subscale would represent the Internalization of Thin-Ideal Factors, and the BPSS Body Factor, BPSS Face Factor, and the MBSRQ-AE would represent the Body Image Factors. The BPSS Face Factor for each time failed to load on the Body Image Factor, and these variables were removed from the measurement model.

Using the EQS program for structural equation modeling and the Maximum Likelihood (ML) estimation procedure to estimate the parameters of the model, the confirmatory factor analysis (CFA) supported the resulting measurement model, and the overall fit of the data was

good (see Table 13). All measured variables loaded positively on the corresponding constructs. Thus, the Internalization of Thin-Ideal Factors represented greater internalization of the thin-ideal, and the Body Image Factors represented positive body image. Again, all factors were allowed to intercorrelate and the error variance for each measured variable at Time 1 was correlated with the error variance for the same measured variable at Time 4. The measurement model with standardized parameter estimates (factor loadings and error variances) is displayed in Figure 26.

Examination of the relationships among the factors in the measurement model (see Figure 26) revealed positive and significant correlations between the Internalization of Thin-ideal Factor at Time 1 and at Time 4 ($r = .82, p < .05$) and between the Body Image Factor at Time 1 and at Time 4 ($r = .76, p < .05$), suggesting that the rank ordering for these constructs was relatively temporally stable. The measurement model also showed negative and significant correlations between the within-time constructs ($r = -.23, p < .05$ for Time 1; $r = -.49, p < .05$ for Time 4) as well as negative and significant cross-time correlations between the Internalization of Thin-Ideal Time 1 Factor and the Body Image Time 4 Factor ($r = -.27, p < .05$) and between the Body Image Time 1 Factor and the Internalization of Thin-Ideal Time 4 Factor ($r = -.33, p < .05$). Again, the Time 1 measures preceded the Time 4 measures, suggesting temporal precedence in the cross-time correlation. Further, both the within-time correlations and the cross-time correlations between the constructs are significant, and the cross-time correlations are generally slightly weaker than the within-time correlations, suggesting that internalization of thin-ideal may also be reciprocally related to body image. A baseline stability model, 3 alternative models representing cross-lagged effects, and 3 models representing simultaneous effects were also constructed to further examine the structural relationships between internalization of thin-ideal

and body image at Time 1 and Time 4. These models are described below and presented in Figure 27.

Structural models. Only the paths from Time 1 latent factors to the corresponding Time 4 latent factors were included in this baseline model. In addition, latent factors at Time 1 were correlated, Time 4 disturbances for the latent factors were correlated, and measurement errors for measured variables at Time 1 were correlated with the corresponding measurement error at Time 4. The baseline model fit the data well (see Table 13).

The empirically supported path from the Internalization of Thin-Ideal Time 1 Factor to the Body Image Time 4 Factor was specified in Model 1 (see Figure 27). This model showed an acceptable fit to the data, and the chi-square change was significant (see Table 13). Model 1 represents an improvement over the baseline model. Additionally, the specified path from the Internalization of Thin-Ideal Time 1 Factor to the Body Image Time 4 Factor was significant, indicating that internalization of the thin-ideal predicts body disparagement over time (see Figure 27).

Testing the competing hypothesis, Model 2 specified a path from the Body Image Time 1 Factor to the Internalization of Thin-Ideal Time 4 Factor (see Figure 27). Again, this model showed an acceptable fit to the data, but the non-significant chi-square change indicated that the baseline model still better represented the data (see Table 13). Further, the specified path from the Body Image Time 1 Factor to the Internalization of Thin-Ideal Time 4 Factor was not significant, demonstrating a lack of support for the competing model in which body disparagement predicts internalization of the thin-ideal over time (see Figure 27).

Model 3 tested an alternate hypothesis in which internalization of the thin-ideal and body image are reciprocally related between Time 1 and Time 4. Paths were included from the Internalization of Thin-Ideal Time 1 Factor to the Body Image Time 4 Factor and from the Body Image Time 1 Factor to the Internalization of Thin-Ideal Time 4 Factor (see Figure 27). Model 3 also showed an acceptable fit to the data (see Table 13). Consistent with Model 1 and Model 2, the path from the Internalization of Thin-Ideal Time 1 Factor to the Body Image Time 4 Factor was significant, but the path from the Body Image Time 1 Factor to the Internalization of Thin-Ideal Time 4 Factor was not significant (see Figure 27). Model 3 did represent a significant improvement over the Baseline Model (see Table 13) and over Model 2 ($\Delta\chi^2 = 6.29, df=1, p < .05$), but not over Model 1 ($\Delta\chi^2 = 3.08, df=1, p > .05$). Although this model indicates that internalization of the thin-ideal predicts body disparagement, Model 3 is not a meaningful improvement over Model 1, and Model 1 would be preferred for the sake of parsimony. Thus, Model 1 appears to be the best representation of the cross-lagged effects between internalization of the thin-ideal and body image.

The time lag between internalization of the thin-ideal and body image as measured at Time 1 and Time 4 was approximately 18 months. However, it is possible that the causal time lag is less than 18 months, and the relationship between internalization of the thin-ideal and body image may be more immediate. Models 4, 5, and 6 examined the simultaneous effects between the Internalization of Thin-Ideal Factor and the Body Image Factor at Time 4. The correlation between the disturbances for the Time 4 factors was removed from these models. Thus, Models 4, 5, and 6 represent reparameterizations of the baseline model and are not nested within the baseline model.

Model 4 tested the empirically supported path from was added from the Internalization of Thin-Ideal Time 4 Factor to the Body Image Time 4 Factor (see Figure 27). This model showed an acceptable fit with the data (see Table 13). Further, the path from the Internalization of Thin-Ideal Time 4 Factor to the Body Image Time 4 Factor was significant (see Figure 27). These results indicate that internalization of the thin-ideal predicts body disparagement. However, it should be noted that this effect has a relatively short time lag.

Model 5 tested the competing simultaneous hypothesis in which body image predicts internalization of the thin-ideal. This model specified a path from the Body Image Time 4 Factor to the Internalization of Thin-Ideal Time 4 Factor (see Figure 27). This model showed an acceptable fit with the data (see Table 13), and the path from the Body Image Time 4 Factor to the Internalization of Thin-Ideal Time 4 Factor was significant (see Figure 27). Thus, support also was provided for the competing model, indicating that body disparagement leads to internalization of the thin-ideal within a relatively short time lag. However, Hoyle and Panter (1995) noted that simply switching the direction of the path between two constructs may not result in any change in the parameter estimate or the overall fit of the model. Thus, it is difficult to establish directionality.

Model 6 tested the contemporaneous reciprocal relationship between internalization of the thin-ideal and body image. This model specified a path from the Internalization of Thin-Ideal Time 4 Factor to the Body Image Time 4 Factor and from the Body Image Time 4 Factor to the Internalization of Thin-Ideal Time 4 Factor (see Figure 27). This model demonstrated acceptable fit with the data (see Table 13), and both the paths between the Internalization of Thin-Ideal Time 4 Factor to the Body Image Time 4 Factor were significant (see Figure 27). Model 6 also was run with the effects constrained to be equal, but this resulted in a significant decrement in

model fit ($\Delta\chi^2 = 156.93, df=1, p < .001$). This indicates that the path from the Internalization of Thin-Ideal Time 4 Factor to the Body Image Time 4 Factor is stronger than the path from the Body Image Time 4 Factor to the Internalization of Thin-Ideal Time 4 Factor. However, both paths are statistically significant. Further, Model 6 represents a significant improvement in fit over Model 4 ($\Delta\chi^2 = 4.96, df=1, p < .05$) and over Model 5 ($\Delta\chi^2 = 14.62, df=1, p = .001$). Thus, Model 6 is the best representation of the simultaneous effects between the Internalization of Thin-Ideal Time 4 Factor and the Body Image Time 4 Factor. The results from Model 6 converge with the results from Model 4 examining the cross lagged effects and indicate that internalization of the thin-ideal results in body disparagement. The results also indicate that body disparagement leads to increased internalization of the thin-ideal, but this effect may have a relatively short time lag.

Internalization of Thin-Ideal and Eating Disturbance at Time 1 and Time 2

Previous research has found that internalization of the thin ideal predicted onset of bulimic pathology (Stice & Argas, 1998) and dieting behaviors (Stice, 2001a), and experimental reduction of the internalization of the thin ideal resulted in a reduction of dieting behaviors and reduced symptoms of bulimia (Stice, Mazotti et al., 2000). The previously presented models in this study also demonstrated results consistent with Stice and colleagues. To provide additional support for the relationship between these constructs, the hypothesized path in which internalization of the thin-ideal predicts eating disturbance as well as 2 alternative paths suggesting that (a) eating disturbance leads to increased internalization of the thin-ideal and (b) internalization of the thin-ideal and eating disturbance are reciprocally related were tested.

Measurement model. Examination of the relationship between Time 1 and Time 2 for the constructs of internalization of the thin-ideal and the constructs of eating disturbance was begun by conducting a confirmatory test of the measurement model. Consistent with the previously presented measurement models for these factors, it was hypothesized a priori that the Beliefs About Attractiveness (BAAR) Physically Fit subscale and Attractive and Thin subscale would represent the Internalization of Thin-Ideal Factors, and the Bulimia Test-Revised (BULIT-R) and the Eating Attitudes Test-26 (EAT-26) would represent the Eating Disturbance Factors.

Using the EQS program for structural equation modeling and the Maximum Likelihood (ML) estimation procedure to estimate the parameters of the model, the confirmatory factor analysis (CFA) supported the resulting measurement model, and the overall fit of the data was good (see Table 14). All measured variables loaded positively on the corresponding constructs. Thus, the Internalization of Thin-Ideal Factors represented greater internalization of the thin-ideal, and the Eating Disturbance Factors represented eating disordered symptomatology. All factors were allowed to intercorrelate and the error variance for each measured variable at Time 1 was correlated with the error variance for the same measured variable at Time 2. The measurement model with standardized parameter estimates (factor loadings and error variances) is displayed in Figure 28.

Examination of the relationships among the factors in the measurement model (see Figure 28) revealed positive and significant correlations between the Internalization of Thin-ideal Factor at Time 1 and at Time 2 ($r = .74, p < .05$) and between the Eating Disturbance Factor at Time 1 and at Time 2 ($r = .82, p < .05$), suggesting that the rank ordering for these constructs was relatively temporally stable. The measurement model also showed positive and significant correlations between the within-time constructs ($r = .42, p < .05$ for Time 1; $r = .47, p < .05$ for

Time 2) as well as positive and significant cross-time correlations between the Internalization of Thin-Ideal Time 1 Factor and the Eating Disturbance Time 2 Factor ($r = .40, p < .05$) and between the Eating Disturbance Time 1 Factor and the Internalization of Thin-Ideal Time 2 Factor ($r = .36, p < .05$). The Time 1 measures preceded the Time 2 measures, suggesting temporal precedence in the cross-time correlation. Further, both the within-time correlations and the cross-time correlations between the constructs are significant, and that the cross-time correlations are slightly weaker than the within-time correlations, suggesting that internalization of thin-ideal also may be reciprocally related to eating disturbance. To properly evaluate the relationships among the constructs, it is recommended that a predictor variable should account for variance in a criterion variable while controlling for the temporal stability of the criterion variable (Newcomb, 1994). Thus, a baseline stability model, 3 alternative models representing cross-lagged effects, and 3 models representing simultaneous effects were constructed to further examine the structural relationships between internalization of thin-ideal and eating disturbance at Time 1 and Time 2. These models are described below and presented in Figure 29.

Structural models. To compare competing models, a baseline stability model was created. Only the paths from Time 1 latent factors to the corresponding Time 2 latent factors were included in this baseline model. In addition, latent factors at Time 1 were correlated and Time 2 disturbances for the latent factors were correlated. The measurement errors for measured variables at Time 1 were correlated with the corresponding measurement error at Time 2 to prevent the stability paths from being inflated as a result of shared measurement error (Bollen, 1989). The baseline model fit the data well (see Table 14).

The empirically supported path from the Internalization of Thin-Ideal Time 1 Factor to the Eating Disturbance Time 2 Factor was specified in Model 1 (see Figure 29). This model showed an acceptable fit to the data, but the chi-square change was non-significant (see Table 14). Thus, Model 1 did not represent an improvement over the baseline model. In addition, the specified path from the Internalization of Thin-Ideal Time 1 Factor to the Eating Disturbance Time 2 Factor was not significant, indicating a lack of support for the expected relationship in which internalization of the thin-ideal would predict eating disturbance over time (see Figure 29).

Testing the competing hypothesis, Model 2 specified a path from the Eating Disturbance Time 1 Factor to the Internalization of Thin-Ideal Time 2 Factor (see Figure 29).). Again, this model showed an acceptable fit to the data, but the non-significant chi-square change indicated that the baseline model still better represented the data (see Table 14). Further, the specified path from the Eating Disturbance Time 1 Factor to the Internalization of Thin-Ideal Time 2 Factor was not significant, demonstrating a lack of support for the competing model in which eating disturbance would predict internalization of the thin-ideal over time (see Figure 29).

Model 3 tested an alternate hypothesis in which internalization of the thin-ideal and eating disturbance would be reciprocally related between Time 1 and Time 2. Paths were included from the Internalization of Thin-Ideal Time 1 Factor to the Eating Disturbance Time 2 Factor and from the Eating Disturbance Time 1 Factor to the Internalization of Thin-Ideal Time 2 Factor (see Figure 29). Model 3 also showed an acceptable fit to the data, but the non-significant chi-square change indicated that the baseline model still better represented the data (see Table 14). Consistent with Model 1 and Model 2, the specified cross-time paths were not

significant (see Figure 29). Thus, these results did not provide evidence for the reciprocal relationship.

The time lag between internalization of the thin-ideal and eating disturbance as measured at Time 1 and Time 2 was approximately 6 months. However, it is possible that the causal time lag is less than 6 months, and the relationship between internalization of the thin-ideal and eating disturbance may be more immediate. Models 4, 5, and 6 examined the simultaneous effects between the Internalization of Thin-Ideal Factor and the Eating Disturbance Factor at Time 2. The correlation between the disturbances for the Time 2 factors was removed from these models. Thus, Models 4, 5, and 6 represent reparameterizations of the baseline model and are not nested within the baseline model.

Model 4 tested the empirically supported path from the Internalization of Thin-Ideal Time 2 Factor to the Eating Disturbance Time 2 Factor (see Figure 29). This model showed an acceptable fit with the data (see Table 14). Further, the path from the Internalization of Thin-Ideal Time 2 Factor to the Eating Disturbance Time 2 Factor was significant (see Figure 29). These results indicate that internalization of the thin-ideal predicts eating disturbance. However, it should be noted that this effect has a relatively short time lag.

Model 5 tested the competing simultaneous hypothesis in which eating disturbance predicts internalization of the thin-ideal. This model specified a path from the Eating Disturbance Time 2 Factor to the Internalization of Thin-Ideal Time 2 Factor (see Figure 29). This model showed an acceptable fit with the data (see Table 14), and the path from the Eating Disturbance Time 2 Factor to the Internalization of Thin-Ideal Time 2 Factor was significant (see Figure 29). Thus, support also was provided for the competing model, indicating that eating disturbance leads to internalization of the thin-ideal within a relatively short time lag. However, Hoyle and

Panter (1995) noted that simply switching the direction of the path between two constructs may not result in any change in the parameter estimate or the overall fit of the model. Thus, it is difficult to establish directionality.

Model 6 tested the contemporaneous reciprocal relationship between internalization of the thin-ideal and eating disturbance. This model specified a path from the Internalization of Thin-Ideal Time 2 Factor to the Eating Disturbance Time 2 Factor and from the Eating Disturbance Time 2 Factor to the Internalization of Thin-Ideal Time 2 Factor (see Figure 29). This model demonstrated acceptable fit with the data (see Table 14), but only the path from the Internalization of Thin-Ideal Time 2 Factor to the Eating Disturbance Time 2 Factor was significant (see Figure 29). Model 6 also was run with the effects constrained to be equal, but this resulted in a significant decrement in model fit ($\Delta\chi^2 = 6.65, df=1, p < .01$). This indicates that the path from the Internalization of Thin-Ideal Time 2 Factor to the Eating Disturbance Time 2 Factor is significantly larger than the path from the Eating Disturbance Time 2 Factor to the Internalization of Thin-Ideal Time 2 Factor. These results are consistent with the results from Model 4, and they indicate that internalization of the thin-ideal leads to eating disturbance within a relatively short time frame. Further, Model 6 represents a significant improvement in fit over Model 5 ($\Delta\chi^2 = 6.91, df=1, p < .01$). However, Model 6 does not represent a significant improvement over Model 4 ($\Delta\chi^2 = 2.36, df=1, p > .05$). Thus, Model 4 is the best representation of the simultaneous effects between the Internalization of Thin-Ideal Time 2 Factor and the Eating Disturbance Time 2 Factor.

Internalization of Thin-Ideal and Eating Disturbance at Time 2 and Time 4

Measurement model. Examination of the relationship between Time 2 and Time 4 for the constructs of internalization of thin-ideal and the constructs of eating disturbance also was begun by conducting a confirmatory test of the measurement model. Again, it was hypothesized a priori that the BAAR Physically Fit subscale and BAAR Attractive and Thin subscale would represent the Internalization of Thin-Ideal Factors and the BULIT-R and the EAT-26 would represent the Eating Disturbance Factors.

Using the EQS program for structural equation modeling and the Maximum Likelihood (ML) estimation procedure to estimate the parameters of the model, the confirmatory factor analysis (CFA) supported the resulting measurement model, and the overall fit of the data was good (see Table 14). All measured variables loaded positively on the corresponding constructs. Thus, the Internalization of Thin-Ideal Factors represented greater internalization of the thin-ideal, and the Eating Disturbance Factors represented eating disordered symptomatology. Again, all factors were allowed to intercorrelate and the error variance for each measured variable at Time 2 was correlated with the error variance for the same measured variable at Time 4. The measurement model with standardized parameter estimates (factor loadings and error variances) is displayed in Figure 30.

Examination of the relationships among the factors in the measurement model (see Figure 30) revealed positive and significant correlations between the Internalization of Thin-ideal Factor at Time 2 and at Time 4 ($r = .85, p < .05$) and between the Eating Disturbance Factor at Time 2 and at Time 4 ($r = .77, p < .05$), suggesting that the rank ordering for these constructs was relatively temporally stable. The measurement model also showed positive and significant correlations between the within-time constructs ($r = .45, p < .05$ for Time 2; $r = .48, p < .05$ for

Time 4) as well as positive and significant cross-time correlations between the Internalization of Thin-Ideal Time 2 Factor and the Eating Disturbance Time 4 Factor ($r = .45, p < .05$) and between the Eating Disturbance Time 2 Factor and the Internalization of Thin-Ideal Time 4 Factor ($r = .39, p < .05$). Again, the Time 2 measures preceded the Time 4 measures, suggesting temporal precedence in the cross-time correlation. Further, both the within-time correlations and the cross-time correlations between the constructs are significant and the cross-time correlations are similar or generally slightly weaker than the within-time correlations, suggesting that internalization of thin-ideal may also be reciprocally related to eating disturbance. A baseline stability model, 3 alternative models representing cross-lagged effects, and 3 models representing simultaneous effects were also constructed to further examine the structural relationships between internalization of thin-ideal and eating disturbance at Time 2 and Time 4. These models are described below and presented in Figure 31.

Structural models. Only the paths from Time 2 latent factors to the corresponding Time 4 latent factors were included in this baseline model. In addition, latent factors at Time 2 were correlated, Time 4 disturbances for the latent factors were correlated, and measurement errors for measured variables at Time 2 were correlated with the corresponding measurement error at Time 4. The baseline model fit the data well (see Table 14).

The empirically supported path from the Internalization of Thin-Ideal Time 2 Factor to the Eating Disturbance Time 4 Factor was specified in Model 1 (see Figure 31). This model showed an acceptable fit to the data, and the significant chi-square change indicated that this model fit the data better than the baseline model (see Table 14). Additionally, the specified path from the Internalization of Thin-Ideal Time 2 Factor to the Eating Disturbance Time 4 Factor

was significant, indicating internalization of the thin-ideal predicts eating disturbance over time (see Figure 31).

Testing the competing hypothesis, Model 2 specified a path from the Eating Disturbance Time 2 Factor to the Internalization of Thin-Ideal Time 4 Factor (see Figure 31). Again, this model showed an acceptable fit to the data, but the non-significant chi-square change indicated that the baseline model still better represented the data (see Table 14). Further, the specified path from the Eating Disturbance Time 2 Factor to the Internalization of Thin-Ideal Time 4 Factor was not significant, demonstrating a lack of support for the competing model in which eating disturbance would predict internalization of the thin-ideal over time (see Figure 31).

Model 3 tested an alternate hypothesis in which internalization of the thin-ideal and eating disturbance would be reciprocally related between Time 2 and Time 4. Paths were included from the Internalization of Thin-Ideal Time 2 Factor to the Eating Disturbance Time 4 Factor and from the Eating Disturbance Time 2 Factor to the Internalization of Thin-Ideal Time 4 Factor (see Figure 31). Model 3 also showed an acceptable fit to the data (see Table 14). Consistent with Model 1 and Model 2, the path from the Internalization of Thin-Ideal Time 2 Factor to the Eating Disturbance Time 4 Factor was significant, but the path from the Eating Disturbance Time 2 Factor to the Internalization of Thin-Ideal Time 4 Factor was not significant (see Figure 31). Model 3 did represent a significant improvement over Model 2 ($\Delta\chi^2 = 4.51$, $df = 1$, $p < .05$), but not over Model 1 ($\Delta\chi^2 = 0.02$, $df = 1$, $p > .05$) or the Baseline Model (see Table 14). Although this model demonstrates support that internalization of the thin-ideal predicts eating disturbance over time, Model 3 is not a meaningful improvement over the baseline model, and the baseline model would be preferred over Model 3 for the sake of parsimony. Thus, Model

1 appears to be the best representation of the cross-lagged effects between internalization of the thin-ideal and eating disturbance.

The time lag between internalization of the thin-ideal and eating disturbance as measured at Time 2 and Time 4 was approximately 12 months, and the relationship between internalization of the thin-ideal and eating disturbance may be more immediate. Models 4, 5, and 6 examined the simultaneous effects between the Internalization of Thin-Ideal Factor and the Eating Disturbance Factor at Time 4. The correlation between the disturbances for the Time 4 factors was removed from these models. Thus, Models 4, 5, and 6 represent reparameterizations of the baseline model and are not nested within the baseline model.

Model 4 tested the empirically supported path from the Internalization of Thin-Ideal Time 4 Factor to the Eating Disturbance Time 4 Factor (see Figure 31). This model showed an acceptable fit with the data (see Table 14). Further, the path from the Internalization of Thin-Ideal Time 4 Factor to the Eating Disturbance Time 4 Factor was significant (see Figure 31). These results indicate that internalization of the thin-ideal predicts eating disturbance. However, it should be noted that this effect has a relatively short time lag.

Model 5 tested the competing simultaneous hypothesis in which eating disturbance predicts internalization of the thin-ideal. This model specified a path from the Eating Disturbance Time 4 Factor to the Internalization of Thin-Ideal Time 4 Factor (see Figure 31). This model showed an acceptable fit with the data (see Table 14), and the path from the Eating Disturbance Time 4 Factor to the Internalization of Thin-Ideal Time 4 Factor was significant (see Figure 31). Thus, support also was provided for the competing model, indicating that eating disturbance leads to internalization of the thin-ideal within a relatively short time lag. However, Hoyle and Panter (1995) noted that simply switching the direction of the path between two constructs may

not result in any change in the parameter estimate or the overall fit of the model. Thus, it is difficult to establish directionality.

Model 6 tested the contemporaneous reciprocal relationship between internalization of the thin-ideal and eating disturbance. This model specified a path from the Internalization of Thin-Ideal Time 4 Factor to the Eating Disturbance Time 4 Factor and from the Eating Disturbance Time 4 Factor to the Internalization of Thin-Ideal Time 4 Factor (see Figure 31). This model demonstrated acceptable fit with the data (see Table 14), but only the path from the Internalization of Thin-Ideal Time 4 Factor to the Eating Disturbance Time 4 Factor was significant (see Figure 31). Model 6 also was run with the effects constrained to be equal, but this resulted in a significant decrement in model fit ($\Delta\chi^2 = 8.20, df=1, p < .01$). This indicates that the path from the Internalization of Thin-Ideal Time 4 Factor to the Eating Disturbance Time 4 Factor is significantly larger than the path from the Eating Disturbance Time 4 Factor to the Internalization of Thin-Ideal Time 4 Factor. Further, Model 6 represents a significant improvement in fit over Model 5 ($\Delta\chi^2 = 8.37, df=1, p < .01$). However, Model 6 does not represent a significant improvement over Model 4 ($\Delta\chi^2 = 0.74, df=1, p > .05$). Thus, Model 4 is the best representation of the simultaneous effects between the Internalization of Thin-Ideal Time 4 Factor and the Eating Disturbance Time 4 Factor. The results from Model 6 converge with the results from Model 4 examining the cross lagged effects and indicate that internalization of the thin-ideal results in eating disturbance but eating disturbance does not predict increased internalization of the thin-ideal.

Internalization of Thin-Ideal and Eating Disturbance at Time 1 and Time 4

Measurement model. Examination of the relationship between Time 1 and Time 4 for the constructs of internalization of thin-ideal and the constructs of eating disturbance also was begun by conducting a confirmatory test of the measurement model. Again, it was hypothesized a priori that the BAAR Physically Fit subscale and BAAR Attractive and Thin subscale would represent the Internalization of Thin-Ideal Factors and the BULIT-R and the EAT-26 would represent the Eating Disturbance Factors.

Using the EQS program for structural equation modeling and the Maximum Likelihood (ML) estimation procedure to estimate the parameters of the model, the confirmatory factor analysis (CFA) supported the resulting measurement model, and the overall fit of the data was good (see Table 14). All measured variables loaded positively on the corresponding constructs. Thus, the Internalization of Thin-Ideal Factors represented greater internalization of the thin-ideal, and the Eating Disturbance Factors represented eating disordered symptomatology. Again, all factors were allowed to intercorrelate and the error variance for each measured variable at Time 1 was correlated with the error variance for the same measured variable at Time 4. The measurement model with standardized parameter estimates (factor loadings and error variances) is displayed in Figure 32.

Examination of the relationships among the factors in the measurement model (see Figure 32) revealed positive and significant correlations between the Internalization of Thin-ideal Factor at Time 1 and at Time 4 ($r = .76, p < .05$) and between the Eating Disturbance Factor at Time 1 and at Time 4 ($r = .73, p < .05$), suggesting that the rank ordering for these constructs was relatively temporally stable. The measurement model also showed positive and significant correlations between the within-time constructs ($r = .45, p < .05$ for Time 1; $r = .47, p < .05$ for

Time 4) as well as positive and significant cross-time correlations between the Internalization of Thin-Ideal Time 1 Factor and the Eating Disturbance Time 4 Factor ($r = .42, p < .05$) and between the Eating Disturbance Time 1 Factor and the Internalization of Thin-Ideal Time 4 Factor ($r = .32, p < .05$). Again, the Time 1 measures preceded the Time 4 measures, suggesting temporal precedence in the cross-time correlation. Further, both the within-time correlations and the cross-time correlations between the constructs are significant and the cross-time correlations are generally slightly weaker than the within-time correlations, suggesting that internalization of thin-ideal may also be reciprocally related to eating disturbance. A baseline stability model, 3 alternative models representing cross-lagged effects, and 3 models representing simultaneous effects were also constructed to further examine the structural relationships between internalization of thin-ideal and eating disturbance at Time 1 and Time 4. These models are described below and presented in Figure 33.

Structural models. Only the paths from Time 1 latent factors to the corresponding Time 4 latent factors were included in this baseline model. In addition, latent factors at Time 1 were correlated, Time 4 disturbances for the latent factors were correlated, and measurement errors for measured variables at Time 1 were correlated with the corresponding measurement error at Time 4. The baseline model fit the data well (see Table 14).

The empirically supported path from the Internalization of Thin-Ideal Time 1 Factor to the Eating Disturbance Time 4 Factor was specified in Model 1 (see Figure 33). This model showed an acceptable fit to the data, but the chi-square change was non-significant (see Table 14). Thus, Model 1 did not represent an improvement over the baseline model. In addition, the specified path from the Internalization of Thin-Ideal Time 1 Factor to the Eating Disturbance

Time 4 Factor was not significant, indicating a lack of support for the expected relationship in which internalization of the thin-ideal would predict eating disturbance over time (see Figure 33).

Testing the competing hypothesis, Model 2 specified a path from the Eating Disturbance Time 1 Factor to the Internalization of Thin-Ideal Time 4 Factor (see Figure 33). Again, this model showed an acceptable fit to the data, but the non-significant chi-square change indicated that the baseline model still better represented the data (see Table 14). Further, the specified path from the Eating Disturbance Time 1 Factor to the Internalization of Thin-Ideal Time 4 Factor was not significant, demonstrating a lack of support for the competing model in which eating disturbance would predict internalization of the thin-ideal over time (see Figure 33).

Model 3 tested an alternate hypothesis in which internalization of the thin-ideal and eating disturbance would be reciprocally related between Time 1 and Time 4. Paths were included from the Internalization of Thin-Ideal Time 1 Factor to the Eating Disturbance Time 4 Factor and from the Eating Disturbance Time 1 Factor to the Internalization of Thin-Ideal Time 4 Factor (see Figure 33). Model 3 also showed an acceptable fit to the data, but the non-significant chi-square change indicated that the baseline model still better represented the data (see Table 14). Consistent with Model 1 and Model 2, the specified cross-time paths were not significant (see Figure 33). Thus, these results did not provide evidence for the reciprocal relationship.

The time lag between internalization of the thin-ideal and eating disturbance as measured at Time 1 and Time 4 was approximately 18 months, and the relationship between internalization of the thin-ideal and eating disturbance may be more immediate. Models 4, 5, and 6 examined the simultaneous effects between the Internalization of Thin-Ideal Factor and the Eating

Disturbance Factor at Time 4. The correlation between the disturbances for the Time 4 factors was removed from these models. Thus, Models 4, 5, and 6 represent reparameterizations of the baseline model and are not nested within the baseline model.

Model 4 tested the empirically supported path from the Internalization of Thin-Ideal Time 4 Factor to the Eating Disturbance Time 4 Factor (see Figure 33). This model showed an acceptable fit with the data (see Table 14). Further, the path from the Internalization of Thin-Ideal Time 4 Factor to the Eating Disturbance Time 4 Factor was significant (see Figure 33). These results indicate that internalization of the thin-ideal predicts eating disturbance. However, it should be noted that this effect has a relatively short time lag.

Model 5 tested the competing simultaneous hypothesis in which eating disturbance predicts internalization of the thin-ideal. This model specified a path from the Eating Disturbance Time 4 Factor to the Internalization of Thin-Ideal Time 4 Factor (see Figure 33). This model showed an acceptable fit with the data (see Table 14), and the path from the Eating Disturbance Time 4 Factor to the Internalization of Thin-Ideal Time 4 Factor was significant (see Figure 33). Thus, support also was provided for the competing model, indicating that eating disturbance leads to internalization of the thin-ideal within a relatively short time lag. However, Hoyle and Panter (1995) noted that simply switching the direction of the path between two constructs may not result in any change in the parameter estimate or the overall fit of the model. Thus, it is difficult to establish directionality.

Model 6 tested the contemporaneous reciprocal relationship between internalization of the thin-ideal and eating disturbance. This model specified a path from the Internalization of Thin-Ideal Time 4 Factor to the Eating Disturbance Time 4 Factor and from the Eating Disturbance Time 4 Factor to the Internalization of Thin-Ideal Time 4 Factor (see Figure 33).

This model demonstrated acceptable fit with the data (see Table 14), but only the path from the Internalization of Thin-Ideal Time 4 Factor to the Eating Disturbance Time 4 Factor was significant (see Figure 33). Model 6 also was run with the effects constrained to be equal, but this resulted in a significant decrement in model fit ($\Delta\chi^2 = 10.08$, $df=1$, $p < .01$). This indicates that the path from the Internalization of Thin-Ideal Time 4 Factor to the Eating Disturbance Time 4 Factor is significantly larger than the path from the Eating Disturbance Time 4 Factor to the Internalization of Thin-Ideal Time 4 Factor. These results are consistent with the results from Model 4 and Model 5, and they indicate that internalization of the thin-ideal leads to eating disturbance within a relatively short time frame. Further, Model 6 represents a significant improvement in fit over Model 5 ($\Delta\chi^2 = 10.31$, $df=1$, $p < .01$). However, Model 6 does not represent a significant improvement over Model 4 ($\Delta\chi^2 = 0.43$, $df=1$, $p > .05$). Thus, Model 4 is the best representation of the simultaneous effects between the Internalization of Thin-Ideal Time 4 Factor and the Eating Disturbance Time 4 Factor.

Body Image and Eating Disturbance at Time 1 and Time 2

Research has demonstrated a strong association between body dissatisfaction and eating pathology (Leon et al., 1993; Mintz & Betz, 1988, Stice, 1994), and longitudinal research has shown that body dissatisfaction resulted in subsequent eating pathology (Attie & Brooks-Gunn, 1989; Striegel-Moore et al., 1988). The previously presented models in this study are also consistent with the previous research. To provide additional support for the relationship between these constructs, the hypothesized path in which body dissatisfaction predicts eating disturbance as well as 2 alternative paths suggesting that (a) eating disturbance leads to increased body

dissatisfaction and (b) body dissatisfaction and eating disturbance are reciprocally related were tested using prospective data.

Measurement model. Examination of the relationship between Time 1 and Time 2 for the constructs body image and the constructs of eating disturbance was begun by conducting a confirmatory test of the measurement model. Consistent with the previously presented measurement models for these factors, it was hypothesized a priori that the Body Parts Satisfaction Scale (BPSS) Body Factor, BPSS Face Factor, and the Multidimensional Body Self Relations Questionnaire-Appearance Evaluation subscale (MBSRQ-AE) would represent the Body Image Factors, and the Bulimia Test-Revised (BULIT-R) and the Eating Attitudes Test-26 (EAT-26) would represent the Eating Disturbance Factors. The BPSS Face Factor for each time point failed to load on the Body Image Factor, and these variables were removed from the measurement model. The BULIT-R and the EAT-26 loaded appropriately on the Eating Disturbance Factors. At this point, the error variance for the BULIT-R Time 1 was exhibiting non-significant error variance. Following Bollen's (1989) recommendations, the error variance was set to 19.95.

Using the EQS program for structural equation modeling and the Maximum Likelihood (ML) estimation procedure to estimate the parameters of the model, the confirmatory factor analysis (CFA) supported the resulting measurement model, and the overall fit of the data was good (see Table 15). All measured variables loaded positively on the corresponding constructs. Thus, the Body Image Factors represented positive body image, and the Eating Disturbance Factors represented eating disordered symptomatology. All factors were allowed to intercorrelate and the error variance for each measured variable at Time 1 was correlated with the error

variance for the same measured variable at Time 2. The measurement model with standardized parameter estimates (factor loadings and error variances) is displayed in Figure 34.

Examination of the relationships among the factors in the measurement model (see Figure 34) revealed positive and significant correlations between Body Image Factor at Time 1 and at Time 2 ($r = .90, p < .05$) and between the Eating Disturbance Factor at Time 1 and at Time 2 ($r = .82, p < .05$), suggesting that the rank ordering for these constructs was relatively temporally stable. The measurement model also showed negative and significant correlations between the within-time constructs ($r = -.51, p < .05$ for Time 1; $r = -.58, p < .05$ for Time 2) as well as negative and significant cross-time correlations between the Body Image Time 1 Factor and the Eating Disturbance Time 2 Factor ($r = -.48, p < .05$) and between the Eating Disturbance Time 1 Factor and the Body Image Time 2 Factor ($r = -.44, p < .05$). The Time 1 measures preceded the Time 2 measures, suggesting temporal precedence in the cross-time correlation. Further, both the within-time correlations and the cross-time correlations between the constructs are significant, and the cross-time correlations are slightly weaker than the within-time correlations, suggesting that body image may also be reciprocally related to eating disturbance. To properly evaluate the relationships among the constructs, it is recommended that a predictor variable should account for variance in a criterion variable while controlling for the temporal stability of the criterion variable (Newcomb, 1994). Thus, a baseline stability model, 3 alternative models representing cross-lagged effects, and 3 models representing simultaneous effects were constructed to further examine the structural relationships between body image and eating disturbance at Time 1 and Time 2. These models are described below and presented in Figure 35.

Structural models. To compare competing models, a baseline stability model was created. Only the paths from Time 1 latent factors to the corresponding Time 2 latent factors were included in this baseline model. In addition, latent factors at Time 1 were correlated and Time 2 disturbances for the latent factors were correlated. The measurement errors for measured variables at Time 1 were correlated with the corresponding measurement error at Time 2 to prevent the stability paths from being inflated as a result of shared measurement error (Bollen, 1989). The baseline model fit the data well (see Table 15).

The empirically supported path from the Body Image Time 1 Factor to the Eating Disturbance Time 2 Factor was specified in Model 1 (see Figure 35). This model showed an acceptable fit to the data, but the chi-square change was non-significant (see Table 15). Thus, Model 1 did not represent an improvement over the baseline model. In addition, the specified path from the Body Image Time 1 Factor to the Eating Disturbance Time 2 Factor was not significant, indicating a lack of support for the expected relationship in which body disparagement would predict eating disturbance.

Testing the competing hypothesis, Model 2 specified a path from the Eating Disturbance Time 1 Factor to the Body Image Time 2 Factor (see Figure 35). Again, this model showed an acceptable fit to the data, but the non-significant chi-square change indicated that the baseline model still better represented the data (see Table 15). Further, the specified path from the Eating Disturbance Time 1 Factor to the Body Image Time 2 Factor was not significant, demonstrating a lack of support for the competing model in which eating disturbance would predict body disparagement (see Figure 35).

Model 3 tested an alternate hypothesis in which body disparagement and eating disturbance would be reciprocally related between Time 1 and Time 2. Paths were included from

the Body Image Time 1 Factor to the Eating Disturbance Time 2 Factor and from the Eating Disturbance Time 1 Factor to the Body Image Time 2 Factor (see Figure 35). Model 3 also showed an acceptable fit to the data, but the non-significant chi-square change indicated that the baseline model still better represented the data (see Table 15). Consistent with Model 1 and Model 2, the specified cross-time paths were not significant (see Figure 35). Thus, these results did not provide evidence for the reciprocal relationship.

The time lag between body image and eating disturbance as measured at Time 1 and Time 2 was approximately 6 months, and the relationship between body image and eating disturbance may be more immediate. Models 4, 5, and 6 examined the simultaneous effects between the Body Image Factor and the Eating Disturbance Factor at Time 4. The correlation between the disturbances for the Time 4 factors was removed from these models. Thus, Models 4, 5, and 6 represent reparameterizations of the baseline model and are not nested within the baseline model.

Model 4 tested the empirically supported path from the Body Image Time 2 Factor to the Eating Disturbance Time 2 Factor (see Figure 35). This model showed an acceptable fit with the data (see Table 15). Further, the path from the Body Image Time 2 Factor to the Eating Disturbance Time 2 Factor was significant (see Figure 35). These results indicate that body disparagement predicts eating disturbance. However, it should be noted that this effect has a relatively short time lag.

Model 5 tested the competing simultaneous hypothesis in which eating disturbance predicts body disparagement. This model specified a path from the Eating Disturbance Time 2 Factor to the Body Image Time 2 Factor (see Figure 35). This model showed an acceptable fit with the data (see Table 15), and the path from the Eating Disturbance Time 2 Factor to the Body

Image Time 2 Factor was significant (see Figure 35). Thus, support also was provided for the competing model, indicating that eating disturbance leads to body disparagement within a relatively short time lag. However, Hoyle and Panter (1995) noted that simply switching the direction of the path between two constructs may not result in any change in the parameter estimate or the overall fit of the model. Thus, it is difficult to establish directionality.

Model 6 tested the contemporaneous reciprocal relationship between body image and eating disturbance. This model specified a path from the Body Image Time 2 Factor to the Eating Disturbance Time 2 Factor and from the Eating Disturbance Time 2 Factor to the Body Image Time 2 Factor (see Figure 35). Only the path from the Body Image Time 2 Factor to the Eating Disturbance Time 2 Factor was significant (see Figure 35). However, several fit indices indicated that this model did not demonstrate acceptable fit with the data (see Table 15). Model 6 also was run with the effects constrained to be equal, but the model failed to converge. Thus, Model 4 and Model 5 appear to be better representation of the simultaneous effects between the Body Image Time 2 Factor and the Eating Disturbance Time 2 Factor.

Body Image and Eating Disturbance at Time 2 and Time 4

Measurement model. Examination of the relationship between Time 2 and Time 4 for the constructs body image and the constructs of eating disturbance was begun by conducting a confirmatory test of the measurement model. Again, it was hypothesized a priori that the BPSS Body Factor, BPSS Face Factor, and the MBSRQ-AE would represent the Body Image Factors, and the BULIT-R and the EAT-26 would represent the Eating Disturbance Factors. The BPSS Face Factor for each time point failed to load on the Body Image Factor, and these variables were removed from the measurement model. The BULIT-R and the EAT-26 loaded

appropriately on the Eating Disturbance Factors. At this point, the error variance for the BULIT-R Time 2 was exhibiting non-significant error variance. Following Bollen's (1989) recommendations, the error variance was set to 22.61.

Using the EQS program for structural equation modeling and the Maximum Likelihood (ML) estimation procedure to estimate the parameters of the model, the confirmatory factor analysis (CFA) supported the resulting measurement model, and the overall fit of the data was good (see Table 15). All measured variables loaded positively on the corresponding constructs. Thus, the Body Image Factors represented positive body image, and the Eating Disturbance Factors represented eating disordered symptomatology. All factors were allowed to intercorrelate and the error variance for each measured variable at Time 2 was correlated with the error variance for the same measured variable at Time 4. The measurement model with standardized parameter estimates (factor loadings and error variances) is displayed in Figure 36.

Examination of the relationships among the factors in the measurement model (see Figure 36) revealed positive and significant correlations between Body Image Factor at Time 2 and at Time 4 ($r = .84, p < .05$) and between the Eating Disturbance Factor at Time 2 and at Time 4 ($r = .78, p < .05$), suggesting that the rank ordering for these constructs was relatively temporally stable. The measurement model also showed negative and significant correlations between the within-time constructs ($r = -.57, p < .05$ for Time 2; $r = -.62, p < .05$ for Time 4) as well as negative and significant cross-time correlations between the Body Image Time 2 Factor and the Eating Disturbance Time 4 Factor ($r = -.44, p < .05$) and between the Eating Disturbance Time 2 Factor and the Body Image Time 4 Factor ($r = -.51, p < .05$). The Time 2 measures preceded the Time 4 measures, suggesting temporal precedence in the cross-time correlation. Further, both the within-time correlations and the cross-time correlations between the constructs are significant,

and the cross-time correlations are slightly weaker than the within-time correlations, suggesting that body image may also be reciprocally related to eating disturbance. A baseline stability model, 3 alternative models representing cross-lagged effects, and 3 models representing simultaneous effects were constructed to further examine the structural relationships between body image and eating disturbance at Time 2 and Time 4. These models are described below and presented in Figure 37.

Structural models. Only the paths from Time 2 latent factors to the corresponding Time 4 latent factors were included in this baseline model. In addition, latent factors at Time 2 were correlated, Time 4 disturbances for the latent factors were correlated, and measurement errors for measured variables at Time 2 were correlated with the corresponding measurement error at Time 4. The baseline model fit the data well (see Table 15).

The empirically supported path from the Body Image Time 2 Factor to the Eating Disturbance Time 4 Factor was specified in Model 1 (see Figure 37). This model showed an acceptable fit to the data, but the chi-square change was non-significant (see Table 15). Thus, Model 1 did not represent an improvement over the baseline model. In addition, the specified path from the Body Image Time 2 Factor to the Eating Disturbance Time 4 Factor was not significant, indicating a lack of support for the expected relationship in which body disparagement would predict eating disturbance.

Testing the competing hypothesis, Model 2 specified a path from the Eating Disturbance Time 2 Factor to the Body Image Time 4 Factor (see Figure 37). Again, this model showed an acceptable fit to the data, but the non-significant chi-square change indicated that the baseline model still better represented the data (see Table 15). Further, the specified path from the Eating

Disturbance Time 2 Factor to the Body Image Time 4 Factor was not significant, demonstrating a lack of support for the competing model in which eating disturbance would predict body disparagement (see Figure 37).

Model 3 tested an alternate hypothesis in which body disparagement and eating disturbance would be reciprocally related between Time 2 and Time 4. Paths were included from the Body Image Time 2 Factor to the Eating Disturbance Time 4 Factor and from the Eating Disturbance Time 2 Factor to the Body Image Time 4 Factor (see Figure 37). Model 3 also showed an acceptable fit to the data, but the non-significant chi-square change indicated that the baseline model still better represented the data (see Table 15). Consistent with Model 1 and Model 2, the specified cross-time paths were not significant (see Figure 37). Thus, these results did not provide evidence for the reciprocal relationship.

The time lag between body image and eating disturbance as measured at Time 2 and Time 4 was approximately 12 months, and the relationship between body image and eating disturbance may be more immediate. Models 4, 5, and 6 examined the simultaneous effects between the Body Image Factor and the Eating Disturbance Factor at Time 4. The correlation between the disturbances for the Time 4 factors was removed from these models. Thus, Models 4, 5, and 6 represent reparameterizations of the baseline model and are not nested within the baseline model.

Model 4 tested the empirically supported path from the Body Image Time 4 Factor to the Eating Disturbance Time 4 Factor (see Figure 37). This model showed an acceptable fit with the data (see Table 15). Further, the path from the Body Image Time 4 Factor to the Eating Disturbance Time 4 Factor was significant (see Figure 37). These results indicate that body

disparagement predicts eating disturbance. However, it should be noted that this effect has a relatively short time lag.

Model 5 tested the competing simultaneous hypothesis in which eating disturbance predicts body disparagement. This model specified a path from the Eating Disturbance Time 4 Factor to the Body Image Time 4 Factor (see Figure 37). This model showed an acceptable fit with the data (see Table 15), and the path from the Eating Disturbance Time 4 Factor to the Body Image Time 4 Factor was significant (see Figure 37). Thus, support also was provided for the competing model, indicating that eating disturbance leads to body disparagement within a relatively short time lag. However, Hoyle and Panter (1995) noted that simply switching the direction of the path between two constructs may not result in any change in the parameter estimate or the overall fit of the model. Thus, it is difficult to establish directionality.

Model 6 tested the contemporaneous reciprocal relationship between body image and eating disturbance. This model specified a path from the Body Image Time 4 Factor to the Eating Disturbance Time 4 Factor and from the Eating Disturbance Time 4 Factor to the Body Image Time 4 Factor (see Figure 37). Only the path from the Body Image Time 4 Factor to the Eating Disturbance Time 4 Factor was significant (see Figure 37). However, several fit indices indicated that this model did not demonstrate acceptable fit with the data (see Table 15). Model 6 also was run with the effects constrained to be equal, but the model failed to converge. Thus, Model 4 and Model 5 appear to be better representation of the simultaneous effects between the Body Image Time 4 Factor and the Eating Disturbance Time 4 Factor.

Body Image and Eating Disturbance at Time 1 and Time 4

Measurement model. Examination of the relationship between Time 1 and Time 4 for the constructs body image and the constructs of eating disturbance was begun by conducting a confirmatory test of the measurement model. Again, it was hypothesized a priori that the BPSS Body Factor, BPSS Face Factor, and the MBSRQ-AE would represent the Body Image Factors, and the BULIT-R and the EAT-26 would represent the Eating Disturbance Factors. The BPSS Face Factor for each time point failed to load on the Body Image Factor, and these variables were removed from the measurement model. The BULIT-R and the EAT-26 loaded appropriately on the Eating Disturbance Factors. At this point, the error variance for the BULIT-R Time 1 was exhibiting non-significant error variance. Following Bollen's (1989) recommendations, the error variance was set to 19.95.

Using the EQS program for structural equation modeling and the Maximum Likelihood (ML) estimation procedure to estimate the parameters of the model, the confirmatory factor analysis (CFA) supported the resulting measurement model, and the overall fit of the data was good (see Table 15). All measured variables loaded positively on the corresponding constructs. Thus, the Body Image Factors represented positive body image, and the Eating Disturbance Factors represented eating disordered symptomatology. All factors were allowed to intercorrelate and the error variance for each measured variable at Time 1 was correlated with the error variance for the same measured variable at Time 4. The measurement model with standardized parameter estimates (factor loadings and error variances) is displayed in Figure 38.

Examination of the relationships among the factors in the measurement model (see Figure 38) revealed positive and significant correlations between Body Image Factor at Time 1 and at Time 4 ($r = .82, p < .05$) and between the Eating Disturbance Factor at Time 1 and at Time 4 ($r =$

.75, $p < .05$), suggesting that the rank ordering for these constructs was relatively temporally stable. The measurement model also showed negative and significant correlations between the within-time constructs ($r = -.51, p < .05$ for Time 1; $r = -.61, p < .05$ for Time 4) as well as negative and significant cross-time correlations between the Body Image Time 1 Factor and the Eating Disturbance Time 4 Factor ($r = -.43, p < .05$) and between the Eating Disturbance Time 1 Factor and the Body Image Time 4 Factor ($r = -.44, p < .05$). The Time 1 measures preceded the Time 4 measures, suggesting temporal precedence in the cross-time correlation. Further, both the within-time correlations and the cross-time correlations between the constructs are significant, and the cross-time correlations are slightly weaker than the within-time correlations, suggesting that body image may also be reciprocally related to eating disturbance. A baseline stability model, 3 alternative models representing cross-lagged effects, and 3 models representing simultaneous effects were constructed to further examine the structural relationships between body image and eating disturbance at Time 1 and Time 4. These models are described below and presented in Figure 39.

Structural models. Only the paths from Time 1 latent factors to the corresponding Time 4 latent factors were included in this baseline model. In addition, latent factors at Time 1 were correlated, Time 4 disturbances for the latent factors were correlated, and measurement errors for measured variables at Time 1 were correlated with the corresponding measurement error at Time 4. The baseline model fit the data well (see Table 15).

The empirically supported path from the Body Image Time 1 Factor to the Eating Disturbance Time 4 Factor was specified in Model 1(see Figure 39). This model showed an acceptable fit to the data, but the chi-square change was non-significant (see Table 15). Thus,

Model 1 did not represent an improvement over the baseline model. In addition, the specified path from the Body Image Time 1 Factor to the Eating Disturbance Time 4 Factor was not significant, indicating a lack of support for the expected relationship in which body disparagement would predict eating disturbance.

Testing the competing hypothesis, Model 2 specified a path from the Eating Disturbance Time 1 Factor to the Body Image Time 4 Factor (see Figure 39). Again, this model showed an acceptable fit to the data, but the non-significant chi-square change indicated that the baseline model still better represented the data (see Table 15). Further, the specified path from the Eating Disturbance Time 1 Factor to the Body Image Time 4 Factor was not significant, demonstrating a lack of support for the competing model in which eating disturbance would predict body disparagement (see Figure 39).

Model 3 tested an alternate hypothesis in which body disparagement and eating disturbance would be reciprocally related between Time 1 and Time 4. Paths were included from the Body Image Time 1 Factor to the Eating Disturbance Time 4 Factor and from the Eating Disturbance Time 1 Factor to the Body Image Time 4 Factor (see Figure 39). Model 3 also showed an acceptable fit to the data, but the non-significant chi-square change indicated that the baseline model still better represented the data (see Table 15). Consistent with Model 1 and Model 2, the specified cross-time paths were not significant (see Figure 39). Thus, these results did not provide evidence for the reciprocal relationship.

The time lag between body image and eating disturbance as measured at Time 1 and Time 4 was approximately 18 months, and the relationship between body image and eating disturbance may be more immediate. Models 4, 5, and 6 examined the simultaneous effects between the Body Image Factor and the Eating Disturbance Factor at Time 4. The correlation

between the disturbances for the Time 4 factors was removed from these models. Thus, Models 4, 5, and 6 represent reparameterizations of the baseline model and are not nested within the baseline model.

Model 4 tested the empirically supported path from the Body Image Time 4 Factor to the Eating Disturbance Time 4 Factor (see Figure 39). This model showed an acceptable fit with the data (see Table 15). Further, the path from the Body Image Time 4 Factor to the Eating Disturbance Time 4 Factor was significant (see Figure 39). These results indicate that body disparagement predicts eating disturbance. However, it should be noted that this effect has a relatively short time lag.

Model 5 tested the competing simultaneous hypothesis in which eating disturbance predicts body disparagement. This model specified a path from the Eating Disturbance Time 4 Factor to the Body Image Time 4 Factor (see Figure 39). This model showed an acceptable fit with the data (see Table 15), and the path from the Eating Disturbance Time 4 Factor to the Body Image Time 4 Factor was significant (see Figure 39). Thus, support also was provided for the competing model, indicating that eating disturbance leads to body disparagement within a relatively short time lag. However, Hoyle and Panter (1995) noted that simply switching the direction of the path between two constructs may not result in any change in the parameter estimate or the overall fit of the model. Thus, it is difficult to establish directionality.

Model 6 tested the contemporaneous reciprocal relationship between body image and eating disturbance. This model specified a path from the Body Image Time 4 Factor to the Eating Disturbance Time 4 Factor and from the Eating Disturbance Time 4 Factor to the Body Image Time 4 Factor (see Figure 39). Only the path from the Body Image Time 4 Factor to the Eating Disturbance Time 4 Factor was significant (see Figure 39). However, several fit indices indicated

that this model did not demonstrate acceptable fit with the data (see Table 15). Model 6 also was run with the effects constrained to be equal, but the model failed to converge. Thus, Model 4 and Model 5 appear to be better representation of the simultaneous effects between the Body Image Time 4 Factor and the Eating Disturbance Time 4 Factor.

Negative Affect and Eating Disturbance at Time 1 and Time 2

Theory suggests that women use food or the lack of it as a means of providing comfort for negative affective experiences. Consistent with theory, bulimic women reported periods of increased depression, anxiety, and feelings of inadequacy prior to engaging in binge-eating behaviors (Davis et al., 1988; Johnson & Larson, 1982; Steinberg et al., 1990), and negative affect has been shown to predict the onset of bulimia (Killen et al., 1996; Stice & Argas, 1998). However, other studies have reported conflicting results regarding the relationship between negative affect and eating disturbance (Cools, Schotte, & McNally, 1992; Leon et al, 1995). Thus, it is possible that (a) negative affect leads to eating disturbance, (b) eating disturbance results in negative affect, or (c) negative affect and eating disturbance are reciprocally related as in a feedback cycle. To further evaluate these hypothesis, the constructs were examined using prospective data.

Measurement model. Examination of the relationship between Time 1 and Time 2 for the constructs of Negative Affect and the constructs of Eating Disturbance was begun by conducting a confirmatory test of the measurement model. Consistent with the previously presented measurement models for these factors, it was hypothesized a priori that the individual items on the Visual Analog Mood Scale (VAMS) would represent the Negative Affect Factors and the

Bulimia Test-Revised (BULIT-R) and the Eating Attitudes Test-26 (EAT-26) would represent the Eating Disturbance Factors. For the Negative Affect Factors, only the VAMS Sad or Depressed item, the VAMS Happy item, and the VAMS Stressed item for each time loaded appropriately on the corresponding Negative Affect Factor. The remaining VAMS items were removed from the measurement model. The BULIT-R and the EAT-26 loaded appropriately on the Eating Disturbance Factors. At this point, the error variance for the BULIT-R Time 1 was constrained at the lower bound. Following Bollen's (1989) recommendations, the error variance was set to 19.95.

Using the EQS program for structural equation modeling and the Maximum Likelihood (ML) estimation procedure to estimate the parameters of the model, the confirmatory factor analysis (CFA) supported the resulting measurement model, and the overall fit of the data was good (see Table 16). In the measurement model, the VAMS Sad or Depressed items and the VAMS Stressed items loaded positively on the corresponding constructs, and the VAMS Happy item loaded negatively on the corresponding constructs, designating the Negative Affect Factor as representative of negative affect. All measured variables for the Eating Disturbance Factor loaded positively on the corresponding constructs designating the overall constructs as representative of eating disordered symptomatology. All factors were allowed to intercorrelate and the error variance for each measured variable at Time 1 was correlated with the error variance for the same measured variable at Time 2. The measurement model with standardized parameter estimates (factor loadings and error variances) is displayed in Figure 40.

Examination of the relationships among the factors in the measurement model (see Figure 40) revealed positive and significant correlations between the Negative Affect Factor at Time 1 and at Time 2 ($r = .56, p < .05$) and between the Eating Disturbance Factor at Time 1 and at

Time 2 ($r = .83, p < .05$), suggesting that the rank ordering for these constructs was relatively temporally stable, although eating disturbance was highly stable over time, whereas negative affect was only moderately stable over time. The measurement model also showed positive and significant correlations between the within-time constructs ($r = .27, p < .05$ for Time 1; $r = .41, p < .05$ for Time 2) as well as positive and significant cross-time correlations between the Negative Affect Time 1 Factor and the Eating Disturbance Time 2 Factor ($r = .33, p < .05$) and between the Eating Disturbance Time 1 Factor and the Negative Affect Time 2 Factor ($r = .27, p < .05$). The Time 1 measures preceded the Time 2 measures, suggesting temporal precedence in the cross-time correlation. Further, both the within-time correlations and the cross-time correlations between the constructs are significant and the cross-time correlations are generally slightly weaker than the within-time correlations, suggesting that negative affect also may be reciprocally related to eating disturbance. To properly evaluate the relationships among the constructs, it is recommended that a predictor variable should account for variance in a criterion variable while controlling for the temporal stability of the criterion variable (Newcomb, 1994). Thus, a baseline stability model, 3 alternative models representing cross-lagged effects, and 3 models representing simultaneous effects were constructed to further examine the structural relationships between Negative Affect and Eating Disturbance at Time 1 and Time 2. These models are described below and presented in Figure 41.

Structural models. To compare competing models, a baseline stability model was created. Only the paths from Time 1 latent factors to the corresponding Time 2 latent factors were included in this baseline model. In addition, latent factors at Time 1 were correlated and Time 2 disturbances for the latent factors were correlated. The measurement errors for measured

variables at Time 1 were correlated with the corresponding measurement error at Time 2 to prevent the stability paths from being inflated as a result of shared measurement error (Bollen, 1989). The baseline model fit the data well (see Table 16).

The empirically supported path from the Negative Affect Time 1 Factor to the Eating Disturbance Time 2 Factor was specified in Model 1 (see Figure 41). This model showed an acceptable fit to the data, and the chi-square change was significant (see Table 16). Thus, Model 1 represents an improvement over the baseline model. In addition, the specified path from the Negative Affect Time 1 Factor to the Eating Disturbance Time 2 Factor was significant indicating that negative affect predicts eating disturbance over time (see Figure 41).

Testing the competing hypothesis, Model 2 specified a path from the Eating Disturbance Time 1 Factor to the Negative Affect Time 2 Factor (see Figure 41). Again, this model showed an acceptable fit to the data, and the significant chi-square change indicated that Model 2 represented an improvement over the baseline model (see Table 16). Further, the specified path from the Eating Disturbance Time 1 Factor to the Negative Affect Time 2 Factor also was significant, demonstrating support for the competing model in which eating disturbance predicts negative affect over time (see Figure 41).

Model 3 tested an alternate hypothesis in which negative affect and Eating Disturbance are reciprocally related over time. Paths were included from the Negative Affect Time 1 Factor to the Eating Disturbance Time 2 Factor and from the Eating Disturbance Time 1 Factor to the Negative Affect Time 2 Factor (see Figure 41). Model 3 also showed an acceptable fit to the data, and the significant chi-square change indicated a better representation of the data compared to the baseline model (see Table 16). Consistent with Model 1 and Model 2, the specified cross-time paths were also significant (see Figure 41). Further, Model 3 represented a significant

improvement over Model 2 ($\Delta\chi^2 = 5.84$, $df=1$, $p < .05$). Although Model 3 did not exhibit a significant chi-square change over Model 1 ($\Delta\chi^2 = 3.73$, $df=1$, $p > .05$), it demonstrated an improvement in chi-square and other fit indices, suggesting that Model 3 is the best representation of the cross-lagged effects between negative affect and eating disturbance. The results indicate negative affect predicts eating disturbance over time and eating disturbance predicts negative affect over time.

The time lag between negative affect and eating disturbance as measured at Time 1 and Time 2 was approximately 6 months. However, it is possible that the causal time lag is less than 6 months, and the relationship between negative affect and eating disturbance may be more immediate. Compared to the correlations between Eating Disturbance Time 1 and Eating Disturbance Time 2 ($r = .83$), the correlations between Negative Affect Time 1 and Negative Affect Time 2 ($r = .56$) indicate variation in mood over the 6-month time period. A shorter causal time lag or simultaneous analyses may reduce the fluctuations in mood. Models 4, 5, and 6 examined the simultaneous effects between the Negative Affect Factor and the Eating Disturbance Factor at Time 2. The correlation between the disturbances for the Time 2 factors was removed from these models. Models 4, 5, and 6 represent reparameterizations of the baseline model and are not nested within the baseline model.

Model 4 tested the empirically supported path from the Negative Affect Time 2 Factor to the Eating Disturbance Time 2 Factor (see Figure 41). This model showed an acceptable fit with the data (see Table 16). Further, the path from the Negative Affect Time 2 Factor to the Eating Disturbance Time 2 Factor was significant (see Figure 41). These results indicate that negative affect predicts eating disturbance. However, it should be noted that this effect has a relatively short time lag.

Model 5 tested the competing simultaneous hypothesis in which Eating Disturbance predicts negative affect. This model specified a path from the Eating Disturbance Time 2 Factor to the Negative Affect Time 2 Factor (see Figure 41). This model showed an acceptable fit with the data (see Table 16), and the path from the Eating Disturbance Time 2 Factor to the Negative Affect Time 2 Factor was significant (see Figure 41). Thus, support also was provided for the competing model, indicating that eating disturbance leads to negative affect within a relatively short time lag. However, Hoyle and Panter (1995) noted that simply switching the direction of the path between two constructs may not result in any change in the parameter estimate or the overall fit of the model. Thus, it is difficult to establish directionality.

Model 6 tested the contemporaneous reciprocal relationship between negative affect and Eating Disturbance. This model specified a path from the Negative Affect Time 2 Factor to the Eating Disturbance Time 2 Factor and from the Eating Disturbance Time 2 Factor to the Negative Affect Time 2 Factor (see Figure 41). This model demonstrated acceptable fit with the data (see Table 16), and both the paths between the Negative Affect Time 2 Factor and the Eating Disturbance Time 2 Factor were significant (see Figure 41). Model 6 also was run with the effects constrained to be equal, but this resulted in a significant decrement in model fit ($\Delta\chi^2 = 9.14, df=1, p < .01$). Model 6 represents a significant improvement in fit over Model 5 ($\Delta\chi^2 = 9.94, df=1, p < .01$). Although Model 6 did not exhibit a significant chi-square change over Model 4 ($\Delta\chi^2 = 3.56, df=1, p > .05$), it demonstrated an improvement in chi-square and other fit indices, suggesting that Model 6 is the best representation of the simultaneous effects between negative affect and eating disturbance. The results indicate negative affect predicts eating disturbance over time and eating disturbance predicts negative affect over time. These results converge with the results from Model 3 examining the cross lagged effects and indicate a

reciprocal relationship between negative affect and eating disturbance across time as well as within the same time frame.

Negative Affect and Eating Disturbance at Time 2 and Time 4

Measurement model. Examination of the relationship between Time 2 and Time 4 for the constructs of negative affect and the constructs of eating disturbance also was begun by conducting a confirmatory test of the measurement model. Again, it was hypothesized a priori that the individual items on the VAMS would represent the Negative Affect Factors and the BULIT-R and EAT-26 would represent the Eating Disturbance Factors. Only the VAMS Sad or Depressed item, the VAMS Happy item, and the VAMS Stressed item for each time loaded appropriately on the corresponding Negative Affect Factors. The remaining VAMS items were removed from the measurement model. The BULIT-R and the EAT-26 loaded appropriately on the Eating Disturbance Factors. At this point, the error variance for the BULIT-R Time 2 was exhibiting non-significant error variance. Following Bollen's (1989) recommendations, the error variance was set to 22.61.

Using the EQS program for structural equation modeling and the Maximum Likelihood (ML) estimation procedure to estimate the parameters of the model, the confirmatory factor analysis (CFA) supported the resulting measurement model, and the overall fit of the data was good (see Table 16). Again, the VAMS Sad or Depressed items and the VAMS Stressed items loaded positively on the corresponding constructs, and the VAMS Happy item loaded negatively on the corresponding constructs, designating the Negative Affect Factor as representative of negative affect. All measured variables for the Eating Disturbance Factor loaded positively on the corresponding constructs designating the overall constructs as representative of eating

disordered symptomatology. Again, all factors were allowed to intercorrelate and the error variance for each measured variable at Time 2 was correlated with the error variance for the same measured variable at Time 4. The measurement model with standardized parameter estimates (factor loadings and error variances) is displayed in Figure 42.

Examination of the relationships among the factors in the measurement model (see Figure 42) revealed positive and significant correlations between the Internalization of Thin-ideal Factor at Time 2 and at Time 4 ($r = .55, p < .05$) and between the Eating Disturbance Factor at Time 2 and at Time 4 ($r = .78, p < .05$), suggesting that the rank ordering for these constructs was relatively temporally stable, although eating disturbance was highly stable over time, whereas negative affect was only moderately stable over time. The measurement model also showed positive and significant correlations between the within-time constructs ($r = .34, p < .05$ for Time 2; $r = .27, p < .05$ for Time 4) as well as positive and significant cross-time correlations between the Negative Affect Time 2 Factor and the Eating Disturbance Time 4 Factor ($r = .27, p < .16$) and between the Eating Disturbance Time 2 Factor and the Negative Affect Time 4 Factor ($r = .26, p < .05$). Again, the Time 2 measures preceded the Time 4 measures, suggesting temporal precedence in the cross-time correlation. Further, both the within-time correlations and the cross-time correlations between the constructs are significant and the cross-time correlations are generally similar or slightly weaker than the within-time correlations, suggesting that negative affect may also be reciprocally related to eating disturbance. A baseline stability model, 3 alternative models representing cross-lagged effects, and 3 models representing simultaneous effects were also constructed to further examine the structural relationships between negative affect and eating disturbance at Time 2 and Time 4. These models are described below and presented in Figure 43.

Structural models. Only the paths from Time 2 latent factors to the corresponding Time 4 latent factors were included in this baseline model. In addition, latent factors at Time 2 were correlated, Time 4 disturbances for the latent factors were correlated, and measurement errors for measured variables at Time 2 were correlated with the corresponding measurement error at Time 4. The baseline model fit the data well (see Table 16).

The empirically supported path from the Negative Affect Time 2 Factor to the Eating Disturbance Time 4 Factor was specified in Model 1 (see Figure 43). This model showed an acceptable fit to the data, but the chi-square change was non-significant. Thus, Model 1 does not represent an improvement over the baseline model (see Table 16). In addition, the specified path from the Negative Affect Time 2 Factor to the Eating Disturbance Time 4 Factor was not significant, indicating a lack of support for the expected relationship in which negative affect would predict eating disturbance over time (see Figure 43).

Testing the competing hypothesis, Model 2 specified a path from the Eating Disturbance Time 2 Factor to the Negative Affect Time 4 Factor (see Figure 43). Again, this model showed an acceptable fit to the data, but the non-significant chi-square change indicated that the baseline model still better represented the data (see Table 16). Further, the specified path from the Eating Disturbance Time 2 Factor to the Negative Affect Time 4 Factor was not significant, demonstrating a lack of support for the competing model in which eating disturbance would predict negative affect over time (see Figure 43).

Model 3 tested an alternate hypothesis in which negative affect and Eating Disturbance would be reciprocally related between Time 2 and Time 4. Paths were included from the Negative Affect Time 2 Factor to the Eating Disturbance Time 4 Factor and from the Eating Disturbance Time 2 Factor to the Negative Affect Time 4 Factor (see Figure 43). Model 3 also

showed an acceptable fit to the data, but the non-significant chi-square change indicated that the baseline model still better represented the data (see Table 16). Consistent with Model 1 and Model 2, the specified cross-time paths were not significant (see Figure 43). Thus, these results did not provide evidence for the reciprocal relationship.

The time lag between negative affect and eating disturbance as measured at Time 2 and Time 4 was approximately 12 months, and the relationship between negative affect and eating disturbance may be more immediate. Compared to the correlations between Eating Disturbance Time 2 and Eating Disturbance Time 4 ($r = .78$), the correlations between Negative Affect Time 2 and Negative Affect Time 4 ($r = .55$) indicate variation in mood over the 12-month time period. A shorter causal time lag or simultaneous analyses may reduce the fluctuations in mood. However, it is possible that the causal time lag is less than 12 months. Models 4, 5, and 6 examined the simultaneous effects between the Negative Affect Factor and the Eating Disturbance Factor at Time 4. The correlation between the disturbances for the Time 4 factors was removed from these models. Thus, Models 4, 5, and 6 represent reparameterizations of the baseline model and are not nested within the baseline model.

Model 4 tested the empirically supported path from the Negative Affect Time 4 Factor to the Eating Disturbance Time 4 Factor (see Figure 43). This model showed an acceptable fit with the data (see Table 16). However, the path from the Negative Affect Time 4 Factor to the Eating Disturbance Time 4 Factor was not significant (see Figure 43). Thus, these results did not support the expected relationship in which negative affect would predict eating disturbance.

Model 5 tested the competing simultaneous hypothesis in which eating disturbance would predict negative affect. This model specified a path from the Eating Disturbance Time 4 Factor to the Negative Affect Time 4 Factor (see Figure 43). This model showed an acceptable fit with

the data (see Table 16), but the path from the Eating Disturbance Time 4 Factor to the Negative Affect Time 4 Factor was not significant (see Figure 43). Thus, no support was provided for the competing model.

Model 6 tested the contemporaneous reciprocal relationship between negative affect and Eating Disturbance. This model specified a path from the Negative Affect Time 4 Factor to the Eating Disturbance Time 4 Factor and from the Eating Disturbance Time 4 Factor to the Negative Affect Time 4 Factor (see Figure 43). This model demonstrated acceptable fit with the data (see Table 16), but neither of the path between the Eating Disturbance Time 4 Factor to the Negative Affect Time 4 Factor were significant (see Figure 43). These results do not support the hypothesis that negative affect predicts body disparagement and that body disparagement predicts negative affect in return within relatively short time lag.

Negative Affect and Eating Disturbance at Time 1 and Time 4

Measurement model. Examination of the relationship between Time 1 and Time 4 for the constructs of negative affect and the constructs of Eating Disturbance also was begun by conducting a confirmatory test of the measurement model. Again, it was hypothesized a priori that the individual items on the VAMS would represent the Negative Affect Factors and the BULIT-R and EAT-26 would represent the Eating Disturbance Factors. Only the VAMS Sad or Depressed item, the VAMS Happy item, and the VAMS Stressed item for each time loaded appropriately on the corresponding Negative Affect Factors. The remaining VAMS items were removed from the measurement model. The BULIT-R and the EAT-26 loaded appropriately on the Eating Disturbance Factors. At this point, the error variance for the BULIT-R Time 1 was

exhibiting non-significant error variance. Following Bollen's (1989) recommendations, the error variance was set to 19.95.

Using the EQS program for structural equation modeling and the Maximum Likelihood (ML) estimation procedure to estimate the parameters of the model, the confirmatory factor analysis (CFA) supported the resulting measurement model, and the overall fit of the data was good (see Table 16). In the measurement model, the VAMS Sad or Depressed items and the VAMS Stressed items loaded positively on the corresponding constructs, and the VAMS Happy item loaded negatively on the corresponding constructs, designating the Negative Affect Factor as representative of negative affect. All measured variables for the Eating Disturbance Factor loaded positively on the corresponding constructs designating the overall constructs as representative of eating disordered symptomatology. Again, all factors were allowed to intercorrelate and the error variance for each measured variable at Time 1 was correlated with the error variance for the same measured variable at Time 4. The measurement model with standardized parameter estimates (factor loadings and error variances) is displayed in Figure 44.

Examination of the relationships among the factors in the measurement model (see Figure 44) revealed positive and significant correlations between the Negative Affect Factor at Time 1 and at Time 4 ($r = .48, p < .05$) and between the Eating Disturbance Factor at Time 1 and at Time 4 ($r = .75, p < .05$), suggesting that the rank ordering for these constructs was relatively temporally stable, although eating disturbance was highly stable over time, whereas negative affect was only moderately stable over time. The measurement model also showed negative and significant correlations between the within-time constructs ($r = .27, p < .05$ for Time 1; $r = .28, p < .05$ for Time 4) as well as negative and significant cross-time correlations between the Negative Affect Time 1 Factor and the Eating Disturbance Time 4 Factor ($r = .32, p < .05$) and

between the Eating Disturbance Time 1 Factor and the Negative Affect Time 4 Factor ($r = .25, p < .05$). Again, the Time 1 measures preceded the Time 4 measures, suggesting temporal precedence in the cross-time correlation. Further, both the within-time correlations and the cross-time correlations between the constructs are significant and the cross-time correlations are generally similar than the within-time correlations, suggesting that internalization of thin-ideal may also be reciprocally related to Eating Disturbance. A baseline stability model, 3 alternative models representing cross-lagged effects, and 3 models representing simultaneous effects were also constructed to further examine the structural relationships between internalization of thin-ideal and Eating Disturbance at Time 1 and Time 4. These models are described below and presented in Figure 45.

Structural models. Only the paths from Time 1 latent factors to the corresponding Time 4 latent factors were included in this baseline model. In addition, latent factors at Time 1 were correlated, Time 4 disturbances for the latent factors were correlated, and measurement errors for measured variables at Time 1 were correlated with the corresponding measurement error at Time 4. The baseline model fit the data well (see Table 16).

The empirically supported path from the Negative Affect Time 1 Factor to the Eating Disturbance Time 4 Factor was specified in Model 1(see Figure 45). This model showed an acceptable fit to the data, and the significant chi-square change indicated that this model fit the data better than the baseline model (see Table 16). Additionally, the specified path from the Negative Affect Time 1 Factor to the Eating Disturbance Time 4 Factor was significant indicating that negative affect predicts eating disturbance over time (see Figure 45).

Testing the competing hypothesis, Model 2 specified a path from the Eating Disturbance Time 1 Factor to the Negative Affect Time 4 Factor (see Figure 45). Again, this model showed an acceptable fit to the data, but the non-significant chi-square change indicated that the baseline model still better represented the data (see Table 16). Further, the specified path from the Eating Disturbance Time 1 Factor to the Negative Affect Time 4 Factor was not significant, demonstrating a lack of support for the competing model in which eating disturbance would predict negative affect over time (see Figure 45).

Model 3 tested an alternate hypothesis in which negative affect and Eating Disturbance would be reciprocally related between Time 1 and Time 4. Paths were included from the Negative Affect Time 1 Factor to the Eating Disturbance Time 4 Factor and from the Eating Disturbance Time 1 Factor to the Negative Affect Time 4 Factor (see Figure 45). Model 3 also showed an acceptable fit to the data (see Table 16). Consistent with Model 1 and Model 2, the path from the Negative Affect Time 1 Factor to the Eating Disturbance Time 4 Factor was significant, but the path from the Eating Disturbance Time 1 Factor to the Negative Affect Time 4 Factor was not significant (see Figure 45). Model 3 did represent a significant improvement over the Baseline Model (see Table 16) and over Model 2 ($\Delta\chi^2 = 6.30, df=1, p < .05$), but not over Model 1 ($\Delta\chi^2 = 3.18, df=1, p > .05$). Although this model indicates that negative affect predicts eating disturbance over time, Model 3 is not a meaningful improvement over the baseline model, and the baseline model would be preferred over Model 3 for the sake of parsimony. Thus, Model 1 appears to be the best representation of the cross-lagged effects between negative affect and Eating Disturbance.

The time lag between negative affect and Eating Disturbance as measured at Time 1 and Time 4 was approximately 18 months. However, it is possible that the causal time lag is less than

18 months, and the relationship between negative affect and eating disturbance may be more immediate. Compared to the correlations between Eating Disturbance Time 1 and Eating Disturbance Time 4 ($r = .75$), the correlations between Negative Affect Time 1 and Negative Affect Time 4 ($r = .48$) indicate variation in mood over the 18-month time period. A shorter causal time lag or simultaneous analyses may reduce the fluctuations in mood. Thus, Models 4, 5, and 6 examined the simultaneous effects between the Negative Affect Factor and the Eating Disturbance Factor at Time 4. The correlation between the disturbances for the Time 4 factors was removed from these models. Thus, Models 4, 5, and 6 represent reparameterizations of the baseline model and are not nested within the baseline model.

Model 4 tested the empirically supported path from the Negative Affect Time 4 Factor to the Eating Disturbance Time 4 Factor (see Figure 45). This model showed an acceptable fit with the data (see Table 16). Further, the path from the Negative Affect Time 4 Factor to the Eating Disturbance Time 4 Factor was significant (see Figure 45). These results indicate that negative affect predicts eating disturbance. However, it should be noted that this effect has a relatively short time lag.

Model 5 tested the competing simultaneous hypothesis in which eating disturbance predicts negative affect. This model specified a path from the Eating Disturbance Time 4 Factor to the Negative Affect Time 4 Factor (see Figure 45). This model showed an acceptable fit with the data (see Table 16), and the path from the Eating Disturbance Time 4 Factor to the Negative Affect Time 4 Factor was significant (see Figure 45). Thus, support also was provided for the competing model, indicating that eating disturbance leads to negative affect within a relatively short time lag. However, Hoyle and Panter (1995) noted that simply switching the direction of

the path between two constructs may not result in any change in the parameter estimate or the overall fit of the model. Thus, it is difficult to establish directionality.

Model 6 tested the contemporaneous reciprocal relationship between negative affect and Eating Disturbance. This model specified a path from the Negative Affect Time 4 Factor to the Eating Disturbance Time 4 Factor and from the Eating Disturbance Time 4 Factor to the Negative Affect Time 4 Factor (see Figure 45). This model demonstrated acceptable fit with the data (see Table 16), but neither path between the Negative Affect Time 4 Factor to the Eating Disturbance Time 4 Factor was significant (see Figure 45). Model 6 also was run with the effects constrained to be equal, but this resulted in a non-significant decrement in model fit ($\Delta\chi^2 = 1.65$, $df=1$, $p > .05$). Further, Model 6 did not represent a meaningful improvement in fit over Model 4 ($\Delta\chi^2 = 1.7$, $df=1$, $p > .05$) or over Model 5 ($\Delta\chi^2 = 2.31$, $df=1$, $p > .001$). Thus, Model 4 and Model 5 are better representation of the simultaneous relationship between negative affect and eating disturbance. This indicates that simultaneous effects are present within a short time lag, but the effects are not reciprocal.

In summary, cross-lagged and simultaneous effects were examined for the following paired constructs: negative affect and body image, internalization of the thin-ideal and body image, internalization of the thin-ideal and eating disturbance, body image and eating disturbance, and negative affect and eating disturbance. Each pair was examined at Time 1 and Time 2, at Time 2 and Time 4, and at Time 1 and Time 4. The findings from the tests of these models provided additional information about the predictive relationships among these factors.

CHAPTER 4

DISCUSSION

Previous research has identified several important areas in the etiology of eating disorders, including attachment (Ward et al., 2000), experienced stressors (Crisp et al., 1980; Kay & Leigh, 1954; Lacey et al., 1986; Morgan & Russell, 1975; Pyle et al., 1981), the appraisal/coping process (Lehman & Rodin, 1989; Leon et al., 1993; Soukoup et al., 1990), psychological distress/negative affect (Bulik et al., 1992; Davis et al., 1988; Dykens & Gerard, 1986; Johnson & Larson, 1982; Killen et al., 1996; Mintz & Betz, 1988; Parmer, 1991; Shisslak et al., 1990; Steinberg et al., 1990; Stice, 2001a; Stice & Argas, 1998; Stice & Shaw, 1994; Vitousek & Manke, 1994; Weiss & Ebert, 1983), body image disturbances (Attie & Brooks-Gunn, 1989; Leon et al., 1993; Mintz & Betz, 1988, Stice, 1994; Striegel-Moore et al., 1988), and internalization of sociocultural beliefs regarding the thin-ideal (Griffiths, Beaumont, et al., 1999; Griffiths, Mallia-Blanco, et al., 2000; Stice & Shaw, 1994; Stice, Mazotti, et al., 2000; Stice, Chase, et al., 2001). The majority of studies on eating pathology focused on single etiological factors, assuming a direct link with the onset of eating disordered symptomatology (O’Kearney, 1996; Stice, 2001b). Although such approaches are useful in identifying individual predisposing factors, they are lacking in that they do not address the complexity of eating disorder development. Multidimensional models, however, address the complexity of eating disorder development. Unfortunately, many of these models have not been integrative, thus ignoring the process in which the factors influence each other and culminate in eating pathology (Garfinkel & Garner, 1982; Stice, 2001b). In this study, I examined not only the multiple factors that may play a role in eating disorder development, but also evaluated a model that hypothesized direct and indirect effects between the multiple precipitating and maintaining

etiological factors. Further, a review of the current literature yielded minimal empirical research using longitudinal data to examine integrative models of eating disorder development (e.g., Ghaderi, 2003; Stice, 1998b; Stice et al., 1996; Thompson et al., 1995) and called for more studies that investigate multidimensional risk factors for the development of eating disorders using longitudinal data (Ghaderi, 2003). This study addressed this need by using prospective longitudinal data.

Several researchers have suggested that college is an environmental risk factor for the development of eating pathology (Compas et al., 1986). The separation associated with leaving home and beginning college is typically viewed as a developmental stressor (Smolak & Levine, 1996) and a naturally occurring “strange situation” that activates the attachment behavioral system (Ainsworth et al., 1987; Bloom, 1980; Kaplan, 1981; Kenny, 1987). Due to the increased demands associated with this transition, it is a time when many individuals are vulnerable to developing psychopathology (Bloom, 1987). Furthermore, eating disturbances primarily affect females and the onset of the disorder usually occurs in late adolescence or early adulthood (APA, 2000; Kaplan & Sadock, 1998). Thus, this study examined freshman women navigating the developmental demands associated with the transition from high school to college in order to provide clarification about the development of eating disorders during this critical time period. These freshman women were then followed over an 18-month period (first two years in school) to examine the ongoing relationships among factors hypothesized to contribute to the development of eating pathology.

Description of Participants

The results of this study are based on an initial sample of female freshmen that matched the larger population of female freshman at the two participating schools in terms of age, race/ethnicity, marital status, living situation, and course load (OU, 2003; UNT, 2002). In addition, the distributions of the current sample's variables are comparable to those of previous studies conducted at similar universities (Tripp, 2002; Tripp & Petrie, 2001), and all demographic variables were within expected ranges. Despite participant attrition and expected changes (i.e., marital status, living situation, course load), all demographic variables remained within expected ranges over the course of the study, and they were consistent with the demographic variables of the larger population of female undergraduate students at both of the participating schools. Further, the women who dropped out of the study were similar to the women who completed the study.

The majority of the women who participated in this study (62.2% at Time 1; 61.3% at Time 2; 59.1% at Time 4) were normal weight (Center for Disease Control, 2001), which is consistent with the results from recent studies using college women (Tripp & Petrie, 2001; Tripp, 2002). Although the results indicate the majority of the women were average in terms of physical size, a significant number of women were also underweight (16.8% at Time 1; 14.9% at Time 2; 13.9% at Time 4), and nearly 25% of the women at each time (26.9% at Time 1; 24.3% at Time 2; 22.4% at Time 4) desired a BMI that would place them in the underweight category. These results suggest that an average weight is not always acceptable and may be viewed as "being overweight" to female undergraduates.

Further, more than 10% of the women exceeded the cutoff score (≥ 20) on the EAT as measured at each point in time (11.3% at Time 1; 10.1% at Time 2; 10.9% at Time 4), suggesting

disturbances in eating attitudes and behaviors. The distribution of women whose BULIT-R scores exceeded the cutoff (≥ 104) fluctuated a little more over time (5% at Time 1; 1.3% at Time 2; and 4.4% at Time 4), but still suggested that many women were experiencing diagnosable symptoms of bulimia nervosa. These prevalence rates are similar to those found by other researchers investigating eating disordered behaviors in college women using the EAT and the BULIT-R (Furnham & Husain, 1999; Thelen et al., 1991) and consistent with prevalence estimates for bulimia in women (APA, 2000). Although, the majority of the women do not meet the criteria for the diagnosis of an eating disorder, female college students are experiencing diagnosable symptom levels, with even high numbers reporting some degree of eating disordered behavior (Hart & Kenny, 1997).

In fact, in the current sample, large percentages of the participants reported engaging in behaviors associated with eating disorder symptomatology. For example, 12.6% to 23.1% of the women reported exercising more than one hour a day to burn calories, and over 41% of the women endorsed dieting as a means by which to control their weight; fewer, though, reported using diuretics or laxatives (4%) and vomiting (7%). Further, a large number of the participants (49.6% at Time 1; 41.6% at Time 2; 35.7% at Time 4) reported engaging in binge-eating behaviors at least 2-3 times a month. A recent study reported that 80% of women dieted and 50% of women engaged in binge-eating behaviors during their first year in college (Striegel-Moore, Silberstein, Grunberg, & Rodin, 1990). Kaplan and Sadock (1998) estimated that approximately 5% of women report subthreshold symptoms of anorexia, and up to 40% of college women exhibit occasional symptoms of bulimia.

Although exact prevalence rates vary, many researchers have noted that alarming numbers of college women engage in some type of eating disordered behavior (Heatherton,

Mahamedi, Striepe, Field, & Keel, 1997; Mintz & Betz, 1988). Although the majority of the women that participated in this study are not exhibiting diagnosable eating disorders, nearly half of them are engaging in some type of disturbed eating behavior that could place them at risk for developing a diagnosable eating disorder or other serious health concerns. Further examination of the changes in behaviors over time reveals decreases in the number of participants who endorsed dieting to lose weight, exercising to burn calories, and rapidly eating large amounts of food. Unfortunately, there was a slight increase in the number of participants who endorsed intentionally vomiting to control weight, and the number of participants who endorsed using laxatives and diuretics remained the same. This calls attention to the seriousness of eating concerns and destructive eating and weight management behaviors that occur on college campuses.

Regarding participants' transition to college, many of the women experienced some level of difficulty adjusting to the changes and increased demands associated with leaving high school and attending college for the first time. One-third to nearly 60% of the participants appraised the transition to college and the new demands they encountered, especially the increased academic demands, as being stressful. In addition, 20-40% of the women reported difficulty coping with the transition. These results are consistent with previous research investigating the general stress and coping with perceived demands in both male and female college freshman (Arthur, 1998). Regarding the current study's participants' mood state at Time 1, nearly 50% reported feeling sad or depressed, and more than 75% indicated feeling stressed and anxious, which is somewhat higher than previous studies investigating negative affect in college women (Stice & Shaw, 1994), and suggests that the women who participated in this study appeared to be experiencing high levels of emotional distress. A primary reason for these differences in negative affect is that

the sample used in Stice and Shaw's investigation included all classes of undergraduate women. Given that the transition to college is viewed as a period of developmental changes and increasing demands (Kenny & Rice, 1995), it is not surprising that freshmen women would report more sadness, less happiness, and more stress than sophomore, junior, and senior women. Freshman women attending college for the first time are in the process of individuating from parents, managing increased academic demands, forming new relationships, and building new support networks. Many also are adapting to new living arrangements and added responsibilities. More advanced students have likely acclimated to the demands of the college environment and developed support systems, both emotional and tangible, to cope more successfully. It is also possible that the elevated levels of emotional distress are the result of the time in the semester during which the data was collected. Data was collected between the 8th and the 12th weeks of the first semester, the time frame during which many students were taking or had just taken their first set of midterm exams. Thus, the additional pressures associated with preparing and taking midterm exams also may have contributed to the increased report of emotional distress.

Analyses of Models with All Measured Factors

In each of the initial models that was proposed (Model A and Model B), the causal relationships among all of the constructs as measured at Time 1, Time 2, and Time 4—parental attachment, history of stress, appraisal/coping, negative affect, internalization of the thin-ideal, body image, and eating disturbance—were simultaneously examined (see Figures 2 and 3). However, these models failed to converge and produce a solution, and attempts to evaluate portions of the specified models also exhibited significant problems indicative of poor model fit. The current sample size is not sufficiently large to test the specified models. However, it is also

possible that the actual time lags are different than the measured time lags. Since the specified paths are consistent with both empirical and theoretical support, it is likely that the failure to converge and produce a solution is due to insufficient sample size and timing of measurement rather than gross misspecification of parameters in the models.

Analysis of Models with Selected Factors

Following the outline of the proposed models, specific factors were selected from each point in time, and the models were retested. These new models are also consistent with Bradford's (2004) work. However, some of the selected factors in these models were measured at the same points in time, so the ability to make predictive inferences among all of the factors is reduced in these models. Reducing the number of factors also reduces the number of parameters being estimated in each model. Thus, these models are more appropriate for the sample size in the study.

Structural Model A.

In the first model, the Parental Attachment Factor and the History of Stress Factor were hypothesized to have a direct effect on the Appraisal/Coping Factor, and the Appraisal/Coping Factor was expected to have a direct influence on the Negative Affect Factor. Both the Negative Affect Factor and the Internalization of the Thin-Ideal Factor were hypothesized to have a direct effect on the Body Image factor, which in turn, was hypothesized to directly influence the Eating Disturbance Factor. Finally, the Internalization of the Thin-Ideal Factor was hypothesized to have a direct effect on the Eating Disturbance Factor, in addition to this path being mediated by the Body Image Factor (see Figure 8 and Figure 12).

The paths in Model A were evaluated using selected factors from Time 1 and Time 2 (see Figure 8), and they were again evaluated using selected factors from Time 1 and Time 4 (see Figure 12), simply substituting the selected factors from Time 2 with the factors from Time 4. Although the exact parameter estimates do differ in the Model and are discussed at length in the previous chapter, the general findings for the model with the selected factors at each point in time are the same.

Structural analyses supported the hypothesized path from the Parental Attachment Factor to the Appraisal Coping Factor. Although data measuring the Parental Attachment Factor and data measuring the Appraisal/Coping factor was gathered at the same time, the measures of parental attachment were a reflection of experiences from the participant's first 16 years, and the measures of the appraisal/coping process reflected the participant's experiences since beginning their first semester of college. Thus, the direct and positive impact of parental attachment on the appraisal/coping process indicates that women with insecure attachments to their parents will experience the transition from high school to college as being more stressful, and they will feel less able to cope with the demands of the situation. These results are consistent with McCarthy, Moller, and Fouladi's (2001) study in which individuals who reported less parental care and more parental overprotection (insecure attachment) also reported higher levels of perceived stress and less confidence in their ability to regulate their negative mood and cope with their feelings. Because parental relationships provide a base from which individuals explore their surroundings (Hartman, 1979), individuals with insecure parental attachments develop internal working models in which the self is viewed as unworthy and incompetent, and/or others are seen as irresponsible and undependable (Ainsworth et al., 1978). From this insecure base, individuals cannot comfortably explore their environment and develop social competence and coping skills

(Kenny, 2000). Consequently, when they begin the transition to college and the separation-individuation process from their parents, they appraise the situation and demands as being more stressful and feel less capable in their ability to cope with the changes.

Along with the Parental Attachment Factor, the History of Stress Factor also had a direct and positive impact on the Appraisal/Coping Factor. Again, data measuring the History of Stress Factor and data measuring the Appraisal/Coping factor were gathered at the same time, the measures of past stressful experiences were a reflection of experiences from the participant's last 6 months, and the measures of the appraisal/coping process reflected the participant's experiences since beginning their first semester of college. Thus, reporting experiencing more negative events in the previous six months predicted experiencing the transition to college as more stressful and feeling less able to cope with the demands of the college environment. These results confirm earlier findings in which increased incidences of prior stressful occurrences were directly and positively related to the stressful appraisal of life events and decreased ability to cope with the events (Shatford & Evans, 1996; Tripp, 2002). Less research has been done regarding the association between negative life events and the transition to college, but researchers have hypothesized that prior experiences with stressful events play a key role in an individual's reaction to subsequent stressors (Dohrenwend & Dohrenwend, 1978). Multiple changes occur during the transition from high school to college, and many individuals probably appraise some level of stress and inability to cope during this time. Women, in particular, also find the separation-individuation process confusing and frustrating due to the mixed messages they receive regarding family needs to maintain connectivity and pressures to develop autonomy (Smolak & Levine, 1996; Strober & Humphrey, 1987), leading to a greater likelihood that these individuals experience some level of stress and difficulty with the transition to college. Women

who have also experienced additional negative events during the previous six months may still be managing those experiences, and if so, may have even fewer psychological, and perhaps tangible, resources available to manage the new demands associated with the transition. Thus, they perceive an even greater level of stress and inability to cope with more immediate events.

A direct and positive path from the Appraisal/Coping factor to the Negative Affect Factor was supported as hypothesized; measurement of the Appraisal/Coping Factor preceded measurement of the Negative Affect Factor Time 2 by 6 months and the Negative Affect Factor Time 4 by 18 months. Thus, this path indicates that appraising the transition to college as more stressful and reporting feeling less confident in the ability to manage the demands associated with the transition predicts increased feelings of sadness or depression and stress, and decreased feelings of happiness. Entering college is closely associated with new academic and social demands, role and identity changes, and high levels of stress (Compas et al., 1986; Rosen, Compas, & Tacy, 1993; Striegel-Moore et al., 1990), and individuals who experience difficulties may feel less confident and have less self-esteem, leaving them vulnerable to developing physical and psychological health problems (Lloyd, 1980; Rodin & Salovey, 1989; Tripp, 2002). Thus, it is not a surprise that individuals who appraised the transition as being more stressful and more difficult to manage would develop later feelings of being more depressed, less happy, and more stressed.

Although the models support an indirect relationship between the appraisal/coping process and the development of eating disorder symptomatology as mediated by negative affect and body disparagement, the models also supported a direct and positive path from the Appraisal/Coping Factor to the Eating Disturbance Factor. Again, measurement of the Appraisal/Coping Factor preceded measurement of the Eating Disturbance Factor Time 2 by 6

months and the Eating Disturbance Factor Time 4 by 18 months. Thus, appraising the transition to college as more stressful and reporting feeling less confident in the ability to manage the demands associated with the transition predicts increased eating disordered attitudes and behaviors. However, this study hypothesized that difficulty with the transition to college would directly lead to the experience of negative affect, which would mediate the relationship between the appraisal coping process and eating disturbance. Although this relationship was significant, the addition of the direct path from the Appraisal/Coping Factor to the Eating Disturbance Factor suggests that an individual's experience with the transition process may influence the development of eating disorder symptoms in more than one way. Although it is possible that there is a direct relationship between the appraisal/coping process and the development of eating disturbances, it also is likely that other mediating factors not assessed in this study could account for the addition of this path. One possible explanation is that individuals who appraise the transition as more stressful and feel less able to cope engage in the use of impaired coping strategies that prevent them from effectively managing life events and stress, leading to the development of eating disturbances. Research has shown that bulimic women use more emotion-focused coping and avoidance-focused coping responses compared to controls (Neckowitz & Morrison, 1991; Shatford & Evans, 1986; Soukoup et al., 1990; Troop et al., 1994). In addition, Katzman and Wolchik (1984) and Johnson and Larson (1982) reported that bulimic and binge-eating women displayed a passive coping style and an inability to express their feelings, characteristics associated with poor adjustment (Billings & Moos, 1981). Future research investigating coping styles in relation to the transition to college and the development of eating disorders may provide more information about this relationship to determine whether coping

style mediates the relationship between appraisal and the subsequent development of disordered eating.

Negative affect and internalization of the thin-ideal regarding beauty were hypothesized to influence body image. As data measuring these constructs were gathered at the same point in time, it is more difficult to make casual statements about the relationships among these constructs. However, the current sample supported a direct pathway in from the Negative Affect Factor to the Body Image Factor, confirming that feelings of sadness, unhappiness, and stress were associated with an individual's perception of their body and validating previous research in which feelings of psychological distress were shown to contribute to body disparagement (Bradford, 2004; Kearney-Cooke & Striegel-Moore, 1994; Tripp, 2002; Tripp & Petrie, 2001). These results contradict Stice, Nemeroff, et al.'s (1996) dual pathway model which posits that both negative affect and dietary restraint mediate the relationship between body dissatisfaction and eating pathology. Instead the current results converge with work by Bradford (2004) and Tripp (2002) who also demonstrated that body dissatisfaction mediates the relationship between negative affect and eating disturbance. However, when the causal relationships between these two constructs were later examined, the cross-lagged models did not reveal any significant findings.

Furthermore the current sample also supported the direct relationship in Model A from the Internalization of the Thin-Ideal Factor to the Body Image Factor, indicating that women who acknowledged awareness and internalization of societal ideals possess more negative body image. When the causal relationship between these two constructs was later examined using cross-lagged models, the findings indicated a reciprocal relationship between the constructs at Time 1 and Time 2; however, the findings then revealed that Time 2 internalization of the thin-

ideal predicted body dissatisfaction at Time 4, and Time 1 internalization of the thin-ideal also predicted body dissatisfaction at Time 4. Research has suggested that messages from parental, peer and media sources impact the development of eating disordered attitudes and behaviors (Smolak & Levine, 1996; Stice, 1998). With social comparison playing a significant role in an individual's self-perception (Levine & Smolak, 1998), and the pervasive presence of messages from media, peers and family promoting unrealistic ideals for women and body types that require drastic means to obtain and maintain, many women find that they do not possess the "ideal body." Women who internalize this thin-ideal may agonize over their current appearance and feel dissatisfied with their bodies (Levine & Smolak, 1998; Smolak & Levine, 1996). Women who are already experiencing psychological distress as a result of difficulty adjusting to and coping with the increased demands of transitioning from high school to college likely experience additional stress as a result of the discrepancy between the internalized beauty ideal and their own reality making them even more vulnerable to developing body disparagement and subsequent eating pathology.

In a meta-analytic review of risk factors for eating pathology, Stice (2002) noted that body disparagement "emerged as one of the most consistent and robust risk factors" for the development of eating disturbances. The current sample supported the hypothesized pathway from the Body Image Factor to the Eating Disturbance Factor, confirming that women who reported greater levels of body disparagement also endorsed more eating disordered attitudes and behaviors. The Body Image Factor Time 2 alone explained 19.10% of the variance in the Eating Disturbance Factor Time 2, and the Body Image Factor Time 4 alone explained 19.90% of the variance in the Eating Disturbance Factor Time 4, lending further validation to Stice's conclusion and previous research in which body disparagement has been shown to predict

increases in dieting, onset of bulimic symptoms, and eating pathology (Stice, 2001a; Stice & Argas, 1998; Wertheim, Koerner, & Paxton, 2001). However, when the causal relationships between these two constructs were later examined, the cross-lagged models did not reveal any significant findings.

The strong relationship between body disparagement and eating disordered behaviors also suggests that women who are unhappy with their current physical appearance may resort to drastic measures such as dieting, excessive exercise, laxative use, vomiting, and fasting in order to achieve a more desirable appearance. Women are socialized to believe that thinness is equated with beauty (Silverstein & Perdue, 1988). Thus, many women who experience dissatisfaction with their body may believe that weight loss through dieting and exercise is the solution. Unfortunately, social reinforcement for success from initial dieting may result in continued dieting behaviors, leading to the development of anorexia. Alternatively, initial dieting behaviors may promote the development of bingeing and purging behaviors because dieting entails rules that may be hard to follow, resulting in the need to employ immediate behaviors to compensate for violation of the dieting rules (Stice & Shaw, 2002). Further, the diagnostic criteria highlight disturbed perception of body weight and shape as a primary symptom of both anorexia and bulimia. Thus, it is clear that body image is an important factor in the development of eating disorders (APA, 2000).

Twamley and Davis (1999) noted that perceived control over weight and shape mediated a relationship between body disparagement and eating pathology. Consistent with this idea, Polivy and Herman (2002) suggested that the media has led women to believe that dieting, exercising, and other techniques to control weight will enable them to achieve this portrayed “ideal thinness.” Research also has seen an increase in weight loss related advertisements (Snow

& Harris, 1986), an increase in the number of articles on dieting and exercise programming in popular women's magazines (Garner et al., 1980), and bulimic women have reported learning to binge and purge as methods of weight control via media sources (Chiodo & Latimer, 1983; Fairburn & Cooper, 1982). Thus, with pervasive messages encouraging the use of a variety of weight control methods and claiming success from these methods, it is likely that many women do believe they can change the size and shape of their body, and they are then more likely to engage in disturbed eating and weight control behaviors. Further, Twamley and Davis suggested that self-esteem moderates the relationship between body disparagement and eating disordered behaviors. Their study of undergraduate women indicated body disparagement in women with lower levels of self-esteem had a larger effect on disturbed eating behavior, suggesting that women who experience body disparagement in addition to a general feeling of negativity about themselves are less likely to dismiss negative thoughts about their body and more likely to engage in harmful methods to change their body. These women likely see few strengths in themselves compared to women who have higher levels of self-esteem. Thus, they may have little confidence in their abilities to change their body through healthy methods and may feel a greater pressure to change their body. Future research investigating self-esteem and control beliefs may provide additional information about how these variables moderate the relationship between body disparagement and eating pathology.

The structural analysis also supported a direct pathway in Model A from the Internalization of the Thin-Ideal Factor to the Eating Disturbance Factor, indicating that women who endorsed awareness and internalization of the thin-ideal reported more eating disordered attitudes and behaviors. Cross-lagged analysis also revealed that Time 2 internalization of the thin-ideal predicted eating disturbance at Time 4. Researchers found that internalization of the

thin-ideal predicted onset of bulimic pathology (Stice & Argas, 1998) and dieting behaviors (Stice, 2001a). Furthermore, experimental reduction of the internalization of the thin-ideal resulted in a decrease in dieting behaviors and reduced symptoms of bulimia (Stice, Mazotti, et al., 2000). Thus, it appears that there is a direct relationship between internalization of the thin-ideal and eating disturbance as well as an indirect influence mediated by body image. This direct pathway highlights the influence sociocultural pressures have on the development of eating disorders. Again, it is possible that this relationship is mediated and/or moderated by other factors in addition to body image. However, previous research has only noted that body image, negative affect, and dieting behaviors play a role in mediating the relationship between internalization and the development of eating disorders (Stice & Argas, 1998; Stice & Shaw, 1994; Thompson et al., 1999). In this study, analyses did not support a path from internalization to negative affect, but the negative affect factor only reflected the feelings of sadness or depression, unhappiness, and stressed. Perhaps a different representation of the negative affect construct, utilizing a symptom rating scale to assess psychological distress (i.e., anxiety and depression) rather than a subjective rating would yield different results. Alternatively, it may be that negative affect needs to be separated into two factors. Given that the results of this study support a pathway in which negative affect, as measured by reports of sadness or depression, unhappiness, and stress, mediates the relationship between appraisal/coping and body image, and Stice's work (Stice, 2001a; Stice & Argas, 1998) supports a pathway in which negative affect (depression, shame, guilt, decreased confidence, and decreased happiness) mediates a relationship between body disparagement and bulimic pathology, research would support the placement of the Negative Affect Factor in two different places in a model in relation to the prediction of eating disorders. Thus, it is possible that appraisal of stress and difficulty coping

with the transition to college results in an initial experience of negative affect (feeling sad or depressed, unhappy, and stressed) that, when combined with an internalization of the thin-ideal, leads to body disparagement and subsequent eating disturbances. This was supported in Model A. Body disparagement also may result in a second experience of psychological distress (depression, shame, guilt, less confidence, and less happiness) that leads to eating disturbances, as supported by Stice's dual-pathway model. Testing these new relationships among the factors may clarify the role psychological distress has in the development of eating pathology. In this model, both dieting behaviors and bulimic symptoms were conceptualized to be part of the eating disturbances factor. However, the examination of the role dieting behaviors play in mediating the relationship between internalization and eating disturbance might also be meaningful.

Structural Model B

Following the outline of the originally hypothesized Model and Bradford's (2004) work, a second model was also tested. The relationships among the Parental Attachment Factor, History of Stress Factor, Appraisal/Coping Factor, and Negative Affect Factor from Model A were hypothesized to remain the same. In Model B, separate paths from both the Psychological Distress Factor and the Body Image Factor were hypothesized to directly influence the Eating Disturbance Factor. In addition, pathways from the Internalization of Sociocultural Beliefs Factor were hypothesized to have a direct effect on the Body Image Factor and on the Eating Disturbances Factor (see Figure 10 and Figure 14). With Model B, the paths also were evaluated using selected paths from Time 1 and Time 2 (see Figure 10), and they were again evaluated using selected paths from Time 1 and Time 4 (see Figure 14), simply substituting the selected

paths from Time 2 with the paths from Time 4. Recall, the model using selected paths from Time 1 and Time 2 required the addition and deletion of specified paths, resulting in the same model specified in Model A. Thus, with the selected paths at Time 1 and Time 2, Model B was not an equivalent model of the data. The resulting model for these time periods is discussed above, so only the selected paths from Time 1 and Time 4 are discussed below.

The same relationships among parental attachment, history of stressful experiences, appraisal/coping, and negative affect that were discussed in the previous model (Model A) were supported in this model. Thus, Model B also supported the hypotheses that both insecure parental attachment and a history of stressful experiences predicts experiencing the transition to college as more stressful and feeling less able to cope with the demands of the college environment, which in turn leads to increased feelings of sadness or depression, decreased feelings of happiness and feeling more stressed. As in Model A, the direct pathways from the Internalization of the Thin-Ideal Factor to the Body Image Factor, from the Internalization of the Thin-Ideal Factor to the Eating Disturbance Factor, and from the Body Image Factor to the Eating Disturbance Factor were again supported, indicating that there is a direct relationship between internalization of the thin-ideal and eating disordered attitudes and behaviors as well as an indirect influence mediated by body image. Further, the new pathway from the Negative Affect Factor to the Eating Disturbance Factor was supported in this model, confirming that women who endorsed greater feelings of stress and anxiety were likely to report increased eating disordered attitudes and behaviors, and adding further validation to research citing a relationship between negative affect and eating disordered attitudes and behaviors (Stice, 2001b). Cross-lagged analyses revealed a reciprocal relationship between negative affect and eating disturbance

at Time 1 and Time 2; however the findings also revealed that Time 1 negative affect predicted Time 4 eating disturbance.

Summary of Findings

The relationships among factors that contribute to the development of eating disorders are complex. Statistical analyses of the constructs evaluated in the above models (parental attachment, history of stress, appraisal/coping, negative affect, internalization of the thin-ideal, body image, and eating disturbance) indicated that parental attachment and a history of stressful events have a strong impact on an individual's appraisal, coping, and adjustment to key events during the transition from high school to college. The results also suggested the importance sociocultural pressures, psychological distress, and body disparagement have as risk factors in the development of eating disordered symptomatology.

Specifically, women who reported insecure attachments to their parents also felt less capable of their ability to cope with the transition to and the new demands of college. Perceived greater stress and less ability to cope with the transition to and demands of college predicted negative affect (e.g., feeling sad or depressed, unhappy, and stressed) as well as eating disorder symptomatology at 6-month and 18-month follow-up. Within the same time period, increased negative affect and greater internalization of societal pressures to achieve the thin-ideal body type were related to increased body disparagement. Further, women who endorsed greater internalization of the thin-ideal and who reported decreased satisfaction with their body endorsed more disordered eating attitudes and behaviors.

These models are consistent with Tripp's (2003) and Bradford's (2004) evaluation of the relationships among the stress-coping process, negative affect, sociocultural influences, body

disparagement, and eating disordered behaviors at a single point in time. The models discussed above and Tripp's model support the direct pathways from previous stressful experiences to the appraisal/coping process, from the appraisal/coping process to negative affect, from negative affect to body disparagement, from internalization of the thin-ideal to body disparagement, and from body disparagement to eating disturbances. Like Bradford's work, the current study's models again support the direct pathway from internalization of the thin-ideal to eating disturbance as well as the relationships among parental attachment, history of stressful experiences, and the stress-coping process. The current models also provide support for Smolak and Levine's (1996) developmental transition Model As well as the role the early attachment relationship and the role sociocultural pressures play in the developmental of eating disturbances.

Results of this study also examined a second model that fits the data. In this model, women who reported insecure attachments with their parents and women who endorsed a higher incidence of stressful life experiences in the past 6 months were more likely to perceive the transition to college and stressful, which in turn, predicted negative affect (e.g., feeling sad or depressed, unhappy, and stressed) at 6-month and 18-month follow-up. Increased negative affect was related to higher levels of eating disturbance within the same time period and predicted higher level of eating disturbance at 18-month follow-up. Greater internalization of societal pressures to achieve the thin-ideal body type was related to increased body disparagement as well as higher levels of eating disturbance within the same time period. Further, endorsing greater internalization of the thin-ideal predicted body disparagement at a 6-month follow-up and higher levels of eating disturbance at an 18-month follow-up. Body disparagement was related to higher levels of eating disturbance within the same time period and predicted higher levels of eating disturbance at 12-month follow-up.

The findings support Bradford's (2004) earlier work using this model to evaluate the relationships among the factors using data collected within the same time frame, and they lend support to research in which both negative affect and body disparagement were found to predict eating pathology (Killen et al., 1996; Stice & Argas, 1998; Stice, Mazotti, et al., 2000). Furthermore, this model emphasizes the important impact body disparagement, negative affect, and internalization of sociocultural beliefs have on the development of eating disorders in college women.

In summary, the models provide validation for recent studies testing the relationships among parental attachment, stress processes, sociocultural influences, psychological distress, and body image and their role in the development of eating disordered behaviors and attitudes. In addition, this study elaborated on these models by further investigating these relationships using longitudinal data to make causal inferences about relationships among the constructs.

Analysis of Individual Cross-Lagged and Simultaneous Effects

The following analyses examining both cross-lagged and simultaneous effects between negative affect and body image, between internalization of the thin-ideal and body image, between internalization of the thin-ideal and eating disturbance, between body image and eating disturbance, and between negative affect and eating disturbance are unique in that these constructs have not been examined using this type of analysis with longitudinal data collected at three points in time over a 18-month period. Stice (1998b) did investigate the competing cross-lagged and simultaneous relationships between dietary restraint and bulimic symptoms and between negative affect and bulimic symptoms using data from 2 data collections, 9 months apart.

Negative Affect and Body Image

As shown in the measurement model, the significant cross-time correlations for the analyses between negative affect and body image at Time 1 and Time 2 indicated that initial negative affect is correlated with later body disparagement and that initial body disparagement is correlated with later negative affect. These correlations are consistent with empirical evidence suggesting that psychological distress is associated with body disparagement (Kearney-Cooke & Striegel-Moore, 1994; Tripp, 2002; Tripp & Petrie, 2001) and with work by Stice (1994) in which body disparagement led to psychological distress. When temporal stability was controlled, a more stringent analysis that statistically controls for initial levels of each construct, initial negative affect failed to predict later body disparagement, and initial body disparagement failed to predict later negative affect. Further, when the simultaneous effects were examined, Time 2 negative affect failed to predict Time 2 body disparagement, but Time 2 body disparagement did result in increased Time 2 negative affect. Although this result is consistent with Stice's work, the causal inference is more tenuous than prospective tests of these effects because they were measured during the same data collection.

Examination of the relationship between negative affect and body image at Time 2 and Time 4 and at Time 1 and Time 4 resulted in a similar pattern of findings. The significant cross-time correlations suggested that negative affect and body disparagement were reciprocally related, but the more stringent analyses controlling for temporal stability failed to predict cross-time relationships. However, the simultaneous effects at Time 4 also were non-significant in these analyses when controlling for initial levels at Time 1 and at Time 2. Thus, negative affect and body disparagement appear to be reciprocally related based on the cross-time correlations

across a 6-month, 12-month, and 18-month time span, but more stringent analyses revealed only a within-time effect from body image to negative affect and only at Time 2.

The different findings produced by the longitudinal models that controlled for temporal stability and the measurement model that did not control for temporal stability raise questions about the relationship between negative affect and body image. There are several possible explanations for this occurrence. First, it is possible that controlling for temporal stability results in an overly conservative analysis. Second, it is possible the difference in findings is the result of a mis-specified causal time lag between negative affect and body image. Given that both cross-time correlations were significant across a 6-month, 12-month, and 18-month time span, it is unlikely that the effects were due to a relatively short time lag. Alternatively, the causal time lag may be shorter. Recall that negative affect was assessed by asking each participant to rate their emotional state during the past 2 weeks. Further, the current findings indicate some instability in the negative affect construct across time (stability coefficients ranged from .56 to .57). Thus, the measurement of negative affect may have reflected a more state-like variable, whereas body image was viewed as more trait-like variable. Thus, prospectively measuring the two constructs within a shorter time frame may better capture the current mood state that influenced body dissatisfaction. The significant relationship from body image at Time 2 to negative affect at Time 2 also suggests that the time lag may be shorter than 6 months. However, it should be noted that the relationship from negative affect at Time 2 to body image at Time 2 is not a prospective relationship, limiting the confidence in the ability to state that negative affect predicts body dissatisfaction. Finally, it is possible that the developmental period during which these relationships were assessed contributed to the difference in findings. Although data suggest that the mean age for developing an eating disorders occurs in late adolescence or early adulthood

(APA, 2000), the current findings indicate a high degree of stability in body image between Time 1 and Time 2 ($\beta = .90$), between Time 2 and Time 4 ($\beta = .84$), and between Time 1 and Time 4 ($\beta = .90$). These results suggest a lack of variation in individual differences for the construct and the possibility that body image is relatively stable during the first two years of college. It would be meaningful to explore the relationship between negative affect and body image using different time lags, particularly those shorter than 6 months. Additionally, it would be useful to test these models during different developmental periods.

Internalization of the Thin-Ideal and Body Image

Examination of the cross-time correlations between internalization of the thin-ideal and body image at Time 1 and Time 2 suggested that initial internalization of the thin-ideal is correlated with later body disparagement and initial body disparagement is correlated with later internalization of the thin-ideal. When temporal stability was controlled, Time 1 internalization of the thin-ideal predicted Time 2 body disparagement and Time 1 body disparagement predicted Time 2 internalization of the thin-ideal, indicating a reciprocal relationship over time. However, examination of the simultaneous effects revealed only that Time 2 internalization of the thin-ideal predicted Time 2 body disparagement. Time 2 body disparagement did not predict Time 2 internalization of the thin-ideal. This inference is more tenuous than the prospective cross-time inferences, but it is more consistent with empirical findings (Stice, Mazotti, et al., 2000; Stice, Chase, et al., 2001) in which experimental reduction of the thin-ideal internalization resulted in improved body satisfaction. Although research documents the causal path from internalization to body image and theory indicates that we are socialized at an early age to value thinness (Brumberg, 1997), the cross-time effects and cross-time correlation in this study indicate a

reciprocal relationship between the constructs. Further, it seems intuitive that women who are dissatisfied with their bodies would be more vulnerable to continued internalization of the thin-ideal. Thus, it is possible that these reciprocal relations are indicative of a cycle that maintains continued body disparagement.

Examination of the relationships at Time 2 and Time 4 and at Time 1 and Time 4 revealed slightly different findings. Although the cross-time reciprocal correlations between internalization of the thin-ideal and body image were significant for the 12-month time span and the 18-month time span, the more stringent analyses showed that initial internalization of the thin-ideal did predict body disparagement 12 months later and 18 months later, but initial body disparagement did not predict later internalization of the thin-ideal. Differences also emerged in the analyses of the simultaneous effects. When controlling for temporal stability at Time 2, internalization of the thin-ideal at Time 4 resulted in increased body disparagement at Time 4. When controlling for temporal stability at Time 1, internalization of the thin-ideal at Time 4 resulted in increased body disparagement at Time 4, and body disparagement at Time 4 resulted in increased internalization of the thin-ideal at Time 4.

Again the difference in findings between the models that controlled for stability and the measurement model which did not control for temporal stability raise questions about the results. Further, the change in results across the different time spans also raises questions. There are several possibilities for these differences. Again, it is possible that controlling for temporal stability (paths from each construct to the corresponding construct as measured at the follow-up) results in an overly conservative analysis. This would account for the non-significant path from body image to internalization of the thin-ideal when temporal stability was controlled in the analyses at Time 2 and Time 4 and at Time 1 and Time 4 while the cross-time correlations were

significant when temporal stability was not controlled. However, when the models were analyzed at Time 1 and Time 2, the measurement models that controlled for temporal stability and the subsequent model that did not produced consistent findings. A second explanation is the possibility that these constructs were assessed in the wrong developmental period. Both constructs demonstrated stability over each time span. With stability coefficients ranging from .73 to .84, the findings revealed only 40-50% variation in individual difference in internalization of the thin-ideal over time. Similarly, the stability coefficients for body image ranged from .76 to .82. This indicates that internalization of the thin-ideal and body image remain somewhat stable during the first two years of college. Thus, it also would be useful to examine the relationship between internalization of the thin-ideal and body image at different stages of development to better understand the stability of these constructs over time.

Nevertheless, the results from the above analyses do indicate that internalization of the thin-ideal predicts body disparagement. Further, they provide some mixed results about the predictive relationship from body image to internalization of the thin-ideal, suggesting that there may be a reciprocal relationship and calling for additional research to investigate this possibility.

Internalization of the Thin-Ideal and Eating Disturbance

Examination of the cross-time correlations between internalization of the thin-ideal and eating disturbance at Time 1 and Time 2 suggested that initial internalization of the thin-ideal is correlated with later eating disturbance and initial eating disturbance is correlated with later internalization of the thin-ideal. However, when temporal stability was controlled, Time 1 internalization of the thin-ideal failed to predict Time 2 eating disturbance, and Time 1 eating disturbance did not predict Time 2 internalization of the thin-ideal. Examination of the

simultaneous effects did reveal that Time 2 internalization of the thin-ideal predicted Time 2 eating disturbance. The simultaneous effect is consistent with empirical evidence documenting a positive relationship between internalization of the thin-ideal and eating pathology (Stice and Shaw, 1994) and with studies demonstrating a reduction in dieting behaviors and bulimic pathology as a result of experimental reduction of the thin-ideal internalization (Stice, Mazotti, et al., 2000; Stice, Chase, et al., 2001), but the predictive inference is tenuous because both constructs were measured at the same time.

Examination of the relationships between internalization of the thin-ideal and eating disturbance at Time 2 and Time 4 and at Time 1 and Time 4 revealed only slightly different findings. The cross-time correlations for both sets of analyses were significant and positive, suggesting a reciprocal relationship between internalization of the thin-ideal and eating disturbance. When temporal stability was controlled, Time 2 internalization of the thin-ideal did predict Time 4 eating disturbance, but Time 2 eating disturbance did not predict Time 4 internalization of the thin-ideal. Analyses at Time 1 and Time 4 again failed to reveal any cross-time predictive relationship between internalization of the thin-ideal and eating disturbance. The simultaneous analyses did reveal that Time 4 internalization of the thin-ideal predicted Time 4 eating disturbance when controlling for temporal stability at Time 1 and at Time 2.

Again, it is possible that controlling for temporal stability (paths from each construct to the corresponding construct as measured at the follow-up) results in an overly conservative analysis. Alternatively, these constructs also demonstrated stability over each time span. With stability coefficients ranging from .74 to .85, the findings revealed only a small amount of variation in individual difference in internalization of the thin-ideal over time. Similarly, the stability coefficients for eating disturbance ranged from .73 to .82. This indicates that

internalization of the thin-ideal and eating disturbance remain relatively stable during the first two years of college, so it also would be useful to test these models at different stages of development to better understand the stability of these constructs over time.

Body Image and Eating Disturbance

Examination of the cross-time correlations between body image and eating disturbance at Time 1 and Time 2 suggested that initial body disparagement is correlated with later eating disturbance and initial eating disturbance is correlated with later body disparagement. However, when temporal stability was controlled, Time 1 body disparagement failed to predict Time 2 eating disturbance, and Time 1 eating disturbance did not predict Time 2 body disparagement. Examination of the simultaneous effects did reveal that Time 2 body disparagement predicted Time 2 eating disturbance. This result is consistent with previous evidence showing that body dissatisfaction predicted growth in dieting (Stice, Mazotti et al., 2000) as well as bulimic symptomatology (Killen et al., 1994; Patton et al., 1990; Stice & Agras, 1998), but the predictive inference is tenuous because both constructs were measured at the same time.

Examination of the relationships between body image and eating disturbance at Time 2 and Time 4 and at Time 1 and Time 4 resulted in a similar pattern of findings. The significant cross-time correlations suggested that body disparagement and eating disturbance were reciprocally related, but the more stringent analyses controlling for temporal stability failed to predict cross-time relationships. The analyses of the simultaneous effects from body image to eating disturbance at Time 4 also were significant when controlling for the initial effects at Time 1 and at Time 2. However, the analyses of the simultaneous effects also showed that Time 4 eating disturbance predicted Time 4 body disparagement. Thus, body disparagement and eating

disturbance appear to be reciprocally related based on the cross-time correlations across a 6-month, 12-month, and 18-month time span, but more stringent analyses revealed that body disparagement was associated with increased eating disturbance only at Time 2 and reciprocal contemporaneous effects at Time 4.

Still, the longitudinal models that controlled for temporal stability and the model that did not control for temporal stability produced different findings which raise questions about the relationship between negative affect and body image. Again, it is possible that controlling for temporal stability results in an overly conservative analysis. Second, it is possible that the developmental period during which these relationships were assessed contributed to the difference in findings. Although data suggests that mean age for the development of eating disorders occurs in late adolescence or early adulthood (APA, 2000), the current findings indicate a high degree of stability in eating disorder symptomatology between Time 1 and Time 2 ($\beta = .82$), between Time 2 and Time 4 ($\beta = .78$), and between Time 1 and Time 4 ($\beta = .75$). Body image also was extremely stable across time (stability coefficients ranged from .82 to .90). Thus, these results suggest a lack of variation in individual differences for these two constructs and the possibility that body image and eating disorder attitudes and behaviors are relatively stable during the first two years of college. Alternatively, other variables not assessed in this study could be influencing these results. Thus, it would be meaningful to explore the relationship between body image and eating disturbance during different developmental periods as well as additional mediating and moderation factors that may influence the relationship between the constructs.

Negative Affect and Eating Disturbance

Examination of the cross-time correlations between negative affect and eating disturbance at Time 1 and Time 2 suggested that initial negative affect is correlated with later eating disturbance and initial eating disturbance is correlated with later negative affect. When temporal stability was controlled, Time 1 negative affect predicted Time 2 eating disturbance and Time 1 eating disturbance predicted Time 2 negative affect, indicating a reciprocal relationship over time. Further, examination of the simultaneous effects also revealed a reciprocal relationship between Time 2 negative affect and Time 2 eating disturbance. These results are consistent with previous research in which negative affect has been found to predict bulimic symptoms (Stice & Argas, 1998), binge-eating behaviors (Stice, Presnell, and Spangler, 2002), and general eating disturbances (Johnson, Cohen, Kasen, and Brook, 2002). The results also lend support to research in which bulimic symptoms were found to predict increases in depression (Stice & Bearman, 2001) and research in which bulimia and depression were found to be reciprocally related (Stice, Burton, & Shaw, 2004). Theory suggests that individuals engage in eating disordered behaviors to cope with their negative mood states (Humphrey, 1986; Johnson & Larson, 1982). However, continuing to engage in these behaviors has been shown to result in increased feelings of shame, guilt and dysphoria as well as symptoms of depression due to nutritional deficits (Kaye, Gendall, & Strober, 1998). Thus, it is possible that these reciprocal relations are indicative of a cycle that maintains continued eating disturbance as well as continued negative affect.

Examination of the relationships at Time 2 and Time 4 revealed slightly different findings. Although the cross-time reciprocal correlations between negative affect and eating disturbance were significant for the 12-month time lag, the more stringent analyses showed that

negative affect did not predict eating disturbance and eating disturbance did not predict negative affect. Further, the analyses of the simultaneous effects at Time 4 when controlling for initial levels at Time 2 also failed to reveal any significant relationships. Continued examination of the relationships at Time 1 and Time 4 also showed that the cross-time reciprocal correlations between internalization of the thin-ideal and body image were significant for the 18-month time lag. This time, the more stringent analyses also showed that negative affect did predict eating disturbance 18 months later, but eating disturbance did not predict negative affect. When controlling for initial levels at Time 1, analyses of the simultaneous effect revealed that negative affect led to increased eating disordered attitudes and behaviors and eating disorder attitudes and behaviors resulted increased negative affect. However, these simultaneous results were only reciprocal at Time 2, when controlling for the initial levels at Time 1.

Again, the differences in the findings between the models controlling for stability and the model not controlling for stability as well as the differences in findings across the different time spans is bothersome and raises questions about the results. It is still possible that controlling for temporal stability is an overly conservative method of analysis, accounting for the non-significant cross-time paths in the analyses at Time 2 and Time 4 and the failure of eating disorder symptomatology at Time 1 to predict negative affect at Time 4 even though the cross-time correlations were significant. Given that the models controlling for stability and the model that did not control for stability in the analyses at Time 1 and Time 2 produced consistent results, it is possible that controlling for stability was an overly conservative method only for the longer time lag only. A second explanation is the possibility that the causal time lag between negative affect and eating disturbance was mis-specified. The reciprocal prospective cross-time effects between Time 1 and Time 2 suggest that 6 months is an appropriate time lag, but the lack of

effects between Time 2 and Time 4 suggest that the 12-month time lag may be incorrectly specified. Then, interestingly, the cross-time path from negative affect to eating disturbance is again significant with the 18-month time lag. Again, recall that negative affect was assessed by asking each participant to rate their emotional state during the past 2 weeks. Further, the current findings indicate some instability in the negative affect construct across time (stability coefficients ranged from .48 to .56). Thus, the measurement of negative affect is more state-like variable, whereas eating disturbance is more trait-like variable. Thus, prospectively measuring the two constructs within a shorter time frame may better capture the current mood state that influenced eating disturbance. Additionally, the findings revealed only a small amount of variation in individual difference in eating disturbance over time (stability coefficients ranging from .75 to .83). Although less stable than eating disturbance, the stability coefficients for negative affect ranged from .48 to .56. This indicates that eating disturbance may remain relatively stable during the first two years of college. Thus, it would be useful to examine the relationship between negative affect and eating disturbance using different time lags. It also would be useful to test these models at different stages of development to better understand the stability of these constructs over time.

Nevertheless, the results from these analyses do support the hypothesis that negative affect predicts eating disorder symptomatology. Further, they provide some mixed results about predictive relationship from eating disturbance to negative affect, suggesting that there may be a reciprocal relationship and calling for the need for additional research to investigate this possibility.

Summary of Findings

The analyses of the cross-lagged effects and simultaneous effects provided additional information about the predictive relationships between specific factors involved in the development of eating disorders. However, the analyses were often contradictory, making it difficult to make firm conclusions about these relationships and raising further questions about the exact nature of the relationships between negative affect and body image, between internalization of the thin-ideal and body image, between internalization of the thin-ideal and eating disturbance, between body image and eating disturbance, and between negative affect and eating disturbance. The findings also raised questions about the structural equation modeling methods used to analyze these relationships (i.e., stringent analyses), the time frames used to make predictions, and the developmental period during which to study these patterns of relationships among the constructs.

Treatment Implications

Based on the results of the current study, several treatment implications are indicated. First, the results of the study highlight the importance the parent-child bond has on the child's subsequent health and development. The attachment bond forms during the child's first year of life (Ainsworth et al., 1978), which is a critical developmental period. Parenting education classes and psychoeducational material discussing the importance of a secure attachment bond could reduce the development of insecure attachment patterns and subsequent developmental difficulties and/or psychological pathology. In addition, since individuals who do not develop secure attachment bonds may be at risk, early identification of those individuals could assist them in developing more secure attachments or enable prompt provision of support services that

might reduce that individual's risk of developing future psychological and/or adjustment difficulties.

The results regarding the importance of the attachment bond also have clinical implications for professionals working with female college students. In addition to assessing maladaptive family functioning, psychologists or other mental health professionals need to recognize and encourage students to continue to use their parents as a secure base and a source of support when students speak positively of their relationships with their parents. College women who continue to seek parental support sometimes experience shame and embarrassment, feeling as though they are failing at developing autonomy (Komarovsky, 1986). However, college is often a stressful period, and activation of the attachment system is an appropriate response. Encouraging these effective coping strategies could empower women and make the transition less stressful.

A second implication of this study concerns the transition individuals encounter when they begin college. From the results of the study, it is clear that the first semester is a period of stressful adjustment for many women. Furthermore, those who have a particularly difficult time managing the increased demands associated with the transition (e.g., academic demands, separation-individuation process, etc.) appear to be more vulnerable to developing later psychological distress. Programs designed to encourage the development of appropriate coping skills and provide increased support during this period could reduce the difficulty many experience and prevent the development of severe stress and anxiety that might lead women to cope with the adjustment using maladaptive strategies, including eating disordered behaviors. These programs might include psychoeducational groups for women prior to beginning college, providing incoming freshman with an upper-class mentor to guide them through the transition,

support groups, and encouraging the use of the counseling, guidance, and other support services available on the college campus.

The results of this study also offer further evidence of the damaging effects the sociocultural pressures to achieve the thin-ideal body size can have on a young woman's body image and potential to develop subsequent eating disordered behaviors. Although changes at a societal level, including recognition of women's achievements beyond beauty and greater appreciation for all body types, are needed, one must also recognize the extensive and time-consuming effort this entails. Looking at a more immediate option, primary and secondary prevention of negative body image through the use of media literacy (a cycle of critique employing awareness of the media message, analysis and purpose of the message, and taking action through activism or advocacy) has proven successful in early studies (Irving, DuPen, & Berel, 1998). Specifically, research by Stice and colleagues (Stice, Chase, et al., 2001; Stice, Mazotti, et al., 2000) showed that an experimental reduction of the thin-ideal internalization resulted in decreased body dissatisfaction, negative affect, and eating pathology. In addition, Posavac, Posavac, and Weigel (2001) found that a psychoeducational discussion of media literacy presented to women with negative body image resulted in decreased social comparison and decreased negative effect from exposure to future images of the thin-ideal. Thus, groups of this nature and/or materials provided to women, especially those navigating the transitional period between high school and college, could aid in the prevention of eating disordered behaviors by reducing the internalization of societal pressures to be thin and by promoting positive body image. It should be noted, however, that these programs generally include ongoing discussions and follow-up. In addition, the focus should be on critiquing the media and

engaging in education discussions to improved body image, not a discussion about behaviors associated with eating disorders that might lead to the development of pathology.

Finally, the results of this study confirm the presence of eating disorders and pathological eating behaviors among women on college campuses. Although the numbers may not seem large, they do indicate the need for both primary and secondary prevention of eating disorders and education regarding healthy diet and exercise habits. Given the stability of the constructs (body image, internalization of the thin-ideal, negative affect, eating disturbance) over time in this study and the number of women who participated in this study indicating the presence of eating disordered attitudes and behaviors during their first semester in college, it may be helpful to implement these programs prior to this transition, either in an orientation to college workshop or during high school. Psychoeducational programs presenting information about eating disorders, healthy weight management, nutrition, body image, and societal pressures have been shown to be effective with both high school and college women (see Stice & Shaw, 2004 for a review).

Limitations and Implications for Future Research

Due to methodology and research design, the current study is limited in several ways. First, the sample size is a problem in this study. As discussed earlier, Stevens (1996) recommends as least 15 cases per measured variable, and Bentler and Chou (1987) encourage no fewer than five cases per parameter estimate. Using smaller samples often results in improper solutions and decreased accuracy of parameter estimates. The number of participants who completed all data collections was insufficient to fully evaluate the proposed cross-lagged Model and only minimally sufficient to evaluate the more simplistic, integrative model. Thus, although

these models fit the data well, the causal inferences may be limited. Future research replicating this work and using a much larger sample would strengthen the ability to make casual inferences about the relationships among the factors. A very large sample also would allow researchers to test a fully cross-lagged model.

In addition, all data were collected using self-report measures. Thus, results may be affected by monomethod bias (Cook & Campbell, 1979). In particular, participants' self-reports of their parent-child attachment relationships may differ from results yielded by direct observation measures. As with all self-report measures, it is difficult to determine whether or not the participants responded to the items honestly, especially those inquiring about eating attitudes and behaviors. Given that a core feature of eating disorders is secrecy about the symptoms themselves, some participants may not have been completely honest in reporting their eating and weight-related behaviors. Further, Beglin and Fairburn (1992) suggests that prevalence rates of eating disorders are often underrepresented because women experiencing eating disturbances often elect to not participate in epidemiological studies. Attempts to evaluate participant honesty were made using a social desirability scale (SDS). Although results indicate that 18% of participants responded to the items on the SDS in a socially desirable manner, this measure had poor internal consistency reliability. Thus, the scale may not be an accurate measure of response bias. However, correlations between the SDS and other measure variables were small and suggested that social desirability was not a concern in this study. Given the lack of relationship between the SDS and other measured variables and the fact that the prevalence rates of this study were similar to those found in other recent studies, there is confidence in the self-report of the participants. However, some individuals will always choose to refrain from participating. Thus, all studies may be affected by an inability to obtain accurate prevalence rates. To improve the

accuracy of information gathered, future research utilizing data multiple methods of gathering data is indicated.

One purpose of this study was to investigate adjustment to college and the effect this transition has on the development of psychological distress and eating disordered behaviors and attitudes. Based on previous research and theory, it is expected that individuals experiencing the greatest amount of difficulty with the transition would be most likely to develop poor coping mechanisms and the subsequent consequences (e.g., negative affect, eating disturbances, etc.) (Shatford & Evans, 1986; Tripp, 2002; Tripp & Petrie, 2001). Unfortunately, those individuals experiencing the highest levels of difficulty are likely functioning at lower levels and may have stopped attending classes, failed to seek out research credit, or not volunteered to participate in this study. Consequently, the self-selection process and means by which participants were encouraged to complete the questionnaires may have limited participation to those individuals functioning at higher levels and experiencing less difficulty with the transition to college and eliminated a large group of individuals that were of interest. However, the prevalence rates in the current study were comparable to past research, suggesting that similar individuals did participate in the current study.

With regards to the Appraisal Coping Factor, it is important to note that only two measured variables (PSS and Academic Demands) ended up loading on this factor. All measures loaded appropriately on the Appraisal/Coping Factor. However, when the standardized solution was examined, the paired items measuring appraisal and coping of stressors related to weight gain concerns, romantic relationship demands, living arrangement difficulties, and friendship demands had very low loadings compared to the PSS and the paired items measuring academic demands. Thus, these measures explained very little variance in the Appraisal/Coping Factor.

Due to the timing of the data collection (i.e., during the 8-12th weeks of the semester), it is possible that many of the participants had not experienced stress related to weight gain concerns, romantic relationship demands, friendship demands, or living arrangement difficulties. Thus, increased academic demands may have been the most salient stressor. Although managing demands associated with new living situations, new friendships, romantic relationships, and weight gain are important in the transition from high school to college, the PSS also offered a general assessment (i.e., life hassles/irritations, personal problems, time pressures, important things/changes, and unexpected occurrences) of appraisal, perception of controllability, and coping and appeared to be a better indicator of the appraisal/coping process. To improve model fit, these were dropped from the measurement model. Thus, despite dropping the four paired items, this factor was well-represented through the academic concerns items and the more general PSS.

An additional limitation is that the data assessing parental attachment and history of stress was collected during the first data collection, along with the remaining measures in the data collection. Although the instructions requested participant's to reflect on their previous experiences with their parents and specific stressors, it is possible that responses to these items were based more on current perceptions rather than actual past experiences. Thus, further longitudinal research investigating parental attachment and previous stressful experiences is needed. Although time-consuming and difficult, it would be especially meaningful to begin data collection regarding parental-child attachment during the early months of the child's life using observational means and to follow that same child into early adulthood. Research of this type would provide a more accurate measure of the attachment relationship and subsequent

developments as well as provide longitudinal data. It also would help identify critical points at which interventions might assist in the prevention of these negative outcomes.

Structural equation modeling tends to be sample dependent. Although the results of this study did offer confirmation of Bradford's (2004) results, the sample employed in this study was a subset of the sample who participate in the 2004 study. Consequently, the current model needs to be tested on an independent sample of college women. In addition, the model needs to be tested on individuals diagnosed with an eating disorder to determine if the constructs representing the development of eating pathology are an accurate representation in a clinical population. Furthermore, the results of this study are only generalizable to freshman females navigating the transition from high school to college and the subsequent development of eating pathology. Testing the model with different age groups and during different transitional periods is also needed.

Finally, although the models tested in this study provided a good fit for the data, the results do not imply that these are the only valid models (Kline, 1998). Other models could provide equally valid explanations for the development of eating disturbances, and additional constructs that were not measured in this study could also contribute additional information regarding the development of eating pathology. Future research might investigate the roles personality traits such as perfectionism and impulse control, social support, reciprocal adult attachments, athletic involvement, perceptions of control, and self-esteem play as risk factors and protective factors in the development of eating disturbances (Stice, 2001b; Twamley & Davis, 1999; West & Sheldon-Keller; 1994).

Conclusions

The results of this study help to clarify the relationships among parental attachment, history of experienced stressors, the appraisal/coping process, psychological distress, body image, internalization of the thin-ideal, and eating disturbance, constructs that have been identified by previous researchers as being important in the development of eating disorders, by examining the constructs in a prospective longitudinal study. Through the use of structural equation modeling, analysis of the data identified the direct and indirect pathways between the factors and eating pathology and noted the causal relationships among factors, providing additional information about the development of eating disorders that can be used to further prevention, treatment, and future research in the area.

Table 1
Descriptive Statistics for Measured Variables

Variable	No. Items	<i>M</i>	<i>SD</i>	Skewness	Kurtosis	Internal Consistency
Time 1						
SDS	12	6.45	3.11	-.18	-.77	.62
PBI-MC	12	29.96	6.44	-1.48	1.88	.90
PBI-MO	13	13.43	6.56	.77	.66	.82
PBI-FC	12	26.29	8.40	-.94	.27	.93
PBI-FO	13	12.58	7.06	.96	.68	.84
ICSRLE	49	101.41	21.26	.49	.58	.92
ICSRLE-AC	3	2.27	0.83	.46	-.53	.76
ICSRLE-AN	5	1.62	0.51	1.12	1.69	.57
ICSRLE-DC	10	2.48	0.63	.20	-.64	.81
ICSRLE-FP	3	1.86	0.76	.87	.03	.80
ICSRLE-GSM	5	1.76	0.60	.79	.16	.80
ICSRLE-RP	3	2.09	0.92	.68	-.61	.76
ICSRLE-TP	7	2.57	0.67	-.05	-.82	.82
PSS	14	26.81	7.62	.10	-.26	.86
APPCOP-APP	5	14.64	3.76	-.08	-.44	.52
APPCOP-COP	5	13.03	3.59	.06	-.40	.48
APPCOP-AC	2	6.57	1.79	.06	-.53	.79
APPCOP-FR	2	5.03	2.44	.36	-.95	.91
APPCOP-LA	2	5.46	2.57	.13	-1.12	.93
APPCOP-RO	2	5.65	2.72	.01	-1.31	.93
APPCOP-WT	2	4.96	2.60	.32	-1.20	.94
SAD	1	1.61	1.12	.65	-.11	n/a
HAPPY	1	2.96	0.84	-.49	.10	n/a
SHAME	1	0.52	0.94	1.18	.88	n/a
GUILT	1	0.61	0.92	1.25	1.75	n/a
CONFIDENT	1	2.50	0.96	-.21	-.15	n/a
ANXIOUS	1	2.37	1.19	.29	-.80	n/a
STRESSED	1	2.82	1.06	-.04	-.96	n/a
BPSS-BODY	7	3.62	1.09	-.23	-.45	.88
BPSS-FACE	3	4.44	0.94	-.67	.61	.69
MBSRQ-AE	7	3.45	0.76	-.58	.15	.87
BAAR-PF	9	4.57	1.03	-.43	.23	.79
BAAR-AT	10	2.34	0.96	.67	-.08	.84
EAT	26	9.54	9.31	2.08	5.21	.86
BULITR	28	62.32	20.00	1.47	3.11	.94

Note. Time 1 = Fall 2003; Time 2 = Spring 2004; Time 4 = Spring 2005; SDS= Social Desirability Scale (tendency to respond in socially desirable direction: scores range from 0[*low social desirability*] to 12[*high social desirability*]); PBI-MC = Parental Bonding Instrument-Mother Care Scale (affective element of mother-child relationship: scores range from 12[*low care*] to 48[*high care*]); PBI-MO = Parental Bonding Instrument-Mother Overprotection Scale (maternal intrusiveness and control: scores range from 13[*low overprotection*] to 52[*high overprotection*]); PBI-FC = Parental Bonding Instrument-Father Care Scale (affective element of father-child relationship: scores range from 12[*low care*] to 48[*high care*]); PBI-FO = Parental Bonding Instrument-Father Overprotection Scale (paternal intrusiveness and control: scores range from 13[*low overprotection*] to 52[*high overprotection*]); ICSRLE = Inventory of College Students' Recent Life Experiences (experience and influence of stressors college students experience: scores range from 49[*minimal experience of stressful events*] to 196[*great experience of stressful events*]); ICSRLE-AC = Inventory of College Students' Recent Life Experiences-Academic Alienation Scale (experience and influence of stressors related to satisfaction in school: scores range from 3[*minimal experience of stressors*] to 12[*great experience of stressors*]); ICSRLE-AN = Inventory of College Students' Recent

Table 1 (continued).

Variable	No. Items	<i>M</i>	<i>SD</i>	Skewness	Kurtosis	Internal Consistency
Time 2						
SAD	1	1.20	0.97	.65	-.11	n/a
HAPPY	1	2.81	0.82	-.49	.10	n/a
SHAME	1	0.65	0.80	1.18	.88	n/a
GUILT	1	0.80	0.87	1.25	1.75	n/a
CONFIDENT	1	2.38	0.95	-.21	-.15	n/a
ANXIOUS	1	1.83	1.12	.29	-.80	n/a
STRESSED	1	2.47	1.03	-.36	-.96	n/a
BPSS-BODY	7	3.62	1.05	-.36	-.43	.85
BPSS-FACE	3	4.21	0.87	-.34	.51	.68
MBSRQ-AE	7	3.53	0.73	-.50	.41	.87
BAAR-PF	9	4.61	1.01	-.53	.20	.79
BAAR-AT	10	2.39	0.96	.95	.91	.85
EAT	26	8.49	8.88	2.14	5.64	.86
BULITR	28	60.36	20.29	1.21	1.03	.94
Time 4						
SAD	1	1.16	0.98	.75	-.04	n/a
HAPPY	1	2.72	0.85	-.63	.53	n/a
SHAME	1	0.70	0.88	1.20	.90	n/a
GUILT	1	0.85	0.93	1.13	1.01	n/a
CONFIDENT	1	2.39	0.84	-.12	-.23	n/a
ANXIOUS	1	1.71	1.13	.25	-.73	n/a
STRESSED	1	2.49	1.02	-.15	-.92	n/a
BPSS-BODY	7	3.52	1.05	-.17	-.69	.86
BPSS-FACE	3	4.16	0.83	-.28	.08	.64
MBSRQ-AE	7	3.51	0.76	-.61	-.03	.88
BAAR-PF	9	4.72	1.04	-.60	.24	.82
BAAR-AT	10	2.45	0.99	.79	.25	.87
EAT	26	8.67	8.60	1.78	3.33	.85
BULITR	28	61.56	20.46	1.43	1.53	.96

Note. Life Experiences-Assorted Annoyances Scale (experience and influence of stressors that evoke anger rather than anxiety: scores range from 5[*minimal experience of stressors*] to 20[*great experience of stressors*]); ICSRLE-DC = Inventory of College Students' Recent Life Experiences-Developmental Challenge Scale (experience and influence of stressors related to developmental and academic changes: scores range from 10[*minimal experience of stressors*] to 40[*great experience of stressors*]); ICSRLE-FP = Inventory of College Students' Recent Life Experiences-Friendship Problems Scale (experience and influence of stressors related to peer relationships: scores range from 3[*minimal experience of stressors*] to 12[*great experience of stressors*]); ICSRLE-GSM = Inventory of College Students' Recent Life Experiences-General Social Mistreatment Scale (experience and influence of stressors related to social mistreatment by unspecified sources: scores range from 5[*minimal experience of stressors*] to 20[*great experience of stressors*]); ICSRLE-RP = Inventory of College Students' Recent Life Experiences-Romantic Problems Scale (experience and influence of stressors related to romantic relationships: scores range from 3[*minimal experience of stressors*] to 12[*great experience of stressors*]); ICSRLE-TP = Inventory of College Students' Recent Life Experiences-Time Pressure Scale (experience and influence of stressors related to demands on time: scores range from 7[*minimal experience of stressors*] to 28[*great experience of stressors*]); PSS = Perceived Stress Scale (evaluation of stress: scores range from 0[*low stress*] to 56[*high stress*]); APPCOP-APP = Appraisal of stress since beginning school (scores range from 5[*low stress*] to 25[*high stress*]); APPCOP-COP = Coping with experienced stress since beginning school question (scores range from 5[*effective coping*] to 25[*ineffective coping*]); APPCOP-AC = Appraisal of academic demands and coping with experienced academic demands since beginning school (scores range from 2[*low stress*] to 10[*high stress*]); APPCOP-FR = Appraisal of new friendship and coping 10[*ineffective coping*]); APPCOP-LA = Appraisal of living arrangement demands and coping with experienced with

experienced new friendship demands since beginning school (scores range from 2[*effective coping*] to living arrangement demands since beginning school (scores range from 2[*effective coping*] to 10[*ineffective coping*]); APPCOP-ROM = Appraisal of romantic demands and coping with experienced romantic demands since beginning school (scores range from 2[*effective coping*] to 10[*ineffective coping*]); APPCOP-WT = Appraisal of weight gain and coping with experienced weight gain since beginning school (scores range from 2[*effective coping*] to 10[*ineffective coping*]); SAD= Visual Analogue Mood Scale Sad or Depressed item (scores range from 0[*not at all*] to 4[*extremely*]); HAPPY= Visual Analogue Mood Scale Happy item (scores range from 0[*not at all*] to 4[*extremely*]); SHAME= Visual Analogue Mood Scale Shameful item (scores range from 0[*not at all*] to 4[*extremely*]); GUILT= Visual Analogue Mood Scale Guilty item (scores range from 0[*not at all*] to 4[*extremely*]); CONFIDENT= Visual Analogue Mood Scale Confident item (scores range from 0[*not at all*] to 4[*extremely*]); ANXIOUS= Visual Analogue Mood Scale Anxious item (scores range from 0[*not at all*] to 4[*extremely*]); STRESS= Visual Analogue Mood Scale Stressed item (scores range from 0[*not at all*] to 4[*extremely*]); BPSS-R BODY= Body Parts Satisfaction Scale- Revised Body Subscale (satisfaction with individual body parts: scores range from 1[*extremely dissatisfied*] to 6[*extremely satisfied*]); BPSS-R FACE= Body Parts Satisfaction Scale- Revised Face Subscale (satisfaction with individual facial parts: scores range from 1[*extremely dissatisfied*] to 6[*extremely satisfied*]); MBSRQ-AE= Multidimensional Body Self Relations Questionnaire Appearance Evaluation Subscale (attitudes concerning body image: scores range from 1[*poor body image*] to 5[*strong body image*]); BAAR-PF= Beliefs About Attractiveness-Revised Physically Fit Factor (importance of being physically fit and in shape: scores ranging from 1[*low importance*] to 7[*high importance*]); BAAR-AT= Beliefs About Attractiveness Revised - Attractive and Thin Factor (importance of attractiveness: scores range 1[*low importance*] to 7[*high importance*]); EAT-26= Eating Attitudes Test-26 (behavioral and attitudinal concerns about dieting: scores range from 0[*few concerns*] to 78[*multiple concerns*]); BULIT-R= Bulimia Test-Revised (bulimic symptomatology: scores range from 28[*few symptoms*] to 140[*multiple symptoms*]).

Skewness ≤ 2.00 is acceptable; Kurtosis ≤ 7.00 is acceptable.

Table 2A

Evaluation of Dropout: Independent Sample t-Tests for Continuous Variables

Variable		<i>N</i>	<i>M</i>	<i>SD</i>	<i>df</i>	<i>t</i>
AGE	Retain	238	18.20	0.48	473	0.49
	Dropout	237	18.18	0.42		
HOURS	Retain	223	13.75	1.65	460	-0.50
	Dropout	229	13.83	1.67		
GPA	Retain	206	85.01	10.61	398	0.29
	Dropout	194	84.67	9.31		
BMI	Retain	238	22.49	4.18	473	-0.05
	Dropout	237	22.51	4.28		
IDEAL BMI	Retain	238	20.61	2.45	473	-1.01
	Dropout	237	20.83	2.30		
SDS	Retain	238	6.45	3.11	473	1.01
	Dropout	237	6.18	2.96		
PBI-MC	Retain	238	29.96	6.44	473	0.38
	Dropout	237	29.73	6.86		
PBI-MO	Retain	238	13.43	6.56	473	0.19
	Dropout	237	13.31	7.11		
PBI-FO	Retain	238	12.58	7.06	473	-0.76
	Dropout	237	13.11	8.02		
PBI-FC	Retain	238	26.29	8.40	473	0.72
	Dropout	237	25.72	8.73		
PSS	Retain	238	26.81	7.62	473	-1.01
	Dropout	237	27.48	6.80		
APPCOP-APP	Retain	238	14.64	3.76	473	-0.27
	Dropout	237	14.73	3.54		
APPCOP-COP	Retain	238	13.03	3.59	473	-1.06
	Dropout	237	13.38	3.63		
APPCOP-AC	Retain	238	6.57	1.79	473	1.17
	Dropout	237	6.67	1.89		
APPCOP-FR	Retain	238	5.03	2.44	473	-0.55
	Dropout	237	4.99	2.38		
APPCOP-LA	Retain	238	5.46	2.57	473	-0.77
	Dropout	237	5.75	2.55		
APPCOP-RO	Retain	238	5.65	2.72	473	1.19
	Dropout	237	5.37	2.60		
APPCOP-WT	Retain	238	4.96	2.60	473	1.74
	Dropout	237	4.81	2.51		
ICSRLE	Retain	238	101.41	21.26	473	-0.66
	Dropout	237	102.70	21.08		
ICSRLE-TP	Retain	238	2.57	0.67	473	-0.47
	Dropout	237	2.55	0.67		
ICSRLE-DC	Retain	238	2.48	0.63	473	-0.87
	Dropout	237	2.50	0.62		
ICSRLE-AC	Retain	238	2.27	0.83	473	-0.83
	Dropout	237	2.28	0.80		
ICSRLE-RP	Retain	238	2.09	0.92	473	-0.27
	Dropout	237	2.14	0.94		

Note. AGE = Age in Years; HOURS = Current Number of Credits Enrolled; GPA = High School Grade Point Average; SDS= Social Desirability Scale (tendency to respond in socially desirable direction: scores range from 0[low social desirability] to 12[high social desirability]); PBI-MC = Parental Bonding Instrument-Mother Care Scale; PBI-MO = Parental Bonding Instrument-Mother Overprotection Scale; PBI-FC = Parental Bonding

(table continues)

Table 2A (continued).

Variable		<i>N</i>	<i>M</i>	<i>SD</i>	<i>df</i>	<i>t</i>
ICSRLE-AN	Retain	238	1.62	0.51	473	0.37
	Dropout	237	1.62	0.51		
ICSRLE-GSM	Retain	238	1.76	0.60	473	0.63
	Dropout	237	1.81	0.65		
ICSRLE-FP	Retain	238	1.86	0.76	473	0.10
	Dropout	237	1.89	0.78		
SAD	Retain	238	1.61	1.12	473	-0.90
	Dropout	237	1.70	1.09		
HAPPY	Retain	238	2.96	0.84	473	1.93
	Dropout	237	2.81	0.87		
SHAME	Retain	238	0.52	0.94	473	0.55
	Dropout	237	0.47	0.83		
GUILT	Retain	238	0.61	0.92	473	0.22
	Dropout	237	0.59	0.92		
CONFIDENT	Retain	238	2.50	0.96	473	0.27
	Dropout	237	2.47	0.95		
ANXIOUS	Retain	238	2.37	1.19	473	-0.17
	Dropout	237	2.38	1.14		
STRESSED	Retain	238	2.82	1.06	473	-1.01
	Dropout	237	2.92	1.01		
BAAR-PF	Retain	238	4.57	1.03	473	1.75
	Dropout	237	4.37	1.30		
BAAR-AT	Retain	238	2.34	0.96	473	0.73
	Dropout	237	2.27	0.94		
MBSRQ-AE	Retain	238	3.45	0.76	473	-1.20
	Dropout	237	3.53	0.75		
BPSS-BODY	Retain	238	3.62	1.09	473	-0.42
	Dropout	237	3.65	1.12		
BPSS-FACE	Retain	238	4.44	0.96	473	-0.98
	Dropout	237	4.49	0.92		
EAT	Retain	238	9.54	9.31	473	0.15
	Dropout	237	9.42	9.28		
BULIT-R	Retain	238	62.32	20.00	473	-1.41
	Dropout	237	64.97	20.94		

Note. Instrument-Father Care Scale ; PBI-FO = Parental Bonding Instrument-Father Overprotection Scale; ICSRLE = Inventory of College Students' Recent Life Experiences; ICSRLE-AC = Inventory of College Students' Recent Life Experiences-Academic Alienation Scale; ICSRLE-AN = Inventory of College Students' Recent Life Experiences-Assorted Annoyances Scale; ICSRLE-DC = Inventory of College Students' Recent Life Experiences-Developmental Challenge Scale; ICSRLE-FP = Inventory of College Students' Recent Life Experiences-Friendship Problems Scale; ICSRLE-GSM = Inventory of College Students' Recent Life Experiences-General Social Mistreatment Scale; ICSRLE-RP = Inventory of College Students' Recent Life Experiences-Romantic Problems Scale; ICSRLE-TP = Inventory of College Students' Recent Life Experiences-Time Pressure Scale; PSS = Perceived Stress Scale; APPCOP-APP = Appraisal of stress since beginning school; APPCOP-COP = Coping with experienced stress since beginning school; APPCOP-AC = Appraisal of academic demands and coping with experienced academic demands since beginning school; APPCOP-FR = Appraisal of new friendship and coping with experienced new friendship demands since beginning school; APPCOP-LA = Appraisal of living arrangement demands and coping with experienced living arrangement demands since beginning school; APPCOP-ROM = Appraisal of romantic demands and coping with experienced romantic demands since beginning school; APPCOP-WT = Appraisal of weight gain and coping with experienced weight gain since beginning school; SAD= Visual Analogue Mood Scale Sad or Depressed item; HAPPY= Visual Analogue Mood Scale Happy item; SHAME= Visual Analogue Mood Scale Shameful item; GUILT= Visual Analogue Mood Scale Guilty item; CONFIDENT= Visual Analogue Mood Scale Confident item; ANXIOUS= Visual Analogue Mood Scale Anxious item; STRESS=

Visual Analogue Mood Scale Stressed item; BPSS-R BODY= Body Parts Satisfaction Scale- Revised Body Subscale; BPSS-R FACE= Body Parts Satisfaction Scale-Revised Face Subscale; MBSRQ-AE= Multidimensional Body Self Relations Questionnaire Appearance Evaluation Subscale; BAAR-PF= Beliefs About Attractiveness-Revised Physically Fit Factor; BAAR-AT= Beliefs About Attractiveness Revised - Attractive and Thin Factor; EAT-26= Eating Attitudes Test-26; BULIT-R= Bulimia Test-Revised.

Table 2B

Evaluation of Dropout: Chi-Square Tests for Categorical Variables

Variable	Category	Dropout N	Retain N	df	χ^2
RACE	White	158	164	1	.430
	Non-White	81	72		
INCOME	< 60,000	86	90	1	.619
	> 60,000	142	135		
MARITAL	Single	235	238	1	.315
	Married/Divorced	1	0		
LIVING	On Campus	189	177	1	.290
	Off Campus	50	59		
SORORITY	Member	46	51	1	.618
	Non-Member	190	188		

Note: RACE = Race/Ethnicity; INCOME = Annual Income for Family of Origin; MARITAL = Marital Status
LIVING = Current Living Arrangements; SORORITY = Current Involvement in a Sorority.

Table 3
Correlation Matrix of Measured Variables and Demographic Variables

Variable	1	2	3	4	5	6	7	8	9
1. INCOME-T1	1								
2. RACE-T1	-.158*	1							
3. MARITAL-T1	-.083	.025	1						
4. MARITAL -T2	-.039	.111	.497**	1					
5. MARITAL -T4	-.149*	.078	.706**	.704**	1				
6. HOURS-T1	.073	.052	-.030	-.039	-.071	1			
7. HOURS-T2	-.015	.023	-.031	-.043	-.043	.182**	1		
8. HOURS-T4	-.019	.102	-.028	-.025	-.039	.186**	.100	1	
9. GPA-T1	.013	-.004	-.082	-.050	-.035	.147*	.069	.063	1
10. GPA-T2	.004	-.098	-.096	-.063	-.105	.119	.095	.168*	.308**
11. GPA-T4	.110	-.041	-.047	-.022	-.066	.043	.043	.161*	.344**
12. BMI-T1	-.149*	.107	.063	-.004	.055	-.001	.006	-.030	-.038
13. BMI-T2	-.165*	.080	.084	.006	.072	.013	.024	-.048	-.053
14. BMI-T4	-.158*	.081	.059	.003	.083	.016	.026	-.041	-.056
15. IDEAL BMI-T1	-.164*	.131*	-.003	-.014	-.006	.023	.031	-.031	-.017
16. IDEAL BMI-T2	-.230**	.130*	-.031	-.042	-.029	.054	.094	-.048	-.054
17. IDEAL BMI-T4	-.217**	.132*	.045	-.009	.038	.019	.107	-.087	-.012
18. LIVING-T1	-.095	.152*	.186**	.209**	.225**	-.214**	-.083	-.054	.053
19. LIVING-T2	-.033	.107	.167*	.123	.124	-.205**	-.248**	-.135*	-.034
20. LIVING-T4	-.019	.057	-.032	.035	.060	-.151*	-.074	-.002	.202**
21. SORORITY-T1	-.248**	.103	.032	-.017	.046	-.043	-.001	-.041	-.200**
22. SORORITY -T2	-.218**	.084	.032	-.020	.045	-.028	.006	-.153*	-.163*
23. SORORITY -T4	-.210**	.060	.033	-.015	.046	-.003	.003	-.156*	-.160*
24. SDS-T1	-.018	-.022	-.076	.009	-.041	-.081	.062	-.044	-.033
25. PBI-MC-T1	.172**	-.137*	.061	.099	.087	-.009	.057	-.062	-.034
26. PBI-MO-T1	-.133*	.045	-.064	-.015	.002	.004	.075	-.016	-.006
27. PBI-FO-T1	-.148*	.152*	-.042	.058	.123	.006	.067	.064	-.007
28. PBI-FC-T1	.196**	-.032	.075	.071	.052	-.025	.034	-.107	-.046
29. PSS-T1	-.130	.009	.070	-.041	.044	.066	-.076	.111	.002
30. APPCOP-APP-T1	-.142*	.030	.058	-.085	-.028	.085	-.143*	.022	.022
31. APPCOP-COP-T1	-.099	-.007	.072	-.076	-.001	.084	-.089	.058	.101
32. APPCOP-AC-T1	-.025	.034	.125	.050	.099	.122	-.003	.081	-.019
33. APPCOP-FR-T1	-.042	-.103	-.001	-.069	-.059	.087	-.133*	.028	-.018
34. APPCOP-LA-T1	-.095	.030	.115	-.051	.019	.110	-.081	.029	.063
35. APPCOP-RO-T1	-.094	.046	-.087	-.130*	-.124	.020	-.081	.071	.016
36. APPCOP-WT-T1	-.099	.028	.076	-.012	.055	-.056	-.039	-.072	.123
37. ICSRLE-T1	-.138*	.078	.020	-.005	.061	.058	-.046	.051	-.022
38. ICSRLE-TP-T1	.015	.060	-.055	-.013	.000	-.042	.001	.085	.018
39. ICSRLE-DC-T1	.042	-.051	-.070	-.052	.011	-.016	-.071	.048	.078
40. ICSRLE-AC-T1	.056	.005	-.099	-.081	-.067	.009	.071	.000	-.012
41. ICSRLE-RP-T1	.106	-.159*	-.053	-.119	-.093	.128	-.043	.033	-.093
42. ICSRLE-AN-T1	-.046	-.028	-.054	-.018	.013	.063	-.082	.069	-.003
43. ICSRLE-GSM-T1	.051	-.072	-.064	-.065	-.028	.072	-.008	.072	.082
44. ICSRLE-FP-T1	.069	-.169**	-.073	-.118	-.104	.054	-.121	.088	-.094
45. SAD-T1	-.091	-.041	.023	-.101	-.051	.143*	-.068	.121	.004

Note. INCOME-T1 = Annual Income for Family of Origin Time 1; RACE-T1 = Race/Ethnicity Time 1; MARITAL-T1 = Marital Status Time 1; MARITAL-T2 = Marital Status Time 2; MARITAL-T4 = Marital Status Time 4; HOURS-T1 = Current Number of Credits Enrolled Time 1; HOURS-T2 = Current Number of Credits Enrolled Time 2; HOURS-T4 = Current Number of Credits Enrolled Time 4; GPA-T1 = High School Grade Point Average Time 1; GPA-T2 = College Grade Point Average Time 2; GPA-T4 = College Grade Point Average Time 4
(table continues)

Table 3 (continued).

Variable	1	2	3	4	5	6	7	8	9
46. SAD-T2	-.048	.017	-.013	-.062	-.066	.029	-.111	-.002	.011
47. SAD-T4	.002	.018	.056	-.021	.032	.114	-.062	.071	.029
48. HAPPY-T1	.112	.020	.081	.007	.004	-.114	.082	-.069	-.130
49. HAPPY-T2	.112	.005	-.143*	-.008	-.034	-.111	.004	-.030	-.013
50. HAPPY-T4	.098	.051	.098	.083	.085	-.144*	-.044	-.021	-.008
51. SHAME-T1	-.035	.085	-.036	.067	.146*	.058	-.074	.023	-.104
52. SHAME-T2	.120	.084	-.052	-.065	-.075	.149*	-.025	.038	.070
53. SHAME-T4	.059	.082	-.052	-.104	-.073	.141*	-.064	-.018	.012
54. GUILT-T1	-.034	.094	.028	.092	.141*	-.001	-.140*	.014	-.091
55. GUILT-T2	.063	.086	-.060	-.083	-.084	.108	.022	.017	.046
56. GUILT-T4	.031	-.040	.010	-.050	.015	.001	-.019	-.022	-.054
57. CONFIDENT-T1	.187**	-.020	.102	.071	.048	-.068	.143*	.027	-.104
58. CONFIDENT-T2	.051	.049	-.095	-.052	-.086	-.042	.095	.022	-.033
59. CONFIDENT-T4	-.041	.064	-.031	-.022	-.043	-.051	.127	-.028	-.049
60. ANXIOUS-T1	-.033	.040	-.129*	-.179**	-.145*	.035	-.075	-.056	-.035
61. ANXIOUS-T2	-.050	-.043	-.047	-.125	-.108	-.025	.005	-.101	.040
62. ANXIOUS-T4	-.020	.041	.074	-.083	-.017	-.004	.044	.076	.126
63. STRESSED-T1	-.095	.030	.011	-.039	.015	.047	-.075	.070	.096
64. STRESSED-T2	.056	-.034	.033	-.029	.002	.051	.054	-.003	.059
65. STRESSED-T4	.075	-.047	.032	-.063	.046	.071	-.014	-.039	.166*
66. PANASX-NA-T4	-.018	.083	-.052	-.028	.035	.047	-.027	-.006	.048
67. BAAR-PF-T1	.180**	-.097	-.143*	-.133*	-.147*	.130*	.008	.067	.049
68. BAAR-PF-T2	.177**	-.163*	-.226**	-.130*	-.142*	.008	.003	-.025	-.001
69. BAAR-PF-T4	.145*	-.002	-.009	.018	.020	-.025	.013	.076	.006
70. BAAR-AT-T1	.177*	-.110	-.136*	-.084	-.049	.032	.005	.072	.092
71. BAAR-AT-T2	.152*	-.111	-.094	-.058	-.028	.026	-.006	.033	.032
72. BAAR-AT-T4	.178**	-.129*	-.095	-.104	-.047	-.042	.017	.101	.050
73. MBSRQ-AE-T1	.124	-.052	.011	.016	-.029	-.007	.118	-.024	-.052
74. MBSRQ-AE-T2	.030	-.001	.105	.085	.068	.035	.089	-.008	-.030
75. MBSRQ-AE-T4	-.042	-.010	.042	.041	-.027	.025	.112	-.015	-.021
76. BPSS-BODY-T1	.030	-.009	-.098	-.100	-.145*	.041	.102	.028	-.027
77. BPSS-BODY-T2	-.015	.109	-.065	-.009	-.030	-.001	.079	.058	-.082
78. BPSS-BODY-T4	-.029	.102	-.050	.034	.011	-.018	.092	-.044	-.033
79. BPSS-FACE-T1	.162*	-.015	-.094	-.009	.010	.043	.181**	-.083	.086
80. BPSS-FACE-T2	.112	.070	-.072	-.013	-.036	-.021	.179**	-.021	.041
81. BPSS-FACE-T4	.094	-.008	-.110	-.034	-.045	-.036	.169**	-.055	.072
82. EAT-T1	.135*	-.102	-.032	-.086	-.060	-.018	.001	-.140*	.109
83. EAT-T2	.091	-.096	-.040	-.089	-.067	-.085	-.069	-.109	.045
84. EAT-T4	.136*	-.083	-.028	-.091	-.061	-.057	-.072	-.107	-.009
85. BULITR-T1	.038	-.028	-.021	-.081	-.041	-.018	-.066	-.011	.018
86. BULITR-T2	.092	-.016	-.033	-.102	-.067	-.044	-.115	-.041	-.011
87. BULITR-T4	.099	.034	-.010	-.069	-.031	-.036	-.087	.007	-.048

Note. 4; LIVING-T1 = Current Living Arrangements Time 1; LIVING-T2 = Current Living Arrangements Time 2; LIVING-T4 = Current Living Arrangements Time 4; SORORITY-T1 = Current Involvement in a Sorority Time 1; SORORITY-T2 = Current Involvement in a Sorority Time 2; SORORITY-T4 = Current Involvement in a Sorority Time 4; SDS-T1 = Social Desirability Scale Time 1; PBI-MC-T1 = Parental Bonding Instrument-Mother Care

(table continues)

Table 3 (continued).

Variable	10	11	12	13	14	15	16	17	18
1. INCOME-T1									
2. RACE-T1									
3. MARITAL-T1									
4. MARITAL -T2									
5. MARITAL -T4									
6. HOURS-T1									
7. HOURS-T2									
8. HOURS-T4									
9. GPA-T1									
10. GPA-T2	1								
11. GPA-T4	.732**	1							
12. BMI-T1	-.063	-.119	1						
13. BMI-T2	-.090	-.122	.939**	1					
14. BMI-T4	-.085	-.150*	.936**	.944**	1				
15. IDEAL BMI-T1	-.060	-.111	.887**	.844**	.830**	1			
16. IDEAL BMI-T2	-.125	-.141*	.786**	.838**	.781**	.875**	1		
17. IDEAL BMI-T4	-.131*	-.181**	.792**	.798**	.818**	.844**	.866**	1	
18. LIVING-T1	.016	.005	-.006	.037	.019	-.008	-.039	.028	1
19. LIVING-T2	-.065	-.069	-.007	.013	.021	-.035	-.056	-.006	.551**
20. LIVING-T4	.057	.160*	-.076	-.086	-.073	-.069	-.116	-.088	.337**
21. SORORITY-T1	-.062	-.160*	.178**	.182**	.171**	.167*	.199**	.207**	.066
22. SORORITY -T2	-.073	-.171*	.136*	.148*	.127	.134*	.174**	.150*	.067
23. SORORITY -T4	-.081	-.212**	.116	.110	.110	.116	.164**	.168**	.064
24. SDS-T1	.159*	.079	.137*	.121	.153*	.156*	.137*	.175**	.024
25. PBI-MC-T1	-.024	-.032	-.030	-.010	-.035	-.023	-.009	-.034	-.038
26. PBI-MO-T1	-.029	-.066	-.079	-.105	-.085	-.069	-.102	-.095	-.055
27. PBI-FO-T1	-.094	-.044	-.035	-.054	-.036	-.058	-.060	-.032	-.011
28. PBI-FC-T1	.131*	.038	-.037	-.049	-.052	.004	-.009	.004	.045
29. PSS-T1	-.163*	-.077	-.085	-.061	-.059	-.164*	-.130*	-.132*	.021
30. APPCOP-APP-T1	.081	.043	-.035	-.047	-.015	-.117	-.107	-.049	-.072
31. APPCOP-COP-T1	.059	.064	-.012	-.038	-.004	-.087	-.100	-.038	-.052
32. APPCOP-AC-T1	-.238**	-.143*	-.204**	-.209**	-.151*	-.287**	-.250**	-.205**	-.036
33. APPCOP-FR-T1	.245**	.170*	-.044	-.020	-.005	-.044	-.066	-.062	-.028
34. APPCOP-LA-T1	.073	.079	-.039	-.057	-.037	-.058	-.067	-.031	.006
35. APPCOP-RO-T1	.050	.064	-.022	-.057	-.061	-.048	-.044	-.043	-.085
36. APPCOP-WT-T1	.011	-.055	.175**	.159*	.180**	.058	.051	.153*	-.041
37. ICSRLE-T1	-.149*	-.080	-.043	-.046	-.007	-.105	-.098	-.082	-.058
38. ICSRLE-TP-T1	.025	.063	-.148*	-.183**	-.129*	-.107	-.128*	-.081	.052
39. ICSRLE-DC-T1	.052	.116	-.052	-.048	-.025	-.021	-.016	-.026	.065
40. ICSRLE-AC-T1	.014	.051	-.064	-.060	-.010	-.041	-.029	-.008	.005
41. ICSRLE-RP-T1	.037	-.012	-.123	-.139*	-.129*	-.114	-.167**	-.176**	.011
42. ICSRLE-AN-T1	-.029	.039	-.032	-.032	.021	.008	-.027	-.025	-.123
43. ICSRLE-GSM-T1	-.027	-.045	.074	.090	.089	.053	.077	.027	-.078
44. ICSRLE-FP-T1	-.009	.011	-.014	.016	.008	.002	.005	-.077	-.128*
45. SAD-T1	.000	.009	-.040	-.017	.005	-.072	-.098	-.093	-.044

Note. Scale Time 1; PBI-MO-T1 = Parental Bonding Instrument-Mother Overprotection Scale Time 1; PBI-FC-T1 = Parental Bonding Instrument-Father Care Scale Time 1; PBI-FO-T1 = Parental Bonding Instrument-Father Overprotection Scale Time 1; PSS-T1 = Perceived Stress Scale Time 1; APPCOP-APP-T1 = Appraisal of stress since beginning school Time 1; APPCOP-COP-T1 = Coping with experienced stress since beginning school

(table continues)

Table 3 (continued).

Variable	10	11	12	13	14	15	16	17	18
46. SAD-T2	.011	-.044	-.033	-.026	-.026	-.084	-.092	-.091	.084
47. SAD-T4	-.052	-.067	-.046	-.045	-.010	-.059	-.073	-.057	.053
48. HAPPY-T1	-.039	-.029	.028	.022	.023	.017	.041	.030	.053
49. HAPPY-T2	.080	.154*	-.051	-.042	-.064	-.018	.001	.002	-.048
50. HAPPY-T4	.013	.054	-.019	-.019	-.058	.001	.021	.019	-.018
51. SHAME-T1	-.179**	-.021	-.062	-.076	-.059	-.151*	-.131*	-.149*	-.009
52. SHAME-T2	.025	-.026	-.011	.012	.009	-.013	-.010	-.011	-.031
53. SHAME-T4	-.016	-.130	-.065	-.073	-.071	-.091	-.130*	-.057	.011
54. GUILT-T1	-.189**	-.084	-.010	-.007	-.007	-.104	-.112	-.093	-.035
55. GUILT-T2	.106	.067	-.027	-.029	-.020	-.057	-.057	-.045	.005
56. GUILT-T4	-.112	-.171**	-.024	-.030	.002	-.053	-.048	-.047	.047
57. CONFIDENT-T1	.068	.066	.088	.081	.070	.130*	.127	.124	-.027
58. CONFIDENT-T2	.035	.086	.034	.055	.038	.124	.176**	.123	-.064
59. CONFIDENT-T4	-.012	.021	-.002	.024	-.019	.029	.123	.077	-.054
60. ANXIOUS-T1	-.017	.070	-.073	-.057	-.049	-.094	-.081	-.112	-.020
61. ANXIOUS-T2	.031	-.003	-.067	-.094	-.071	-.094	-.055	-.061	-.070
62. ANXIOUS-T4	.138*	.126	.010	.033	.015	-.025	.039	-.020	-.071
63. STRESSED-T1	-.090	-.030	-.072	-.086	-.058	-.142*	-.130*	-.117	-.027
64. STRESSED-T2	-.012	-.012	-.091	-.110	-.060	-.108	-.101	-.047	.076
65. STRESSED-T4	.067	-.018	-.113	-.110	-.066	-.100	-.135*	-.095	.137*
66. PANASX-NA-T4	-.046	-.067	-.068	-.060	-.033	-.087	-.036	-.053	-.015
67. BAAR-PF-T1	.039	.048	-.076	-.060	-.033	-.146*	-.143*	-.121	-.172**
68. BAAR-PF-T2	.083	.055	-.107	-.116	-.051	-.148*	-.201**	-.137*	-.092
69. BAAR-PF-T4	.015	.029	-.105	-.086	-.091	-.182**	-.187**	-.170**	-.003
70. BAAR-AT-T1	.068	.034	-.072	-.069	-.010	-.119	-.175**	-.127	-.025
71. BAAR-AT-T2	.024	.037	-.028	-.025	.004	-.141*	-.198**	-.136*	-.071
72. BAAR-AT-T4	.105	.046	-.029	-.041	.006	-.086	-.149*	-.107	-.055
73. MBSRQ-AE-T1	.010	.033	-.259**	-.240**	-.248**	-.127	-.090	-.117	-.053
74. MBSRQ-AE-T2	.002	.017	-.284**	-.269**	-.280**	-.144*	-.053	-.121	.000
75. MBSRQ-AE-T4	-.074	-.011	-.248**	-.211**	-.296**	-.124	-.010	-.097	-.023
76. BPSS-BODY-T1	-.045	-.021	-.410**	-.372**	-.416**	-.230**	-.127	-.223**	-.049
77. BPSS-BODY-T2	.033	.043	-.349**	-.379**	-.388**	-.224**	-.150*	-.207**	-.059
78. BPSS-BODY-T4	.007	.022	-.288**	-.297**	-.334**	-.174**	-.078	-.137*	-.063
79. BPSS-FACE-T1	-.063	-.058	.050	.068	.037	.112	.113	.093	-.009
80. BPSS-FACE-T2	-.096	-.001	.025	.017	.005	.091	.108	.065	-.099
81. BPSS-FACE-T4	.005	-.044	-.001	-.001	-.019	.074	.113	.091	-.065
82. EAT-T1	-.019	.000	-.033	-.045	-.026	-.091	-.084	-.046	-.088
83. EAT-T2	-.078	-.028	.038	.005	.010	-.032	-.034	.004	.035
84. EAT-T4	.019	.015	-.005	-.029	.002	-.069	-.064	-.038	-.022
85. BULITR-T1	-.008	-.025	.101	.076	.101	-.042	-.060	-.019	-.015
86. BULITR-T2	-.088	-.049	.079	.093	.110	-.031	-.066	-.015	.004
87. BULITR-T4	-.020	.005	.025	.019	.051	-.077	-.108	-.088	.044

Note. question Time 1; APPCOP-AC-T1 = Appraisal of academic demands and coping with experienced academic demands since beginning school Time 1; APPCOP-FR-T1 = Appraisal of new friendship and coping with experienced new friendship demands since beginning school Time 1; APPCOP-LA-T1 = Appraisal of living arrangement demands and coping with experienced living arrangement demands since beginning school Time 1;

(table continues)

Table 3 (continued).

Variable	19	20	21	22	23	24	25	26	27
1. INCOME-T1									
2. RACE-T1									
3. MARITAL-T1									
4. MARITAL -T2									
5. MARITAL -T4									
6. HOURS-T1									
7. HOURS-T2									
8. HOURS-T4									
9. GPA-T1									
10. GPA-T2									
11. GPA-T4									
12. BMI-T1									
13. BMI-T2									
14. BMI-T4									
15. IDEAL BMI-T1									
16. IDEAL BMI-T2									
17. IDEAL BMI-T4									
18. LIVING-T1									
19. LIVING-T2	1								
20. LIVING-T4	.209**	1							
21. SORORITY-T1	.109	-.465**	1						
22. SORORITY -T2	.174**	-.499**	.905**	1					
23. SORORITY -T4	.168	-.440**	.776**	.838**	1				
24. SDS-T1	-.022	.015	.020	-.017	.004	1			
25. PBI-MC-T1	-.121	.094	-.130*	-.122	-.123	.092	1		
26. PBI-MO-T1	-.111	-.145*	.134*	.159*	.142*	-.196**	-.460**	1	
27. PBI-FO-T1	-.065	-.078	.152*	.175**	.132*	-.172**	-.233**	.462**	1
28. PBI-FC-T1	.027	.064	-.120	-.151*	-.092	.046	.275**	-.246**	-.354**
29. PSS-T1	.122	-.087	.051	.079	.048	-.362**	-.232**	.289**	.247**
30. APPCOP-APP-T1	.063	-.116	.003	.021	.037	-.243**	-.197**	.198**	.170**
31. APPCOP-COP-T1	.011	-.083	-.014	.002	.005	-.265**	-.188**	.230**	.178**
32. APPCOP-AC-T1	-.017	-.035	-.131*	-.112	-.114	-.211**	-.032	.154*	.187**
33. APPCOP-FR-T1	-.003	-.142*	.168**	.184**	.156*	-.009	-.092	.083	.023
34. APPCOP-LA-T1	.117	-.032	-.002	.044	.076	-.158*	-.153*	.201**	.118
35. APPCOP-RO-T1	.027	-.101	.001	.006	.029	-.241**	-.091	.107	.108
36. APPCOP-WT-T1	-.023	.011	-.081	-.112	-.112	-.158*	-.192**	.111	.112
37. ICSRLE-T1	.024	-.067	.019	.043	.023	-.319**	-.170**	.274**	.261**
38. ICSRLE-TP-T1	-.016	-.022	.038	.070	.053	.021	.020	.009	-.025
39. ICSRLE-DC-T1	.049	.008	.083	.074	.033	.180**	.022	-.077	-.039
40. ICSRLE-AC-T1	.004	-.025	.056	.059	.015	.090	.019	-.048	.003
41. ICSRLE-RP-T1	.010	-.125	.070	.092	.097	-.103	.063	.007	.026
42. ICSRLE-AN-T1	-.104	-.078	.044	.073	.018	.022	-.029	.028	.063
43. ICSRLE-GSM-T1	-.137*	-.148*	.110	.113	.059	.132*	-.037	-.008	.022
44. ICSRLE-FP-T1	-.074	-.116	-.011	-.001	-.029	-.042	-.054	-.003	.015
45. SAD-T1	.007	-.078	.091	.119	.088	-.227**	-.165*	.188**	.066

Note. APPCOP-ROM-T1 = Appraisal of romantic demands and coping with experienced romantic demands since beginning school Time 1; APPCOP-WT-T1 = Appraisal of weight gain and coping with experienced weight gain since beginning school Time 1; ICSRLE-T1 = Inventory of College Students' Recent Life Experiences Total Time 1; ICSRLE-TP-T1 = Inventory of College Students' Recent Life Experiences-Time Pressure Scale Time 1;

(table continues)

Table 3 (continued).

Variable	19	20	21	22	23	24	25	26	27
46. SAD-T2	.060	-.046	.076	.096	.054	-.184**	-.225**	.155*	.093
47. SAD-T4	.094	-.060	.016	.057	.062	-.102	-.246**	.093	.060
48. HAPPY-T1	-.104	.102	-.186**	-.214**	-.198**	.192**	.241**	-.151*	-.150*
49. HAPPY-T2	-.104	.105	-.140*	-.175**	-.142*	.148*	.238**	-.100	-.175**
50. HAPPY-T4	-.084	.115	-.124	-.182**	-.164*	.111	.223**	-.135*	-.192**
51. SHAME-T1	-.033	.019	.037	.046	.010	-.094	-.123	.125	.249**
52. SHAME-T2	-.007	-.080	.035	.085	.111	-.286**	-.183**	.156*	.210**
53. SHAME-T4	.028	-.140*	-.040	.050	.053	-.167**	-.143*	.121	.216**
54. GUILT-T1	-.067	.008	.097	.072	.034	-.255**	-.110	.171**	.219**
55. GUILT-T2	.003	-.116	.069	.122	.150*	-.184**	-.146*	.161*	.232**
56. GUILT-T4	.165*	-.143*	.069	.130*	.124	-.242**	-.156*	.145*	.174**
57. CONFIDENT-T1	-.084	-.026	.048	-.005	-.010	.176**	.177**	-.088	-.134*
58. CONFIDENT-T2	-.184**	.057	-.019	-.050	-.045	.183**	.169**	-.176**	-.220**
59. CONFIDENT-T4	-.124	.056	.057	.000	.011	.186**	.201**	-.114	-.176**
60. ANXIOUS-T1	.001	-.034	.091	.139*	.057	-.159*	-.017	.103	.120
61. ANXIOUS-T2	-.012	-.047	-.063	.022	.067	-.068	-.205**	.213**	.086
62. ANXIOUS-T4	-.017	-.033	-.060	.008	-.016	-.157*	-.062	.078	.111
63. STRESSED-T1	.033	.017	-.045	-.012	-.035	-.202**	-.074	.156*	.166*
64. STRESSED-T2	.000	-.086	.049	.041	.045	-.102	-.104	.130*	.076
65. STRESSED-T4	.108	-.004	-.061	.000	.006	-.180**	-.041	.044	-.054
66. PANASX-NA-T4	.056	-.109	-.004	.055	.035	-.281**	-.195**	.220**	.251**
67. BAAR-PF-T1	-.135*	-.004	-.140*	-.088	-.160*	-.127	-.074	-.024	.044
68. BAAR-PF-T2	-.036	.055	-.096	-.056	-.129*	-.102	-.033	.020	-.026
69. BAAR-PF-T4	-.031	.018	-.129*	-.076	-.104	-.279**	-.034	-.037	.117
70. BAAR-AT-T1	-.040	.066	-.149*	-.103	-.146*	-.098	-.090	-.006	-.010
71. BAAR-AT-T2	-.043	.029	-.145*	-.081	-.141*	-.225**	-.066	.009	.074
72. BAAR-AT-T4	-.065	.030	-.104	-.068	-.119	-.158*	-.129*	.013	.080
73. MBSRQ-AE-T1	-.006	-.019	.045	.061	.099	.095	.162*	-.070	-.105
74. MBSRQ-AE-T2	-.032	-.090	.118	.127	.130*	.071	.166*	-.068	-.077
75. MBSRQ-AE-T4	-.097	-.011	.077	.066	.103	.100	.164*	-.050	-.061
76. BPSS-BODY-T1	.052	-.058	.067	.111	.121	.035	.109	-.043	-.121
77. BPSS-BODY-T2	-.019	-.102	.161*	.134*	.144*	-.013	.090	.005	-.045
78. BPSS-BODY-T4	-.057	-.058	.147*	.115	.140*	.069	.061	.000	-.041
79. BPSS-FACE-T1	-.081	.022	-.020	-.009	-.017	.091	.175**	-.072	-.104
80. BPSS-FACE-T2	-.057	-.044	.027	-.002	.000	.067	.221**	-.063	-.107
81. BPSS-FACE-T4	-.068	-.006	.031	-.006	-.009	.116	.142*	-.035	-.105
82. EAT-T1	-.078	.007	-.261**	-.185**	-.156*	-.049	.018	.027	.030
83. EAT-T2	.013	.054	-.174**	-.132*	-.070	-.062	-.155*	.126	.039
84. EAT-T4	-.025	-.019	-.153*	-.085	-.069	-.130*	-.161*	.106	.057
85. BULITR-T1	.020	.045	-.159*	-.139*	-.135*	-.209**	-.148*	.080	.155*
86. BULITR-T2	.060	.085	-.122	-.101	-.107	-.187**	-.223**	.149*	.140*
87. BULITR-T4	.054	.046	-.156*	-.085	-.114	-.184**	-.213**	.154*	.181**

Note. ICSRLE-DC-T1 = Inventory of College Students' Recent Life Experiences-Developmental Challenge Scale Time 1; ICSRLE-AC-T1 = Inventory of College Students' Recent Life Experiences-Academic Alienation Scale Time 1; ICSRLE-RP-T1 = Inventory of College Students' Recent Life Experiences-Romantic Problems Scale Time 1; ICSRLE-AN-T1 = Inventory of College Students' Recent Life Experiences-Assorted Annoyances Scale Time 1; (table continues)

Table 3 (continued).

Variable	28	29	30	31	32	33	34	35	36
1. INCOME-T1									
2. RACE-T1									
3. MARITAL-T1									
4. MARITAL -T2									
5. MARITAL -T4									
6. HOURS-T1									
7. HOURS-T2									
8. HOURS-T4									
9. GPA-T1									
10. GPA-T2									
11. GPA-T4									
12. BMI-T1									
13. BMI-T2									
14. BMI-T4									
15. IDEAL BMI-T1									
16. IDEAL BMI-T2									
17. IDEAL BMI-T4									
18. LIVING-T1									
19. LIVING-T2									
20. LIVING-T4									
21. SORORITY-T1									
22. SORORITY -T2									
23. SORORITY -T4									
24. SDS-T1									
25. PBI-MC-T1									
26. PBI-MO-T1									
27. PBI-FO-T1									
28. PBI-FC-T1	1								
29. PSS-T1	-.235**	1							
30. APPCOP-APP-T1	-.130*	.517**	1						
31. APPCOP-COP-T1	-.136*	.551**	.893**	1					
32. APPCOP-AC-T1	-.096	.565**	.482**	.479**	1				
33. APPCOP-FR-T1	-.145*	.197**	.509**	.548**	.107	1			
34. APPCOP-LA-T1	-.071	.344**	.646**	.621**	.212**	.231	1		
35. APPCOP-RO-T1	-.050	.299**	.642**	.642**	.178**	.219**	.231**	1	
36. APPCOP-WT-T1	-.050	.284**	.563**	.545**	.176**	.026	.201**	.215**	1
37. ICSRLE-T1	-.210*	.681**	.631**	.617**	.567**	.240**	.358**	.434**	.343**
38. ICSRLE-TP-T1	.002	.109	.114	.133	.115	.105	.087	.077	.005
39. ICSRLE-DC-T1	-.020	.088	.030	.057	.117	.021	.049	.014	-.040
40. ICSRLE-AC-T1	-.074	.048	.025	.036	.136*	.037	-.002	.015	-.056
41. ICSRLE-RP-T1	.015	.052	.000	-.023	.091	.037	-.028	.040	-.144*
42. ICSRLE-AN-T1	-.074	.058	-.012	-.002	.048	.022	.040	-.011	-.101
43. ICSRLE-GSM-T1	.000	-.037	-.138*	-.107	-.034	-.050	-.122	-.100	-.052
44. ICSRLE-FP-T1	.059	.007	.002	.001	-.033	.064	-.001	.052	-.086
45. SAD-T1	-.207**	.585**	.476**	.506**	.363**	.424**	.271**	.305**	.155*

Note. ICSRLE-GSM-T1 = Inventory of College Students' Recent Life Experiences-General Social Mistreatment Scale Time 1; ICSRLE-FP = Inventory of College Students' Recent Life Experiences-Friendship Problems Scale Time 1; SAD-T1 = Visual Analogue Mood Scale Sad or Depressed item Time 1; SAD-T2 = Visual Analogue Mood Scale Sad or Depressed item Time 2; SAD-T4 = Visual Analogue Mood Scale Sad or Depressed item Time 4
(table continues)

Table 3 (continued).

Variable	28	29	30	31	32	33	34	35	36
46. SAD-T2	-.197**	.403**	.293**	.277**	.219**	.197**	.154*	.149*	.163*
47. SAD-T4	-.064	.312**	.250**	.222**	.232**	.195**	.091	.174**	.055
48. HAPPY-T1	.280**	-.471**	-.306**	-.342**	-.126	-.369**	-.172**	-.217**	-.086
49. HAPPY-T2	.260**	-.369**	-.189**	-.189**	-.111	-.160*	-.085	-.118	-.101
50. HAPPY-T4	.277**	-.266**	-.107	-.116	-.134*	-.081	-.054	-.086	-.004
51. SHAME-T1	-.169**	.340**	.185**	.202**	.290**	.063	.096	.069	.122
52. SHAME-T2	-.142*	.271**	.162*	.162*	.059	.074	.153*	.092	.101
53. SHAME-T4	-.090	.254**	.216**	.229**	.157*	.200**	.071	.138*	.119
54. GUILT-T1	-.218**	.422**	.229**	.256**	.342**	.104	.068	.119	.161*
55. GUILT-T2	-.146*	.167**	.165*	.137*	-.010	.071	.116	.058	.193**
56. GUILT-T4	-.075	.317**	.112	.161*	.115	.094	.022	.087	.105
57. CONFIDENT-T1	.312**	-.483**	-.359**	-.362**	-.287**	-.231**	-.146*	-.231**	-.219**
58. CONFIDENT-T2	.309**	-.351**	-.238**	-.260**	-.164*	-.151*	-.095	-.175**	-.173**
59. CONFIDENT-T4	.149*	-.282**	-.175**	-.193**	-.139*	-.053	-.088	-.118	-.165*
60. ANXIOUS-T1	-.052	.354**	.254**	.272**	.314**	.159*	.147*	.172**	.054
61. ANXIOUS-T2	-.125	.329**	.297**	.285**	.203**	.080	.250**	.125	.232**
62. ANXIOUS-T4	-.105	.187**	.232**	.200**	.137*	.140*	.095	.180**	.104
63. STRESSED-T1	-.145*	.664**	.457**	.457**	.580**	.156*	.253**	.273**	.214**
64. STRESSED-T2	-.024	.379**	.232**	.209**	.313**	.059	.104	.108	.137*
65. STRESSED-T4	.065	.312**	.336**	.306**	.240**	.179**	.219**	.151*	.202**
66. PANASX-NA-T4	-.211**	.386**	.295**	.283**	.245**	.164*	.071	.216**	.200**
67. BAAR-PF-T1	-.096	.221**	.197**	.213**	.150*	.141*	.056	.103	.182**
68. BAAR-PF-T2	-.042	.131*	.192**	.157*	.110	.095	.037	.073	.217**
69. BAAR-PF-T4	-.122	.187**	.209**	.199**	.148*	.103	.053	.114	.208**
70. BAAR-AT-T1	-.079	.181**	.224**	.264**	.165*	.125	.067	.114	.272**
71. BAAR-AT-T2	-.110	.226**	.212**	.187**	.210**	.050	.027	.109	.232**
72. BAAR-AT-T4	-.143*	.139*	.149*	.160*	.093	.125	.030	.064	.159*
73. MBSRQ-AE-T1	.230**	-.360**	-.291**	-.315**	-.197**	-.157*	-.035	-.129*	-.404**
74. MBSRQ-AE-T2	.248**	-.283**	-.237**	-.243**	-.124	-.098	-.043	-.087	-.367**
75. MBSRQ-AE-T4	.155*	-.296**	-.206**	-.216**	-.153*	-.066	-.087	-.033	-.307**
76. BPSS-BODY-T1	.172**	-.273**	-.327**	-.336**	-.144*	-.167**	-.078	-.115	-.484**
77. BPSS-BODY-T2	.156*	-.226**	-.216**	-.241**	-.108	-.147*	-.054	-.024	-.355**
78. BPSS-BODY-T4	.143*	-.312**	-.283**	-.293**	-.168**	-.191**	-.117	-.117	-.282**
79. BPSS-FACE-T1	.156*	-.387**	-.277**	-.267**	-.203**	-.273**	-.089	-.096	-.186**
80. BPSS-FACE-T2	.193**	-.342**	-.245**	-.262**	-.174**	-.208**	-.074	-.089	-.237**
81. BPSS-FACE-T4	.206**	-.368**	-.258**	-.236**	-.194**	-.249**	-.076	-.109	-.144*
82. EAT-T1	-.018	.256**	.240**	.280**	.206**	.003	.091	.160*	.333**
83. EAT-T2	-.115	.268**	.242**	.275**	.196**	.059	.094	.158*	.282**
84. EAT-T4	-.025	.272**	.310**	.328**	.250**	.113	.134*	.167**	.316**
85. BULITR-T1	-.097	.421**	.385**	.393**	.299**	.048	.108	.195**	.540**
86. BULITR-T2	-.157*	.397**	.338**	.355**	.271**	.111	.110	.128*	.446**
87. BULITR-T4	-.116	.414**	.347**	.357**	.300**	.124	.144*	.135*	.390**

Note. 4; HAPPY-T1 = Visual Analogue Mood Scale Happy item Time 1; HAPPY-T2 = Visual Analogue Mood Scale Happy item Time 2; HAPPY-T4 = Visual Analogue Mood Scale Happy item Time 4; SHAME-T1 = Visual Analogue Mood Scale Shameful Time 1; SHAME-T2 = Visual Analogue Mood Scale Shameful Time 2; SHAME-T4 = Visual Analogue Mood Scale Shameful Time 4; GUILT-T1 = Visual Analogue Mood Scale Guilty item Time
(table continues)

Table 3 (continued).

Variables	37	38	39	40	41	42	43	44	45
1. INCOME-T1									
2. RACE-T1									
3. MARITAL-T1									
4. MARITAL -T2									
5. MARITAL -T4									
6. HOURS-T1									
7. HOURS-T2									
8. HOURS-T4									
9. GPA-T1									
10. GPA-T2									
11. GPA-T4									
12. BMI-T1									
13. BMI-T2									
14. BMI-T4									
15. IDEAL BMI-T1									
16. IDEAL BMI-T2									
17. IDEAL BMI-T4									
18. LIVING-T1									
19. LIVING-T2									
20. LIVING-T4									
21. SORORITY-T1									
22. SORORITY -T2									
23. SORORITY -T4									
24. SDS-T1									
25. PBI-MC-T1									
26. PBI-MO-T1									
27. PBI-FO-T1									
28. PBI-FC-T1									
29. PSS-T1									
30. APPCOP-APP-T1									
31. APPCOP-COP-T1									
32. APPCOP-AC-T1									
33. APPCOP-FR-T1									
34. APPCOP-LA-T1									
35. APPCOP-RO-T1									
36. APPCOP-WT-T1									
37. ICSRLE-T1	1								
38. ICSRLE-TP-T1	.123	1							
39. ICSRLE-DC-T1	.062	.638**	1						
40. ICSRLE-AC-T1	.067	.487**	.621**	1					
41. ICSRLE-RP-T1	-.005	.228**	.305**	.241**	1				
42. ICSRLE-AN-T1	.051	.370**	.463**	.371**	.241**	1			
43. ICSRLE-GSM-T1	-.035	.233**	.321**	.308**	.215**	.504**	1		
44. ICSRLE-FP-T1	-.055	.127	.199**	.192**	.233**	.468**	.494**	1	
45. SAD-T1	.540**	.193**	.088	.070	.126	.019	-.034	.029	1

Note. 1; GUILT- T2 = Visual Analogue Mood Scale Guilty item Time 2; GUILT-T4 = Visual Analogue Mood Scale Guilty item Time 4; CONFIDENT-T1 = Visual Analogue Mood Scale Confident item Time 1; CONFIDENT-T2 = Visual Analogue Mood Scale Confident item Time 2; CONFIDENT-T4 = Visual Analogue

(table continues)

Table 3 (continued).

Variables	37	38	39	40	41	42	43	44	45
46. SAD-T2	.289**	.005	.021	-.022	.007	-.007	-.037	-.080	.388**
47. SAD-T4	.266**	-.004	-.003	-.019	.048	.010	-.050	-.044	.286**
48. HAPPY-T1	-.283**	-.112	-.068	-.041	-.053	-.037	.034	-.030	-.499**
49. HAPPY-T2	-.151*	-.044	-.064	-.072	-.024	-.050	-.043	-.091	-.229**
50. HAPPY-T4	-.202**	-.008	-.060	-.139*	-.065	-.042	-.025	-.023	-.174**
51. SHAME-T1	.396**	.079	.038	-.014	.073	.048	.011	-.041	.304**
52. SHAME-T2	.294**	.048	-.023	.047	.053	.040	.091	.117	.285**
53. SHAME-T4	.240**	.061	-.067	-.017	.090	-.046	-.019	.031	.251**
54. GUILT-T1	.466**	.003	-.008	-.011	.050	.031	.002	-.108	.422**
55. GUILT-T2	.164*	.022	-.014	-.005	-.008	-.018	.016	.022	.127*
56. GUILT-T4	.227**	.048	.013	-.011	.019	.001	.061	.100	.218**
57. CONFIDENT-T1	-.347**	-.078	-.002	.010	-.008	-.003	.130*	.104	-.395**
58. CONFIDENT-T2	-.263**	-.084	-.050	-.020	-.040	-.051	-.014	-.033	-.194**
59. CONFIDENT-T4	-.266**	.030	.023	.044	.004	-.038	.027	-.004	-.152*
60. ANXIOUS-T1	.374**	.003	.036	-.003	.032	.057	.065	.091	.232**
61. ANXIOUS-T2	.272**	.018	.019	-.017	-.061	-.004	-.003	-.035	.260**
62. ANXIOUS-T4	.195**	.091	.042	.021	.077	.076	.012	.026	.139*
63. STRESSED-T1	.595**	.166*	.132*	.102	.023	.059	-.017	-.065	.476**
64. STRESSED-T2	.339**	.106	.066	.030	-.023	.000	.003	-.070	.279**
65. STRESSED-T4	.376**	.126	.099	.054	.060	-.030	.016	-.105	.297**
66. PANASX-NA-T4	.435**	.048	.049	.033	-.069	.007	.005	-.048	.263**
67. BAAR-PF-T1	.218**	.047	.003	-.052	-.057	.073	.062	.073	.216**
68. BAAR-PF-T2	.138*	.049	-.002	-.024	-.011	.123	.022	.128*	.094
69. BAAR-PF-T4	.204**	.014	-.071	-.083	-.128*	-.012	-.104	-.030	.124
70. BAAR-AT-T1	.222**	.008	.017	.027	-.095	.093	.019	.053	.128*
71. BAAR-AT-T2	.216**	.002	-.037	-.067	-.071	.057	.024	.100	.110
72. BAAR-AT-T4	.150*	.014	-.007	-.035	-.082	.008	-.035	.005	.081
73. MBSRQ-AE-T1	-.301**	.008	.022	.049	.016	.058	-.028	.054	-.293**
74. MBSRQ-AE-T2	-.267**	-.004	.001	.039	.070	.005	.032	.085	-.163*
75. MBSRQ-AE-T4	-.282**	-.076	-.088	-.052	-.039	-.055	-.059	.072	-.216**
76. BPSS-BODY-T1	-.284**	-.039	-.025	-.011	.054	-.030	-.053	.023	-.179**
77. BPSS-BODY-T2	-.217**	-.019	-.049	-.024	.103	-.011	-.047	.015	-.125
78. BPSS-BODY-T4	-.295**	-.083	-.061	-.069	-.007	-.054	-.032	-.019	-.249**
79. BPSS-FACE-T1	-.245**	-.018	.000	-.011	-.025	-.019	-.035	-.043	-.311**
80. BPSS-FACE-T2	-.271**	-.033	-.060	-.023	-.053	-.074	-.089	-.021	-.265**
81. BPSS-FACE-T4	-.338**	-.106	-.037	-.017	-.060	-.047	.004	.040	-.333**
82. EAT-T1	.262**	.084	.071	.002	-.093	.007	-.010	-.003	.140*
83. EAT-T2	.225**	.044	.029	-.069	-.074	-.017	-.042	-.045	.185**
84. EAT-T4	.271**	.092	.052	-.021	-.076	.000	.059	.076	.238**
85. BULITR-T1	.416**	.029	.015	-.002	-.056	-.003	.028	-.015	.296**
86. BULITR-T2	.388**	.056	.037	-.008	-.056	.037	.041	-.029	.322**
87. BULITR-T4	.407**	.095	.061	.043	-.025	.039	.073	.012	.347**

Note. Mood Scale Confident item Mood Scale Anxious item Time 2; ANXIOUS-T4 = Visual Analogue Mood Scale Anxious item Time 4; STRESSED-T1 = Visual Analogue Mood Scale Stressed item Time 1; STRESSED-T2 = Visual Analogue Mood Scale Stressed item Time 2; STRESSED-T4 = Visual Analogue Mood Scale Stressed item Time 4; PANASX-NA-Time 4; ANXIOUS-T1 = Visual Analogue Mood Scale Anxious item Time 1;

(table continues)

Table 3 (continued).

Variable	46	47	48	49	50	51	52	53	54
46. SAD-T2	1								
47. SAD-T4	.455**	1							
48. HAPPY-T1	-.287**	-.280**	1						
49. HAPPY-T2	-.524**	-.314**	.398**	1					
50. HAPPY-T4	-.268**	-.552**	.295**	.479**	1				
51. SHAME-T1	.182**	.153*	-.136*	-.088	-.120	1			
52. SHAME-T2	.319**	.236**	-.196**	-.200**	-.237**	.161*	1		
53. SHAME-T4	.403**	.338**	-.188**	-.244**	-.292**	.275**	.444**	1	
54. GUILT-T1	.271**	.240**	-.234**	-.101	-.152*	.692**	.241**	.234**	1
55. GUILT-T2	.350**	.195**	-.074	-.201**	-.247**	.180**	.555**	.340**	.196**
56. GUILT-T4	.325**	.302**	-.197**	-.274**	-.191**	.176**	.329**	.531**	.264**
57. CONFIDENT-T1	-.340**	-.204**	.512**	.324**	.201**	-.216	-.074	-.240**	-.228**
58. CONFIDENT-T2	-.447**	-.210**	.263**	.543**	.322**	-.125	-.175**	-.234**	-.186**
59. CONFIDENT-T4	-.267**	-.421**	.195**	.283**	.504**	-.110	-.263**	-.293**	-.185**
60. ANXIOUS-T1	.163*	.181**	-.088	-.064	-.170**	.118	.169**	.150*	.226**
61. ANXIOUS-T2	.392**	.240**	-.122	-.180**	-.167**	.107	.208**	.167**	.164*
62. ANXIOUS-T4	.208**	.290**	-.047	-.080	-.100	.077	.249**	.235**	.187**
63. STRESSED-T1	.371**	.255**	-.250**	-.236**	-.124	.218**	.156*	.110	.322**
64. STRESSED-T2	.560**	.372**	-.064	-.256**	-.177**	.081	.216**	.244**	.145*
65. STRESSED-T4	.326**	.495**	-.163*	-.196**	-.295**	.122	.205**	.264**	.206**
66. PANASX-NA-T4	.363**	.397**	-.236**	-.248**	-.353**	.238**	.414**	.454**	.311**
67. BAAR-PF-T1	.158*	.149*	-.096	-.141*	-.152*	.252**	.234**	.188**	.231**
68. BAAR-PF-T2	.148*	.097	-.024	-.022	-.035	.143*	.215**	.130*	.085
69. BAAR-PF-T4	.132*	.088	-.082	-.093	-.120	.287**	.242**	.194**	.276**
70. BAAR-AT-T1	.213**	.175**	-.060	-.120	-.127*	.082	.190**	.181**	.105
71. BAAR-AT-T2	.170**	.089	-.120	-.139*	-.143*	.225**	.217**	.174**	.183**
72. BAAR-AT-T4	.139*	.124	-.057	-.128*	-.148*	.209**	.200**	.233**	.182**
73. MBSRQ-AE-T1	-.331**	-.161*	.220**	.209**	.161*	-.197**	-.148*	-.258**	-.180**
74. MBSRQ-AE-T2	-.275**	-.126	.177**	.259**	.188**	-.137*	-.140*	-.177**	-.131*
75. MBSRQ-AE-T4	-.270**	-.179**	.170**	.202**	.226**	-.215**	-.221**	-.232**	-.177**
76. BPSS-BODY-T1	-.219**	-.142*	.144*	.173**	.098	-.170**	-.141*	-.150*	-.187**
77. BPSS-BODY-T2	-.203**	-.150*	.111	.296**	.194**	-.070	-.156*	-.157*	-.080
78. BPSS-BODY-T4	-.266**	-.197**	.200**	.273**	.218**	-.131*	-.224**	-.263**	-.144*
79. BPSS-FACE-T1	-.181**	-.144*	.195**	.236**	.143*	-.163*	-.107	-.174**	-.182**
80. BPSS-FACE-T2	-.255**	-.232**	.173**	.278**	.237**	-.124	-.222**	-.234**	-.146*
81. BPSS-FACE-T4	-.256**	-.259**	.241**	.318**	.282**	-.286**	-.246**	-.330**	-.285**
82. EAT-T1	.203**	.155*	.024	-.017	-.058	.180**	.160*	.212**	.116
83. EAT-T2	.351**	.267**	-.035	-.083	-.114	.267**	.246**	.286**	.258**
84. EAT-T4	.247**	.217**	-.051	-.083	-.141*	.281**	.310**	.289**	.217**
85. BULITR-T1	.321**	.255**	-.077	-.097	-.164*	.358**	.286**	.340**	.352**
86. BULITR-T2	.392**	.260**	-.124	-.152*	-.183**	.323**	.366**	.345**	.371**
87. BULITR-T4	.328**	.256**	-.120	-.143*	-.205**	.372**	.375**	.388**	.319**

Note. ANXIOUS-T2 = Visual Analogue T4 = Positive and Negative Affect Scheduled Expanded Version-Negative Affect Scale Time 4; BAAR-PF-T1= Beliefs About Attractiveness-Revised Physically Fit Factor Time 1; BAAR-PF-T2 = Beliefs About Attractiveness-Revised Physically Fit Factor Time 2; BAAR-PF-T4 = Beliefs About Attractiveness-Revised Physically Fit Factor Time 4; BAAR-AT-T1 = Beliefs About Attractiveness Revised -

(table continues)

Table 3 (continued).

Variable	55	56	57	58	59	60	61	62	63
46. SAD-T2									
47. SAD-T4									
48. HAPPY-T1									
49. HAPPY-T2									
50. HAPPY-T4									
51. SHAME-T1									
52. SHAME-T2									
53. SHAME-T4									
54. GUILT-T1									
55. GUILT-T2	1								
56. GUILT-T4	.324**	1							
57. CONFIDENT-T1	-.072	-.192**	1						
58. CONFIDENT-T2	-.246**	-.274**	.457**	1					
59. CONFIDENT-T4	-.269**	-.318**	.320**	.470**	1				
60. ANXIOUS-T1	.072	.221**	-.141*	-.109	-.132*	1			
61. ANXIOUS-T2	.174**	.166*	-.142*	-.135*	-.151*	.335**	1		
62. ANXIOUS-T4	.224**	.253**	-.070	-.094	-.158*	.243**	.435**	1	
63. STRESSED-T1	.030	.158*	-.369**	-.268**	-.166*	.448**	.322**	.264**	1
64. STRESSED-T2	.292**	.241**	-.215**	-.343**	-.227**	.310**	.505**	.295**	.461**
65. STRESSED-T4	.222**	.275**	-.207**	-.176**	-.283**	.274**	.286**	.350**	.385**
66. PANASX-NA-T4	.328**	.514**	-.157*	-.271**	-.385**	.262**	.300**	.356**	.359**
67. BAAR-PF-T1	.197**	.186**	-.162*	-.124	-.189**	.131*	.190**	.127*	.160*
68. BAAR-PF-T2	.160*	.124	-.129*	-.078	-.109	.177**	.133*	.119	.102
69. BAAR-PF-T4	.259**	.182**	-.114	-.061	-.147*	.079	.167**	.228**	.150*
70. BAAR-AT-T1	.142*	.148*	-.129*	-.135*	-.142*	.211**	.243**	.098	.164*
71. BAAR-AT-T2	.167**	.169**	-.144*	-.117	-.159*	.184**	.120	.111	.194**
72. BAAR-AT-T4	.211**	.264**	-.056	-.083	-.204**	.133*	.141*	.212**	.130*
73. MBSRQ-AE-T1	-.143*	-.162*	.470**	.436**	.275**	-.197**	-.195**	-.121	-.319**
74. MBSRQ-AE-T2	-.172**	-.133*	.419**	.506**	.320**	-.081	-.066	-.088	-.278**
75. MBSRQ-AE-T4	-.199**	-.255**	.312**	.402**	.494**	-.102	-.076	-.097	-.224**
76. BPSS-BODY-T1	-.140*	-.100	.318**	.327**	.233**	-.098	-.134*	-.157*	-.233**
77. BPSS-BODY-T2	-.144*	-.117	.287**	.399**	.225**	-.150*	-.061	-.063	-.189**
78. BPSS-BODY-T4	-.222**	-.162*	.279**	.317**	.347**	-.149*	-.055	-.114	-.243**
79. BPSS-FACE-T1	-.097	-.088	.277**	.278**	.222**	-.121	-.163*	-.094	-.279**
80. BPSS-FACE-T2	-.123	-.179**	.296**	.421**	.326**	-.120	-.101	-.066	-.220**
81. BPSS-FACE-T4	-.304**	-.157*	.302**	.335**	.379**	-.134*	-.032	-.160*	-.227**
82. EAT-T1	.155*	.172**	-.194**	-.120	-.190**	.168**	.328**	.225**	.208**
83. EAT-T2	.223**	.255**	-.167**	-.167*	-.195**	.144*	.367**	.209**	.194**
84. EAT-T4	.276**	.261**	-.111	-.098	-.273**	.138*	.286**	.192**	.183**
85. BULITR-T1	.334**	.289**	-.227**	-.174**	-.233**	.174**	.355**	.261**	.301**
86. BULITR-T2	.345**	.302**	-.264**	-.247**	-.248**	.180**	.249**	.206**	.273**
87. BULITR-T4	.333**	.308**	-.188**	-.147*	-.301**	.184**	.270**	.249**	.275**

Note. Attractive and Thin Factor Time 1; BAAR-AT-T2 = Beliefs About Attractiveness Revised - Attractive and Thin Factor Time 2; BAAR-AT-T4 = Beliefs About Attractiveness Revised - Attractive and Thin Factor Time 4; MBSRQ-AE-T1 = Multidimensional Body Self Relations Questionnaire Appearance Evaluation Subscale Time 1; MBSRQ-AE-T2 = Multidimensional Body Self Relations Questionnaire Appearance Evaluation Subscale Time 2; (table continues)

Table 3 (continued).

Variable	64	65	66	67	68	69	70	71	72
46. SAD-T2									
47. SAD-T4									
48. HAPPY-T1									
49. HAPPY-T2									
50. HAPPY-T4									
51. SHAME-T1									
52. SHAME-T2									
53. SHAME-T4									
54. GUILT-T1									
55. GUILT-T2									
56. GUILT-T4									
57. CONFIDENT-T1									
58. CONFIDENT-T2									
59. CONFIDENT-T4									
60. ANXIOUS-T1									
61. ANXIOUS-T2									
62. ANXIOUS-T4									
63. STRESSED-T1									
64. STRESSED-T2	1								
65. STRESSED-T4	.424**	1							
66. PANASX-NA-T4	.307**	.418**	1						
67. BAAR-PF-T1	.157*	.204**	.268**	1					
68. BAAR-PF-T2	.120	.200**	.194**	.564**	1				
69. BAAR-PF-T4	.095	.154*	.307**	.622**	.443**	1			
70. BAAR-AT-T1	.184**	.282**	.250**	.580**	.646**	.472**	1		
71. BAAR-AT-T2	.085	.198**	.284**	.497**	.634**	.662**	.595**	1	
72. BAAR-AT-T4	.083	.228**	.295**	.545**	.540**	.637**	.694**	.748**	1
73. MBSRQ-AE-T1	-.265**	-.137*	-.176**	-.170**	-.188**	-.170**	-.223**	-.215**	-.192**
74. MBSRQ-AE-T2	-.189**	-.112	-.148*	-.237**	-.244**	-.200**	-.278**	-.306**	-.268**
75. MBSRQ-AE-T4	-.174**	-.220**	-.229**	-.266**	-.253**	-.199**	-.316**	-.341**	-.375**
76. BPSS-BODY-T1	-.198**	-.159*	-.165*	-.192**	-.189**	-.206**	-.251**	-.265**	-.257**
77. BPSS-BODY-T2	-.168**	-.129*	-.152*	-.229**	-.211**	-.198**	-.280**	-.270**	-.277**
78. BPSS-BODY-T4	-.168**	-.206**	-.183**	-.243**	-.243**	-.255**	-.358**	-.355**	-.407**
79. BPSS-FACE-T1	-.149*	-.036	-.141*	-.163*	-.125	-.099	-.092	-.068	-.085
80. BPSS-FACE-T2	-.191**	-.143*	-.231**	-.147*	-.184**	-.107	-.159*	-.151*	-.128*
81. BPSS-FACE-T4	-.125	-.146*	-.242**	-.115	-.122	-.192**	-.180**	-.209**	-.218**
82. EAT-T1	.301**	.207**	.221**	.289**	.223**	.273**	.238**	.237**	.230**
83. EAT-T2	.285**	.261**	.326**	.262**	.238**	.296**	.275**	.319**	.329**
84. EAT-T4	.206**	.255**	.356**	.305**	.295**	.353**	.334**	.344**	.413**
85. BULITR-T1	.299**	.288**	.399**	.336**	.248**	.400**	.322**	.357**	.327**
86. BULITR-T2	.256**	.301**	.421**	.306**	.281**	.330**	.341**	.416**	.362**
87. BULITR-T4	.198**	.308**	.438**	.300**	.263**	.349**	.350**	.393**	.423**

Note. MBSRQ-AE-T4 = Multidimensional Body Self Relations Questionnaire Appearance Evaluation Subscale Time 4; BPSS-BODY-T1= Body Parts Satisfaction Scale-Revised Body Subscale Time 1; BPSS-BODY-T2 = Body Parts Satisfaction Scale-Revised Body Subscale Time 2; BPSS-BODY-T4 = Body Parts Satisfaction Scale-Revised Body Subscale Time 4; BPSS-FACE-T1= Body Parts Satisfaction Scale-Revised Face Subscale Time 1; (table continues)

Table 3 (continued).

Variable	73	74	75	76	77	78	79	80	81
46. SAD-T2									
47. SAD-T4									
48. HAPPY-T1									
49. HAPPY-T2									
50. HAPPY-T4									
51. SHAME-T1									
52. SHAME-T2									
53. SHAME-T4									
54. GUILT-T1									
55. GUILT-T2									
56. GUILT-T4									
57. CONFIDENT-T1									
58. CONFIDENT-T2									
59. CONFIDENT-T4									
60. ANXIOUS-T1									
61. ANXIOUS-T2									
62. ANXIOUS-T4									
63. STRESSED-T1									
64. STRESSED-T2									
65. STRESSED-T4									
66. PANASX-NA-T4									
67. BAAR-PF-T1									
68. BAAR-PF-T2									
69. BAAR-PF-T4									
70. BAAR-AT-T1									
71. BAAR-AT-T2									
72. BAAR-AT-T4									
73. MBSRQ-AE-T1	1								
74. MBSRQ-AE-T2	.732**	1							
75. MBSRQ-AE-T4	.621**	.733**	1						
76. BPSS-BODY-T1	.749**	.659**	.635**	1					
77. BPSS-BODY-T2	.604**	.666**	.573**	.683**	1				
78. BPSS-BODY-T4	.578**	.593**	.717**	.645**	.783**	1			
79. BPSS-FACE-T1	.462**	.348**	.335**	.358**	.248**	.319**	1		
80. BPSS-FACE-T2	.470**	.485**	.444**	.423**	.547**	.426**	.577**	1	
81. BPSS-FACE-T4	.402**	.410**	.481**	.416**	.430**	.604**	.619**	.592**	1
82. EAT-T1	-.294**	-.141*	-.228**	-.281**	-.260**	-.255**	-.052	-.112	-.090
83. EAT-T2	-.305**	-.254**	-.251**	-.279**	-.242**	-.264**	-.026	-.168**	-.158
84. EAT-T4	-.295**	-.230**	-.404**	-.300**	-.296**	-.368**	-.128*	-.222**	-.210**
85. BULITR-T1	-.480**	-.346**	-.404**	-.451**	-.361**	-.389**	-.216**	-.255**	-.267**
86. BULITR-T2	-.448**	-.473**	-.473**	-.409**	-.451**	-.441**	-.192**	-.351**	-.324**
87. BULITR-T4	-.413**	-.379**	-.563**	-.396**	-.384**	-.502**	-.271**	-.338**	-.401**

Note. BPSS-FACE-T 2 = Body Parts Satisfaction Scale-Revised Face Subscale Time 2; BPSS-FACE-T4 = Body Parts Satisfaction Scale-Revised Face Subscale Time 4; EAT-T1=Eating Attitudes Test-26 Time 1; EAT-T2 = Eating Attitudes Test-26 Time 2; EAT-T4 = Eating Attitudes Test-26 Time 4; BULITR-T1 = Bulimia Test-Revised Time 1; BULITR-T2 = Bulimia Test-Revised Time 2; BULITR-T4= Bulimia Test-Revised Time 4.

(table continues)

Table 3 (continued).

Variable	82	83	84	85	86	87
46. SAD-T2						
47. SAD-T4						
48. HAPPY-T1						
49. HAPPY-T2						
50. HAPPY-T4						
51. SHAME-T1						
52. SHAME-T2						
53. SHAME-T4						
54. GUILT-T1						
55. GUILT-T2						
56. GUILT-T4						
57. CONFIDENT-T1						
58. CONFIDENT-T2						
59. CONFIDENT-T4						
60. ANXIOUS-T1						
61. ANXIOUS-T2						
62. ANXIOUS-T4						
63. STRESSED-T1						
64. STRESSED-T2						
65. STRESSED-T4						
66. PANASX-NA-T4						
67. BAAR-PF-T1						
68. BAAR-PF-T2						
69. BAAR-PF-T4						
70. BAAR-AT-T1						
71. BAAR-AT-T2						
72. BAAR-AT-T4						
73. MBSRQ-AE-T1						
74. MBSRQ-AE-T2						
75. MBSRQ-AE-T4						
76. BPSS-BODY-T1						
77. BPSS-BODY-T2						
78. BPSS-BODY-T4						
79. BPSS-FACE-T1						
80. BPSS-FACE-T2						
81. BPSS-FACE-T4						
82. EAT-T1	1					
83. EAT-T2	.679**	1				
84. EAT-T4	.615**	.674**	1			
85. BULITR-T1	.670**	.636**	.644**	1		
86. BULITR-T2	.510**	.660**	.648**	.807**	1	
87. BULITR-T4	.489**	.513**	.774**	.756**	.826**	1

Note (Continued).

Time 1 = Fall 2003; Time 2 = Spring 2004; Time 4 = Spring 2005.

* = $p < .05$; ** = $p < .01$.

Table 4

Frequency Reports of Eating Disordered Behaviors on the Bulimia Test-Revised (BULIT-R)

	Time %	1 N	Time %	2 N	Time %	4 N
<i>I use laxative or suppositories to help control my weight (Item 6)</i>						
1 time a day or more	0.4%	1	0.4%	1	0.4%	1
3 to 6 times a week	0%	0	0%	0	0%	0
1 to 2 times a week	0%	0	0.8%	2	0.8%	2
2 to 3 times a month	1.7%	4	0.4%	1	0.8%	2
1 time a month or less	97.9%	233	98.3%	234	97.9%	233
<i>There are times when I rapidly eat a large amount of food. (Item 8)</i>						
More than twice a week	8.0%	19	5.5%	13	8.0%	19
Twice a week	7.6%	18	3.8%	9	4.6%	11
Once a week	8.8%	21	8.4%	20	8.8%	21
2-3 times a month	25.2%	60	23.9%	57	14.3%	34
Once a month or less (or never)	50.4%	120	58.4%	139	64.3%	153
<i>How long have you been binge eating? (Item 9)</i>						
I don't binge eat	83.6%	199	74.8%	178	75.2%	179
Less than 3 months	2.5%	6	5.9%	14	4.2%	10
3 months – 1 year	4.6%	11	4.2%	10	2.5%	6
1 – 3 years	5.5%	13	6.3%	15	6.7%	16
3 or more years	3.8%	9	8.8%	21	11.3%	27
<i>I exercise in order to burn calories. (Item 11)</i>						
More than 2 hours a day	2.9%	7	1.7%	4	2.1%	5
About 2 hours a day	5.0%	12	5.0%	12	1.7%	4
More than 1 hour a day	12.2%	29	16.4%	39	8.8%	21
One hour or less a day	39.1%	93	35.3%	84	36.5%	87
I exercise but not to burn calories (or I don't exercise)	40.8%	97	41.6%	99	50.8%	121
<i>How often do you intentionally vomit after eating? (Item 15)</i>						
2 or more times a week	0.4%	1	0.4%	1	1.7%	4
Once a week	0.4%	1	0.4%	1	1.3%	3
2 – 3 time a month	2.5%	6	1.7%	4	2.1%	5
Once a month	1.7%	4	2.9%	7	1.7%	4
Less than once a month (or never)	95.0%	226	94.50%	225	93.2%	222
<i>I have tried to lose weight by fasting or going on diets. (Item 19)</i>						
Never or not in the past year	52.9%	126	57.6%	137	58.4%	139
Once in the past year	20.6%	49	16.4%	39	18.4%	44
2 – 3 times in the past year	12.2%	29	12.2%	29	10.5%	25
4 – 5 times in the past year	8.4%	20	9.2%	22	7.1%	17
Most or all of the time	5.9%	14	4.6%	11	5.5%	13
<i>In the last 3 months, on average how often did you binge eat? (Item 34)</i>						
More than twice a week	1.7%	4	2.1%	5	4.2%	10
Twice a week	5.0%	12	3.4%	8	5.5%	13
Once a week	6.3%	15	5.9%	14	4.6%	11
2-3 times a month	9.7%	23	10.9%	26	8.0%	19
Once a month or less (or never)	77.3%	184	77.7%	185	77.8%	135

(table continues)

Table 4 (continued).

	Time %	1 N	Time %	2 N	Time %	4 N
<i>I use diuretics (water pills) to help control my weight. (Item 36)</i>						
3 times a week or more	0%	0	0.8%	2	0.4%	1
Once or twice a week	0.4%	1	0.4%	1	0.8%	2
2-3 times a month	1.3%	3	0.8%	2	0.8%	2
Once a month	1.3%	3	1.3%	3	0.8%	2
Never	97.1%	231	96.6%	230	97.1%	231

Table 5
Standardized Parameter Estimates for All Factors in the Measurement Model

Latent Variables	Observed Variables	Factor Loadings	Error Variance
Parental Attachment-T1	PBI Maternal Care-T1	-.528	.849
	PBI Maternal Overprotection-T1	.860	.510
	PBI Paternal Overprotection-T1	.535	.845
History of Stress-T1	ICSRLE Time Pressure-T1	.711	.704
	ICSRLE Develop. Challenge-T1	.902	.432
	ICSRLE Academic Alienation-T1	.686	.727
Appraisal/Coping-T1	APPCOP Academic-T1	.606	.796
	PSS-T1	.908	.419
Psychological Distress – T1	VAMS Sad or Depressed – T1	.660	.757
	VAMS Happy – T1	-.503	.864
	VAMS Stressed – T1	.718	.696
Body Image – T1	BPSS-R Body Factor – T1	.820	.572
	MBSRQ-AE – T1	.902	.431
Internalization of Sociocultural Beliefs – T1	BAAR-Physically Fit – T1	.726	.688
	BAAR-Attractive & Thin – T1	.829	.560
Eating Disturbances - T1	BULIT-R – T1	.969	.247
	EAT-26 – T1	.598	.802
Psychological Distress – T2	VAMS Sad or Depressed – T2	.872	.490
	VAMS Happy – T2	-.595	.804
	VAMS Stressed – T2	.611	.791
Body Image – T2	BPSS-R Body Factor – T2	.737	.676
	MBSRQ-AE – T2	.892	.452
Internalization of Sociocultural Beliefs – T2	BAAR-Physically Fit – T2	.661	.750
	BAAR-Attractive & Thin – T2	.942	.337
Eating Disturbances – T2	BULIT-R – T2	.969	.248
	EAT-26 – T2	.649	.761
Psychological Distress – T4	VAMS Sad or Depressed – T4	.704	.710
	VAMS Happy – T4	-.543	.840
	VAMS Stressed – T4	.618	.786
	PANAS-X Negative Affect – T4	.641	.767

Note. T1 = Time 1; T2 = Time 2; T4 = Time 4; PBI-Maternal Care = Parental Bonding Instrument-Mother Care Scale; PBI-Maternal Overprotection = Parental Bonding Instrument-Mother Overprotection Scale; PBI-Paternal Overprotection = Parental Bonding Instrument-Father Overprotection Scale; ICSRLE-Time Pressure = Inventory of College Students' Recent Life Experiences-Time Pressure Scale; ICSRLE- Developmental Challenge = Inventory of College Students' Recent Life Experiences-Developmental Challenge Scale; ICSRLE-Academic Alienation = Inventory of College Students' Recent Life Experiences-Academic Alienation Scale; APPCOP Academic = Appraisal of academic demands and coping with experienced academic demands since beginning school; PSS = Perceived Stress Scale; VAMS Sad or Depressed = Analogue Mood Scale Sad or Depressed item; VAMS Happy = Analogue Mood Scale Happy item; VAMS Stressed = Analogue Mood Scale Stressed item; PANAS-X Negative

(table continues)

Table 5 (continued).

Latent Variables	Observed Variables	Factor Loadings	Error Variance
Body Image – T4	BPSS-R Body Factor – T4	.784	.620
	MBSRQ-AE – T4	.886	.464
Internalization of Sociocultural Beliefs – T4	BAAR-Physically Fit – T4	.711	.704
	BAAR-Attractive & Thin – T4	.966	.260
Eating Disturbances – T4	BULIT-R – T4	.966	.259
	EAT-26 – T4	.764	.645

Note. Affect = Positive and Negative Affect Scheduled Expanded Version-Negative Affect Scale; BPSS-R Body = Body Parts Satisfaction Scale-Revised Body Subscale; MBSRQ-AE = Multidimensional Body Self Relations Questionnaire Appearance Evaluation Subscale; BAAR-PF= Beliefs About Attractiveness-Revised Physically Fit Factor; BAAR-AT= Beliefs About Attractiveness-Revised Attractive and Thin Factor; BULIT-R = Bulimia Test-Revised; EAT-26 = Eating Attitudes Test-26.

Table 6

Standardized Parameter Estimates: Structural Model for Factors at Time 1 and Time 2

Latent Variables	Observed Variables	Factor Loadings	Error Variance
Parental Attachment-T1	PBI Maternal Care-T1	-.539	.842
	PBI Maternal Overprotection-T1	.847	.532
	PBI Paternal Overprotection-T1	.537	.844
History of Stress-T1	ICSRLE Time Pressure-T1	.709	.705
	ICSRLE Developmental Challenge-T1	.903	.429
	ICSRLE Academic Alienation-T1	.685	.728
Appraisal/Coping-T1	APPCOP Academic-T1	.567	.824
	PSS-T1	.980	.198
Psychological Distress – T2	VAMS Sad or Depressed – T2	.890	.455
	VAMS Happy – T2	-.582	.813
	VAMS Stressed – T2	.605	.796
Body Image – T2	BPSS-R Body Factor – T2	.767	.641
	MBSRQ-AE – T2	.868	.497
Internalization of Sociocultural Beliefs – T2	BAAR-Physically Fit – T2	.699	.715
	BAAR-Attractive & Thin – T2	.914	.406
Eating Disturbances – T2	BULIT-R – T2	.970	.245
	EAT-26 – T2	.640	.768

Note. T1 = Time 1; T2 = Time 2; T4 = Time 4; PBI-Maternal Care = Parental Bonding Instrument-Mother Care Scale; PBI-Maternal Overprotection = Parental Bonding Instrument-Mother Overprotection Scale; PBI-Paternal Overprotection = Parental Bonding Instrument-Father Overprotection Scale; ICSRLE-Time Pressure = Inventory of College Students' Recent Life Experiences-Time Pressure Scale; ICSRLE- Developmental Challenge = Inventory of College Students' Recent Life Experiences-Developmental Challenge Scale; ICSRLE-Academic Alienation = Inventory of College Students' Recent Life Experiences-Academic Alienation Scale; APPCOP Academic = Appraisal of academic demands and coping with experienced academic demands since beginning school; PSS = Perceived Stress Scale; VAMS Sad or Depressed = Analogue Mood Scale Sad or Depressed item; VAMS Happy = Analogue Mood Scale Happy item; VAMS Stressed = Analogue Mood Scale Stressed item; PANAS-X Negative Affect = Positive and Negative Affect Scheduled Expanded Version-Negative Affect Scale; BPSS-R Body = Body Parts Satisfaction Scale-Revised Body Subscale; MBSRQ-AE = Multidimensional Body Self Relations Questionnaire Appearance Evaluation Subscale; BAAR-PF= Beliefs About Attractiveness-Revised Physically Fit Factor; BAAR-AT= Beliefs About Attractiveness-Revised Attractive and Thin Factor; BULIT-R = Bulimia Test-Revised; EAT-26 = Eating Attitudes Test-26.

Table 7

Standardized Parameter Estimates: Structural Model for Factors at Time 1 and Time 4

Latent Variables	Observed Variables	Factor Loadings	Error Variance
Parental Attachment-T1	PBI Maternal Care-T1	-.538	.843
	PBI Maternal Overprotection-T1	.842	.540
	PBI Paternal Overprotection-T1	.543	.840
History of Stress-T1	ICSRLE Time Pressure-T1	.711	.703
	ICSRLE Developmental Challenge-T1	.901	.434
	ICSRLE Academic Alienation-T1	.686	.727
Appraisal/Coping-T1	APPCOP Academic-T1	.592	.806
	PSS-T1	.939	.343
Psychological Distress – T4	VAMS Sad or Depressed – T4	.838	.546
	VAMS Happy – T4	-.612	.791
	VAMS Stressed – T4	.578	.816
Body Image – T4	BPSS-R Body Factor – T4	.831	.557
	MBSRQ-AE – T4	.853	.521
Internalization of Sociocultural Beliefs – T4	BAAR-Physically Fit – T4	.781	.625
	BAAR-Attractive & Thin – T4	.884	.468
Eating Disturbances – T4	BULIT-R – T4	.980	.200
	EAT-26 – T4	.756	.654

Note. T1 = Time 1; T2 = Time 2; T4 = Time 4; PBI-Maternal Care = Parental Bonding Instrument-Mother Care Scale; PBI-Maternal Overprotection = Parental Bonding Instrument-Mother Overprotection Scale; PBI-Paternal Overprotection = Parental Bonding Instrument-Father Overprotection Scale; ICSRLE-Time Pressure = Inventory of College Students' Recent Life Experiences-Time Pressure Scale; ICSRLE- Developmental Challenge = Inventory of College Students' Recent Life Experiences-Developmental Challenge Scale; ICSRLE-Academic Alienation = Inventory of College Students' Recent Life Experiences-Academic Alienation Scale; APPCOP Academic = Appraisal of academic demands and coping with experienced academic demands since beginning school; PSS = Perceived Stress Scale; VAMS Sad or Depressed = Analogue Mood Scale Sad or Depressed item; VAMS Happy = Analogue Mood Scale Happy item; VAMS Stressed = Analogue Mood Scale Stressed item; PANAS-X Negative Affect = Positive and Negative Affect Scheduled Expanded Version-Negative Affect Scale; BPSS-R Body = Body Parts Satisfaction Scale-Revised Body Subscale; MBSRQ-AE = Multidimensional Body Self Relations Questionnaire Appearance Evaluation Subscale; BAAR-PF= Beliefs About Attractiveness-Revised Physically Fit Factor; BAAR-AT= Beliefs About Attractiveness-Revised Attractive and Thin Factor; BULIT-R = Bulimia Test-Revised; EAT-26 = Eating Attitudes Test-26.

Table 8

Decomposition of Effects: Structural Model A for Factors at Time 1 and Time 2

Criterion Variable	Predictor/Mediating Variable	Non-Standardized Values			Standardized Value
		Parameter Estimate	Standard Error	Test Statistic	Parameter Estimate
Appraisal /Coping-T1	Parental Attachment-T1 ^a	2.812	.542	5.185*	.399
	History of Stress-T1 ^a	1.041	.495	2.104*	.148
Negative Affect-T2	Parental Attachment-T1 ^b	.137	.036	3.771*	.216
	History of Stress-T1 ^b	.051	.026	1.965*	.080
	Appraisal/ Coping-T1 ^a	.049	.009	5.185*	.541
Body Image-T2	Parental Attachment-T1 ^b	-.059	.020	-3.009*	-.075
	History of Stress-T1 ^b	-.022	.012	-1.828	-.028
	Appraisal/ Coping-T1 ^b	-.021	.006	-3.600*	-.188
	Internalization Thin-Ideal-T2 ^a	-.261	.061	-4.271*	-.330
	Negative Affect-T2 ^a	-.432	.105	-4.095*	-.347
Eating Disturb.-T2	Parental Attachment-T1 ^b	.648	.190	3.415*	.129
	History of Stress-T1 ^b	.240	.126	1.909	.048
	Appraisal/ Coping-T1 ^a	.172	.049	3.525*	.242
	Appraisal/ Coping-T1 ^b	.059	.018	3.171*	.082
	Negative Affect-T2 ^b	1.197	.350	3.423*	.190
	Body Image-T2 ^a	-2.774	.518	-5.359*	-.437
	Internalization Thin-Ideal-T2 ^a	1.165	.342	3.405*	.232
	Internalization Thin-Ideal-T2 ^b	.724	.205	3.278*	.144

Note. T1 = Time 1; T2 = Time 2.

^a Direct Effect.

^b Indirect Effect.

* = $p < .05$.

Table 9
Decomposition of Effects: Structural Model A for Factors at Time 1 and Time 4

Criterion Variable	Predictor/Mediating Variable	Non-Standardized Values			Standardized Value
		Parameter Estimate	Standard Error	Test Statistic	Parameter Estimate
Appraisal /Coping-T1	Parental Attachment-T1 ^a	2.744	.537	5.110*	.413
	History of Stress-T1 ^a	1.080	.487	2.217*	.162
Negative Affect-T4	Parental Attachment-T1 ^b	-.097	.027	-3.536*	-.190
	History of Stress-T1 ^b	-.038	.019	-2.020*	-.075
	Appraisal/ Coping-T1 ^a	-.035	.008	-4.453*	-.460
Body Image-T4	Parental Attachment-T1 ^b	-.030	.012	-2.518*	-.047
	History of Stress-T1 ^b	-.012	.007	-1.760	-.018
	Appraisal/ Coping-T1 ^b	-.011	.004	-2.810*	-.113
	Internalization Thin-Ideal-T4 ^a	-.285	.049	-5.858*	-.443
	Negative Affect-T4 ^a	.310	.098	-3.172*	-.246
Eating Disturb.-T4	Parental Attachment-T1 ^b	.825	.228	3.625*	.140
	History of Stress-T1 ^b	.325	.160	2.036*	.055
	Appraisal/ Coping-T1 ^a	.254	.061	4.202*	.288
	Appraisal/ Coping-T1 ^b	.047	.018	2.616*	.053
	Negative Affect-T4 ^b	-1.318	.460	-2.866*	-.115
	Body Image-T4 ^a	-4.255	.725	-5.870*	-.446
	Internalization Thin-Ideal-T4 ^a	1.058	.403	2.624*	.180
	Internalization Thin-Ideal-T4 ^b	1.212	.276	4.396*	.206

Note. T1 = Time 1; T4 = Time 4.

^a Direct Effect.

^b Indirect Effect.

* = $p < .05$.

Table 10

Decomposition of Effects: Structural Model B for Factors at Time 1 and Time 4

Criterion Variable	Predictor/Mediating Variable	Non-Standardized Values			Standardized Value
		Parameter Estimate	Standard Error	Test Statistic	Parameter Estimate
Appraisal /Coping-T1	Parental Attachment-T1 ^a	2.759	.543	5.081*	.394
	History of Stress-T1 ^a	1.041	.496	2.100*	.149
Negative Affect-T4	Parental Attachment-T1 ^b	-.086	.026	-3.280*	-.171
	History of Stress-T1 ^b	-.032	.017	-1.887	-.065
	Appraisal/ Coping-T1 ^a	-.031	.008	-4.084*	-.435
Body Image-T4	Internalization Thin-Ideal-T4 ^a	-.316	.050	-6.302*	-.483
Eating Disturb.-T4	Parental Attachment-T1 ^b	.184	.082	2.249*	.031
	History of Stress-T1 ^b	.070	.043	1.610	.012
	Appraisal/ Coping-T1 ^b	.067	.027	2.463*	.079
	Negative Affect-T4 ^a	-2.155	.766	-2.813*	-.181
	Body Image-T4 ^a	-4.637	.756	-6.132*	-.511
	Internalization Thin-Ideal-T4 ^a	1.002	.432	2.321*	.169
	Internalization Thin-Ideal-T4 ^b	1.466	.315	4.647*	.247

Note. T1 = Time 1; T4 = Time 4.

^a Direct Effect.

^b Indirect Effect.

* = $p < .05$.

Table 11
Model Fit (N = 236)

Model	<i>df</i>	χ^2	S-B χ^2	NFI	NNFI	CFI	RCFI	SRMR	RMSEA
Measurement Model – All Factors	489	647.82***	608.86***	.943	.981	.985	.927	.069	.037
Measurement Model – T1 and T2	100	133.60*	127.88*	.974	.991	.993	.946	.046	.038
Structural Model A– T1 and T2	114	169.90***	163.79**	.967	.987	.989	.903	.075	.046
Respecified Model A – T1 and T2	113	156.21**	150.80**	.970	.989	.991	.926	.064	.040
Structural Model B– T1 and T2	113	176.86***	169.51***	.960	.982	.985	.906	.090	.049
Measurement Model – T1 and T4	99	139.99**	136.34**	.978	.991	.993	.911	.051	.042
Structural Model A– T1 and T4	113	183.55***	178.86***	.971	.986	.989	.844	.085	.052
Respecified Model A – T1 and T4	112	164.15***	160.16**	.974	.990	.992	.886	.072	.045
Structural Model B– T1 and T4	113	187.66***	182.66***	.971	.985	.988	.835	.096	.053

Note. T1 = Time 1, T2 = Time 2, T4 = Time 4.

S-B χ^2 = Satorra-Bentler scaled chi square; NFI = normed fit index; NNFI = non-normed fit index; CFI = comparative fit index; RCFI = robust comparative fit index; SRMR = standardized root mean squared residual; RMSEA = root mean square error of approximation.

* = $p < .05$, ** = $p < .01$, *** = $p < .001$

Table 12

Model Fit and Comparison of Competing Models: Negative Affect and Body Image (N = 236)

Model	df	χ^2	S-B χ^2	NFI	NNFI	CFI	RCFI	SRMR	RMSEA	χ^2 Change ^a
Relations between Time 1 and Time 2										
Measurement Model	26	45.94**	42.61*	.989	.992	.995	.948	.053	.057	--
Baseline Model	28	51.00**	46.02*	.988	.991	.995	.944	.060	.059	--
Model 1	27	49.49**	45.63*	.989	.991	.995	.942	.061	.060	1.51 n.s.
Model 2	27	47.52**	45.14*	.989	.992	.995	.949	.051	.057	3.48 n.s.
Model 3	26	45.94**	42.60*	.989	.992	.995	.948	.053	.057	5.06 n.s.
Model 4	28	52.59**	47.23*	.988	.990	.994	.940	.063	.061	n/a ^b
Model 5	28	47.59*	43.05*	.989	.992	.995	.953	.051	.055	n/a ^b
Model 6	26	47.02**	42.19*	.978	.982	.990	.966	.053	.059	n/a ^b
Relations between Time 2 and Time 4										
Measurement Model	24	37.59*	34.48	.966	.976	.987	.989	.052	.049	--
Baseline Model	26	38.60	35.93	.989	.994	.997	.979	.055	.043	--
Model 1	25	37.73	34.76	.989	.994	.996	.980	.052	.044	0.87 n.s.
Model 2	25	38.51	35.87	.989	.993	.996	.977	.057	.045	0.09 n.s.
Model 3	24	37.66*	34.69	.989	.993	.996	.977	.054	.046	0.94 n.s.
Model 4	26	37.74	35.29	.989	.995	.997	.981	.052	.041	n/a ^b
Model 5	26	40.88*	38.17	.988	.993	.996	.974	.056	.047	n/a ^b
Model 6	25	37.70	35.10	.989	.994	.997	.979	.054	.044	n/a ^b
Relations between Time 1 and Time 4										
Measurement Model	24	37.27*	35.04	.959	.971	.985	.986	.050	.049	--
Baseline Model	26	37.46	35.00	.959	.977	.987	.989	.050	.043	--
Model 1	25	37.32	34.89	.959	.974	.986	.988	.049	.046	0.14 n.s.
Model 2	25	37.39	35.12	.959	.974	.986	.987	.052	.046	0.07 n.s.
Model 3	24	37.27	35.04	.959	.971	.985	.986	.050	.049	0.19 n.s.
Model 4	26	37.49	35.12	.959	.977	.987	.989	.048	.043	n/a ^b
Model 5	26	39.78	37.47	.956	.972	.984	.986	.054	.047	n/a ^b
Model 6	25	37.48	35.22	.959	.974	.985	.987	.048	.046	n/a ^b

Note. S-B χ^2 = Satorra-Bentler scaled chi square; NFI = normed fit index; NNFI = non-normed fit index; CFI = comparative fit index; RCFI = robust comparative fit index; SRMR = standardized root mean squared residual; RMSEA = root mean square error of approximation.

^a Chi-square change values reflect a comparison to the baseline model.

^b Models 4, 5, and 6 represent reparameterizations of the baseline models, so they were not nested within the baseline models.

* = $p < .05$, ** = $p < .01$, *** = $p < .001$

Table 13

Model Fit and Comparison of Competing Models: Internalization of Thin-Ideal and Body Image (N = 236)

Model	df	χ^2	S-B χ^2	NFI	NNFI	CFI	RCFI	SRMR	RMSEA	χ^2 Change ^a
Relations between Time 1 and Time 2										
Measurement Model	10	4.74	4.43	.995	1.015	1.000	1.000	.011	.000	--
Baseline Model	12	15.40	13.69	.985	.992	.997	.998	.043	.035	--
Model 1	11	9.42	8.46	.991	1.004	1.000	1.000	.031	.000	5.98*
Model 2	11	10.53	9.74	.990	1.001	1.000	1.000	.027	.000	4.87*
Model 3	10	4.74	4.43	.995	1.015	1.000	1.000	.011	.000	10.66**
Model 4	12	9.52	8.39	.991	1.006	1.000	1.000	.030	.000	n/a ^b
Model 5	12	11.42	10.39	.989	1.001	1.000	1.000	.029	.000	n/a ^b
Model 6	11	5.69	5.18	.994	1.014	1.000	1.000	.012	.000	n/a ^b
Relations between Time 2 and Time 4										
Measurement Model	10	5.28	4.26	.996	1.011	1.000	1.000	.013	.000	--
Baseline Model	12	16.17	10.05	.990	.999	1.000	1.000	.031	.012	--
Model 1	11	10.97	4.99	.995	1.010	1.000	1.000	.018	.000	5.20*
Model 2	11	15.69	8.98	.991	1.000	1.000	1.000	.026	.003	0.48 n.s.
Model 3	10	10.71	4.26	.996	1.011	1.000	1.000	.013	.000	5.46 n.s.
Model 4	12	19.58	11.70	.988	.995	.998	1.000	.030	.031	n/a ^b
Model 5	12	12.29	6.24	.994	1.009	1.000	1.000	.021	.000	n/a ^b
Model 6	11	5.65	5.02	.995	1.011	1.000	1.000	.013	.000	n/a ^b
Relations between Time 1 and Time 4										
Measurement Model	10	10.17	9.55	.990	1.000	1.000	1.000	.016	.008	--
Baseline Model	12	19.86	18.95	.980	.981	.992	.991	.053	.053	--
Model 1	11	13.25	12.50	.987	.994	.998	.998	.032	.030	6.61*
Model 2	11	16.46	15.62	.983	.986	.994	.994	.066	.046	3.40 n.s.
Model 3	10	10.17	9.55	.990	1.000	1.000	1.000	.016	.008	9.69**
Model 4	12	15.86	14.97	.984	.991	.996	.996	.037	.037	n/a ^b
Model 5	12	25.52*	23.57*	.974	.967	.986	.985	.046	.069	n/a ^b
Model 6	11	10.90	10.06	.995	1.000	1.000	1.000	.016	.000	n/a ^b

Note. S-B χ^2 = Satorra-Bentler scaled chi square; NFI = normed fit index; NNFI = non-normed fit index; CFI = comparative fit index; RCFI = robust comparative fit index; SRMR = standardized root mean squared residual; RMSEA = root mean square error of approximation.

^a Chi-square change values reflect a comparison to the baseline model.

^b Models 4, 5, and 6 represent reparameterizations of the baseline models, so they were not nested within the baseline models.

* = $p < .05$, ** = $p < .01$, *** = $p < .001$

Table 14

Model Fit and Comparison of Competing Models: Internalization of Thin-Ideal and Eating Disturbance (N = 236)

Model	df	χ^2	S-B χ^2	NFI	NNFI	CFI	RCFI	SRMR	RMSEA	χ^2 Change ^a
Relations between Time 1 and Time 2										
Measurement Model	10	9.25	9.86	.991	1.002	1.000	1.000	.022	.000	--
Baseline Model	12	11.57	10.37	.988	1.001	1.000	1.000	.032	.000	--
Model 1	11	10.27	9.69	.990	1.002	1.000	1.000	.026	.000	0.30 n.s.
Model 2	11	10.26	9.32	.990	1.002	1.000	1.000	.026	.000	0.31 n.s.
Model 3	10	9.25	8.86	.991	1.002	1.000	1.000	.022	.000	2.32 n.s.
Model 4	12	13.28	11.02	.987	.997	.999	1.000	.029	.021	n/a ^b
Model 5	12	17.83	19.07	.982	.986	.994	.994	.026	.045	n/a ^b
Model 6	11	10.92	10.00	.989	1.000	1.000	1.000	.022	.000	n/a ^b
Relations between Time 2 and Time 4										
Measurement Model	10	11.17	9.83	.991	.997	.999	1.000	.016	.022	--
Baseline Model	12	15.83	13.52	.987	.993	.997	.998	.032	.037	--
Model 1	11	11.19	10.10	.991	1.000	1.000	1.000	.017	.009	4.64*
Model 2	11	15.68	13.04	.987	.990	.996	.997	.030	.043	0.15 n.s.
Model 3	10	11.17	9.83	.991	.997	.999	1.000	.016	.022	4.66 n.s.
Model 4	12	12.86	11.46	.990	.998	.999	1.000	.019	.017	n/a ^b
Model 5	12	20.49	16.92	.983	.984	.993	.994	.031	.055	n/a ^b
Model 6	11	12.12	10.57	.990	.998	.999	1.000	.016	.021	n/a ^b
Relations between Time 1 and Time 4										
Measurement Model	10	10.62	9.22	.989	.998	.999	1.000	.021	.016	--
Baseline Model	12	13.45	11.65	.987	.997	.999	1.000	.030	.023	--
Model 1	11	10.77	9.46	.989	1.001	1.000	1.000	.020	.000	2.68 n.s.
Model 2	11	13.43	11.50	.987	.994	.998	.999	.030	.031	0.02 n.s.
Model 3	10	10.62	9.22	.989	.998	.999	1.000	.021	.016	2.83 n.s.
Model 4	12	13.62	12.11	.986	.996	.998	1.000	.024	.024	n/a ^b
Model 5	12	23.50*	19.90	.977	.972	.988	.989	.037	.064	n/a ^b
Model 6	11	13.19	11.45	.987	.994	.998	.999	.022	.029	n/a ^b

Note. S-B χ^2 = Satorra-Bentler scaled chi square; NFI = normed fit index; NNFI = non-normed fit index; CFI = comparative fit index; RCFI = robust comparative fit index; SRMR = standardized root mean squared residual; RMSEA = root mean square error of approximation.

^a Chi-square change values reflect a comparison to the baseline model.

^b Models 4, 5, and 6 represent reparameterizations of the baseline models, so they were not nested within the baseline models.

* = $p < .05$, ** = $p < .01$, *** = $p < .001$

Table 15

Model Fit and Comparison of Competing Models: Body Image and Eating Disturbance (N = 236)

Model	df	χ^2	S-B χ^2	NFI	NNFI	CFI	RCFI	SRMR	RMSEA	χ^2 Change ^a
Relations between Time 1 and Time 2										
Measurement Model	11	19.46	16.48	.995	.995	.998	.974	.029	.057	--
Baseline Model	13	21.28*	18.08	.995	.996	.998	.976	.030	.052	--
Model 1	12	19.80	16.52	.995	.996	.998	.978	.030	.053	1.48 n.s.
Model 2	12	21.20*	18.32	.995	.995	.998	.970	.029	.057	0.08 n.s.
Model 3	11	19.46	16.48	.995	.995	.998	.974	.029	.057	1.75 n.s.
Model 4	13	42.46***	32.99***	.990	.984	.993	.904	.041	.098	n/a ^b
Model 5	13	49.75***	41.28***	.988	.980	.991	.857	.041	.110	n/a ^b
Model 6	12	78.78***	53.53***	.935	.868	.943	.952	.040	.154	n/a ^b
Relations between Time 2 and Time 4										
Measurement Model	11	19.07	14.67	.995	.995	.998	.982	.032	.056	--
Baseline Model	13	20.24	16.05	.995	.996	.998	.985	.032	.049	--
Model 1	12	19.35	15.33	.996	.996	.998	.983	.031	.051	0.89 n.s.
Model 2	12	19.29	14.95	.996	.996	.998	.985	.032	.051	0.95 n.s.
Model 3	11	18.85	14.49	.996	.995	.998	.982	.031	.055	1.33 n.s.
Model 4	13	47.60***	36.65***	.989	.983	.992	.880	.050	.106	n/a ^b
Model 5	13	41.53***	28.46**	.991	.986	.994	.992	.041	.097	n/a ^b
Model 6	12	138.75***	96.99***	.978	.955	.980	.837	.096	.203	n/a ^b
Relations between Time 1 and Time 4										
Measurement Model	11	7.10	6.30	.998	1.002	1.000	1.000	.022	.000	--
Baseline Model	13	8.46	7.35	.998	1.002	1.000	1.000	.024	.000	--
Model 1	12	7.21	6.30	.998	1.003	1.000	1.000	.021	.000	1.25 n.s.
Model 2	12	8.13	7.18	.998	1.002	1.000	1.000	.023	.000	0.33 n.s.
Model 3	11	7.10	6.30	.998	1.002	1.000	1.000	.022	.000	1.36 n.s.
Model 4	13	33.01**	27.60*	.992	.989	.989	.930	.044	.081	n/a ^b
Model 5	13	32.72**	24.23*	.992	.992	.990	.947	.041	.078	n/a ^b
Model 6	12	100.98***	72.02***	.913	.816	.921	.931	.050	.178	n/a ^b

Note. S-B χ^2 = Satorra-Bentler scaled chi square; NFI = normed fit index; NNFI = non-normed fit index; CFI = comparative fit index; RCFI = robust comparative fit index; SRMR = standardized root mean squared residual; RMSEA = root mean square error of approximation.

^a Chi-square change values reflect a comparison to the baseline model.

^b Models 4, 5, and 6 represent reparameterizations of the baseline models, so they were not nested within the baseline models.

* = $p < .05$, ** = $p < .01$, *** = $p < .001$

Table 16

Model Fit and Comparison of Competing Models: Negative Affect and Eating Disturbance (N = 236)

Model	df	χ^2	S-B χ^2	NFI	NNFI	CFI	RCFI	SRMR	RMSEA	χ^2 Change ^a
Relations between Time 1 and Time 2										
Measurement Model	25	40.91*	31.18	.990	.993	.996	.978	.062	.052	--
Baseline Model	27	51.18**	39.32	.987	.990	.994	.957	.072	.062	--
Model 1	26	44.64*	34.72	.999	.992	.995	.969	.068	.055	6.54*
Model 2	26	46.75**	35.26	.998	.991	.995	.968	.065	.058	4.43*
Model 3	25	40.91*	31.19	.990	.993	.996	.978	.062	.052	10.27**
Model 4	27	44.78*	11.81	.999	.992	.996	.976	.067	.053	n/a ^b
Model 5	27	51.16**	38.20	.997	.990	.994	.961	.066	.062	n/a ^b
Model 6	26	41.22*	29.29	.998	.999	.993	.995	.061	.053	n/a ^b
Relations between Time 2 and Time 4										
Measurement Model	26	37.11	33.89	.992	.995	.997	.961	.056	.045	--
Baseline Model	27	38.43	35.92	.991	.996	.997	.965	.064	.042	--
Model 1	26	38.37	36.03	.991	.995	.997	.960	.064	.045	0.06 n.s.
Model 2	26	37.16	34.77	.992	.996	.997	.965	.056	.043	0.27 n.s.
Model 3	25	37.11	34.89	.992	.995	.997	.961	.056	.045	0.32 n.s.
Model 4	27	38.64	36.31	.991	.995	.997	.963	.064	.043	n/a ^b
Model 5	27	37.28	34.69	.992	.996	.998	.969	.056	.040	n/a ^b
Model 6	26	37.11	35.05	.992	.996	.997	.964	.056	.043	n/a ^b
Relations between Time 1 and Time 4										
Measurement Model	25	38.51	35.02	.991	.995	.991	.964	.062	.042	--
Baseline Model	27	44.81*	43.98*	.999	.992	.995	.939	.074	.053	--
Model 1	26	38.69	38.13	.990	.994	.997	.957	.070	.046	6.12*
Model 2	26	41.81*	41.13*	.999	.993	.996	.946	.066	.051	3.00 n.s.
Model 3	25	25.51	35.02	.991	.995	.997	.964	.062	.042	9.30**
Model 4	27	41.23*	40.03	.999	.994	.996	.953	.068	.047	n/a ^b
Model 5	27	41.84*	40.06	.999	.993	.996	.952	.066	.048	n/a ^b
Model 6	26	39.53*	36.86	.996	.999	.983	.984	.064	.050	n/a ^b

Note. S-B χ^2 = Satorra-Bentler scaled chi square; NFI = normed fit index; NNFI = non-normed fit index; CFI = comparative fit index; RCFI = robust comparative fit index; SRMR = standardized root mean squared residual; RMSEA = root mean square error of approximation.

^a Chi-square change values reflect a comparison to the baseline model.

^b Models 4, 5, and 6 represent reparameterizations of the baseline models, so they were not nested within the baseline models.

* = $p < .05$, ** = $p < .01$, *** = $p < .001$

Table 17

Correlations Among Factors at Time 1, Time 2, and Time 4 in Final Measurement Model

Variable	1	2	3	4	5	6	7	8	9	10
1. PAR ATT –T1	1									
2. STRESS –T1	-.075*	1								
3. APP/COP – T1	.324	.122*	1							
4. AFFECT –T1	-.229	-.155*	-.915	1						
5. AFFECT –T2	-.239	-.082*	-.576	.617	1					
6. AFFECT –T4	-.224	-.041*	-.516	.275	.712	1				
7. BODY –T1	-.124*	-.050*	-.354	.440	.427	.297	1			
8. BODY –T2	-.093*	-.032*	-.295	.360	.477	.302	.920	1		
9. BODY –T4	-.076*	-.002*	-.369	.420	.450	.451	.848	.964	1	
10. INTERN –T1	.013*	-.008*	.281	-.275	-.225	-.382	-.270	-.332	-.357	1
11. INTERN –T2	.047*	-.030*	.266	-.247	-.215	-.318	-.308	-.401	-.456	.735
12. INTERN –T4	.063*	-.105*	.177	-.166	-.206	-.385	-.306	-.389	-.512	.836
13. EATING –T1	.148	.025*	.441	-.361	-.326	-.458	-.553	-.436	-.484	.486
14. EATING –T2	.236	.036*	.392	-.377	-.388	-.477	-.505	-.563	-.569	.422
15. EATING –T4	.239	.079*	.403	-.356	-.295	-.499	-.474	-.463	-.637	.431

Note: PAR ATT – T1 = Parental Attachment Time 1; STRESS – T1 = History of Stress Time 1; APP/COP – T1 = Appraisal/Coping Time 1; AFFECT – T1 = Negative Affect Time 1; AFFECT – T2 = Negative Affect Time 2; AFFECT – T4 = Negative Affect Time 4; BODY – T1 = Body Image Time 1; BODY – T2 = Body Image Time 2; BODY – T4 = Body Image Time 4; INTERN – T1 = Internalization of the Thin-Ideal Time 1; INTERN – T2 = Internalization of the Thin-Ideal Time 2; INTERN – T4 = Internalization of the Thin-Ideal Time 4; EATING – T1 = Eating Disturbance Time 1; EATING – T2 = Eating Disturbance Time 2; EATING – T4 = Eating Disturbance Time 4.

* = Non-significant Correlations

(table continues)

Table 17 (continued).

Variable	11	12	13	14	15
1. PAR ATT – T1					
2. STRESS – T1					
3. APP/COP – T1					
4. AFFECT – T1					
5. AFFECT – T2					
6. AFFECT – T4					
7. BODY – T1					
8. BODY – T2					
9. BODY – T4					
10. INTERN – T1					
11. INTERN – T2	1				
12. INTERN – T4	.909	1			
13. EATING – T1	.399	.384	1		
14. EATING – T2	.469	.428	.860	1	
15. EATING – T4	.437	.480	.802	.862	1

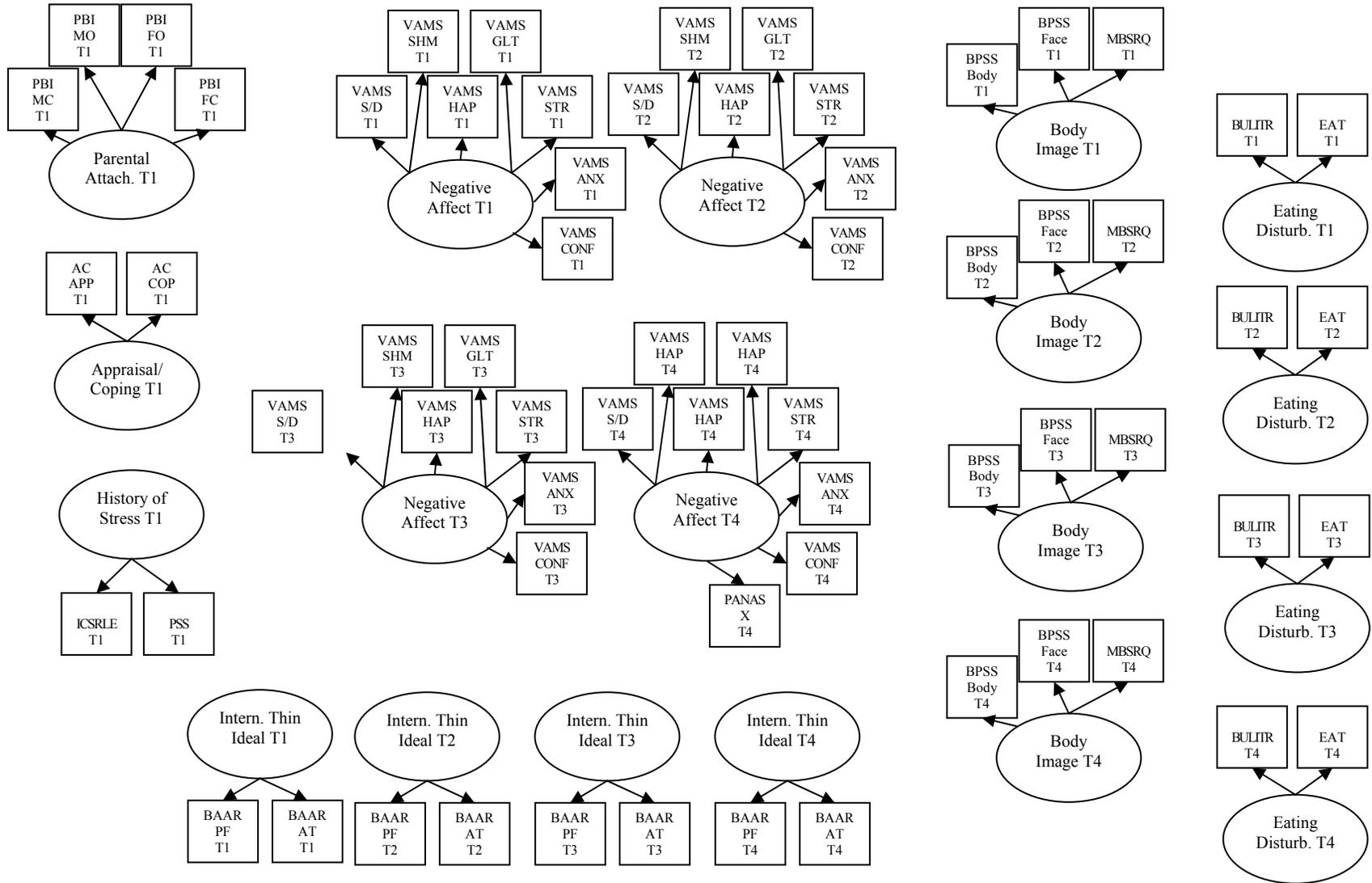


Figure 1. Diagram of the proposed measurement model for all factors at Time 1, Time 2, Time 3, and Time 4.

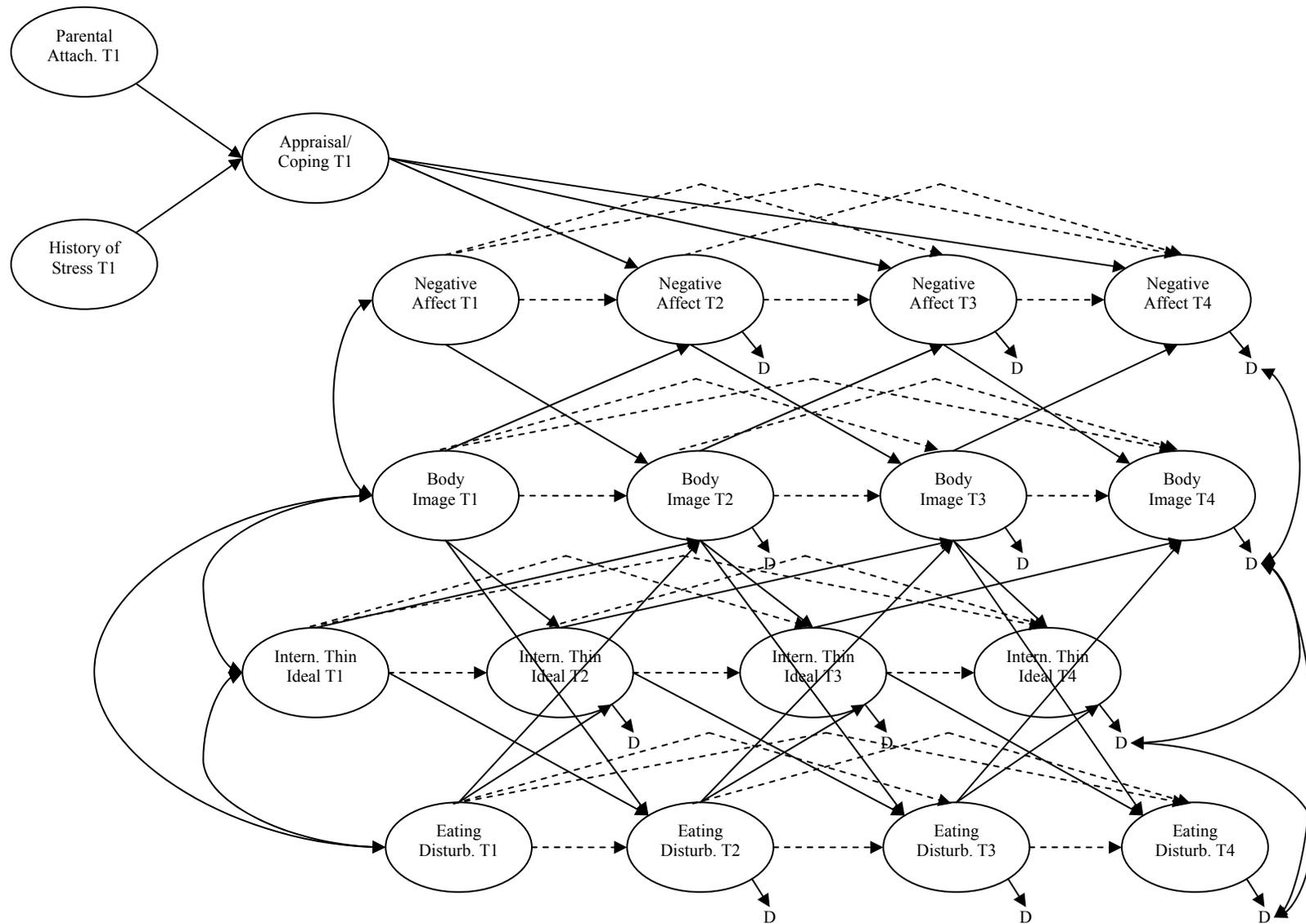


Figure 2. Diagram of the proposed structural Model A for Time 1, Time 2, Time 3, and Time 4.

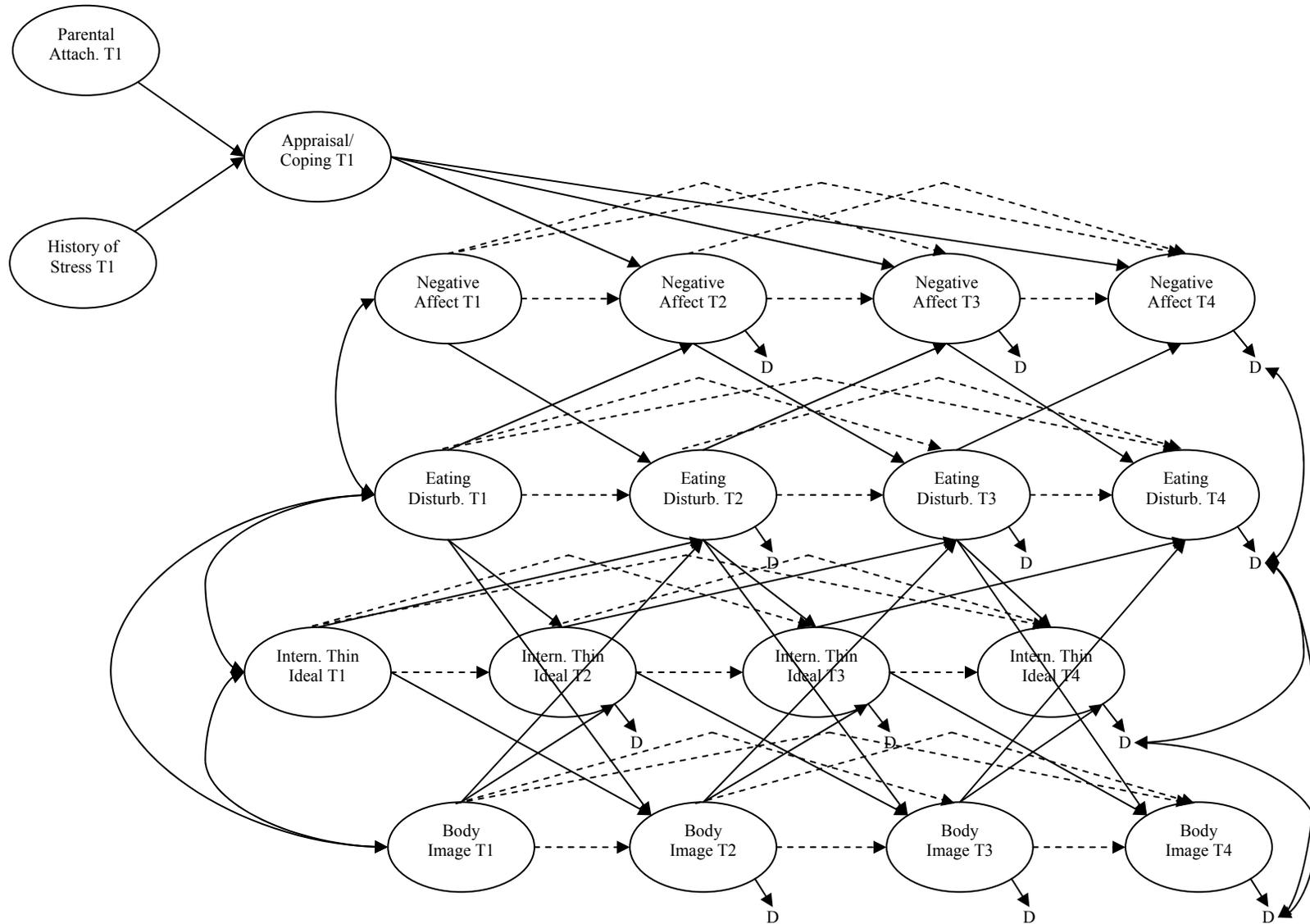


Figure 3. Diagram of the proposed structural Model B for Time 1, Time 2, Time 3, and Time 4.

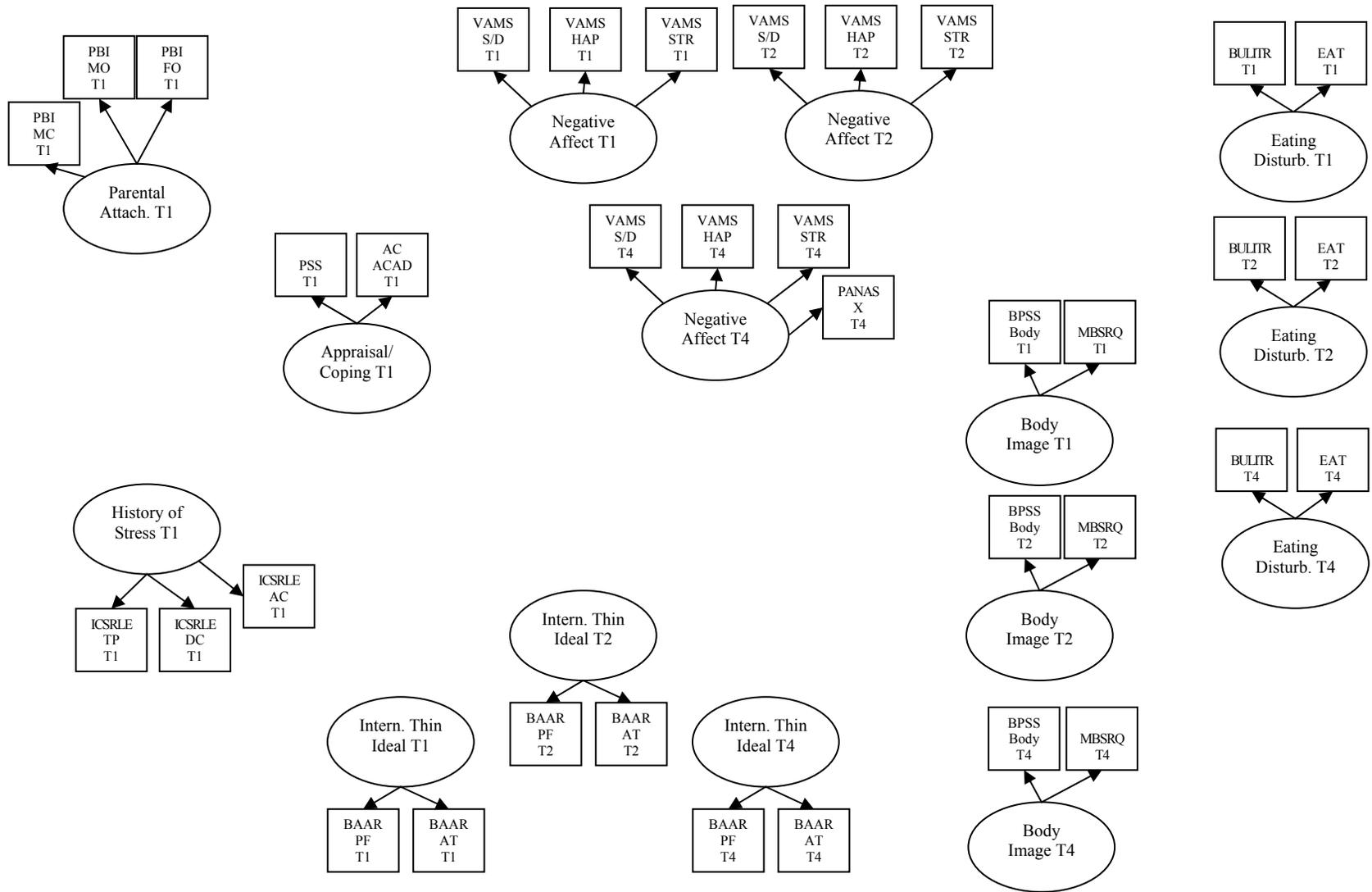


Figure 4. Diagram of the final measurement model for all factors at Time 1, Time 2, and Time 4.

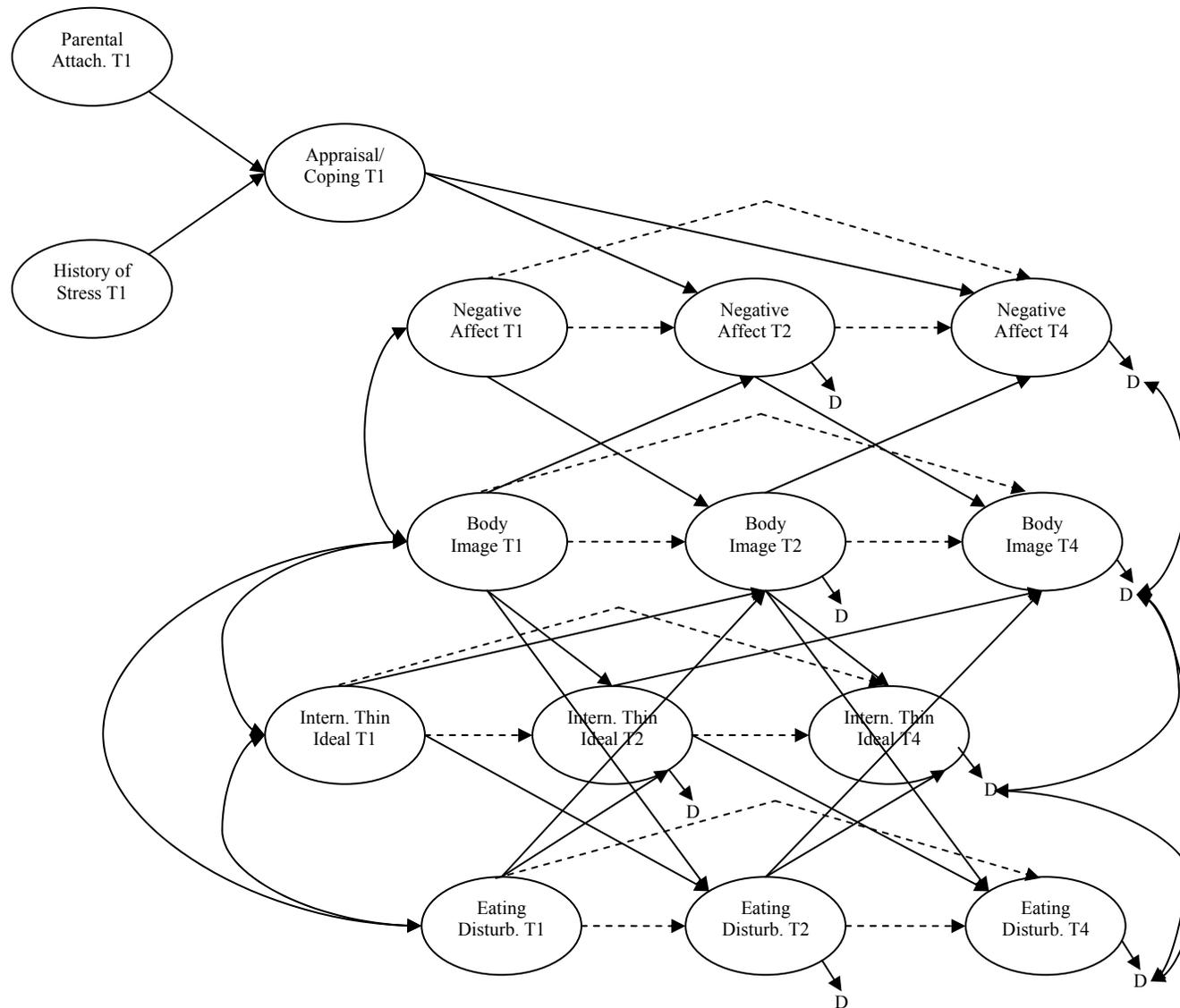


Figure 5. Diagram of structural Model A for all factors at Time 1, Time 2, and Time 4.

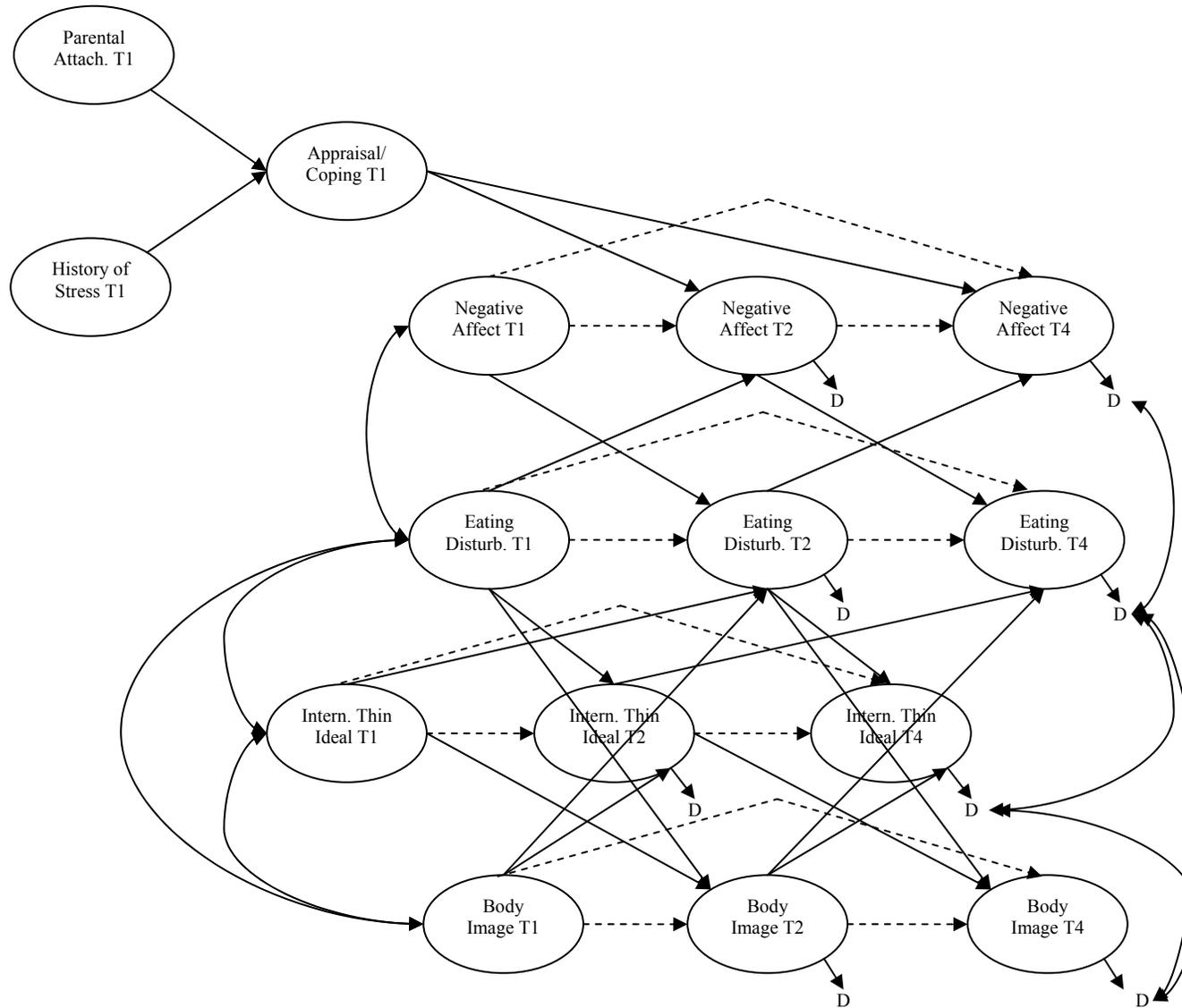


Figure 6. Diagram of the proposed structural Model B for Time 1, Time 2, and Time 4.

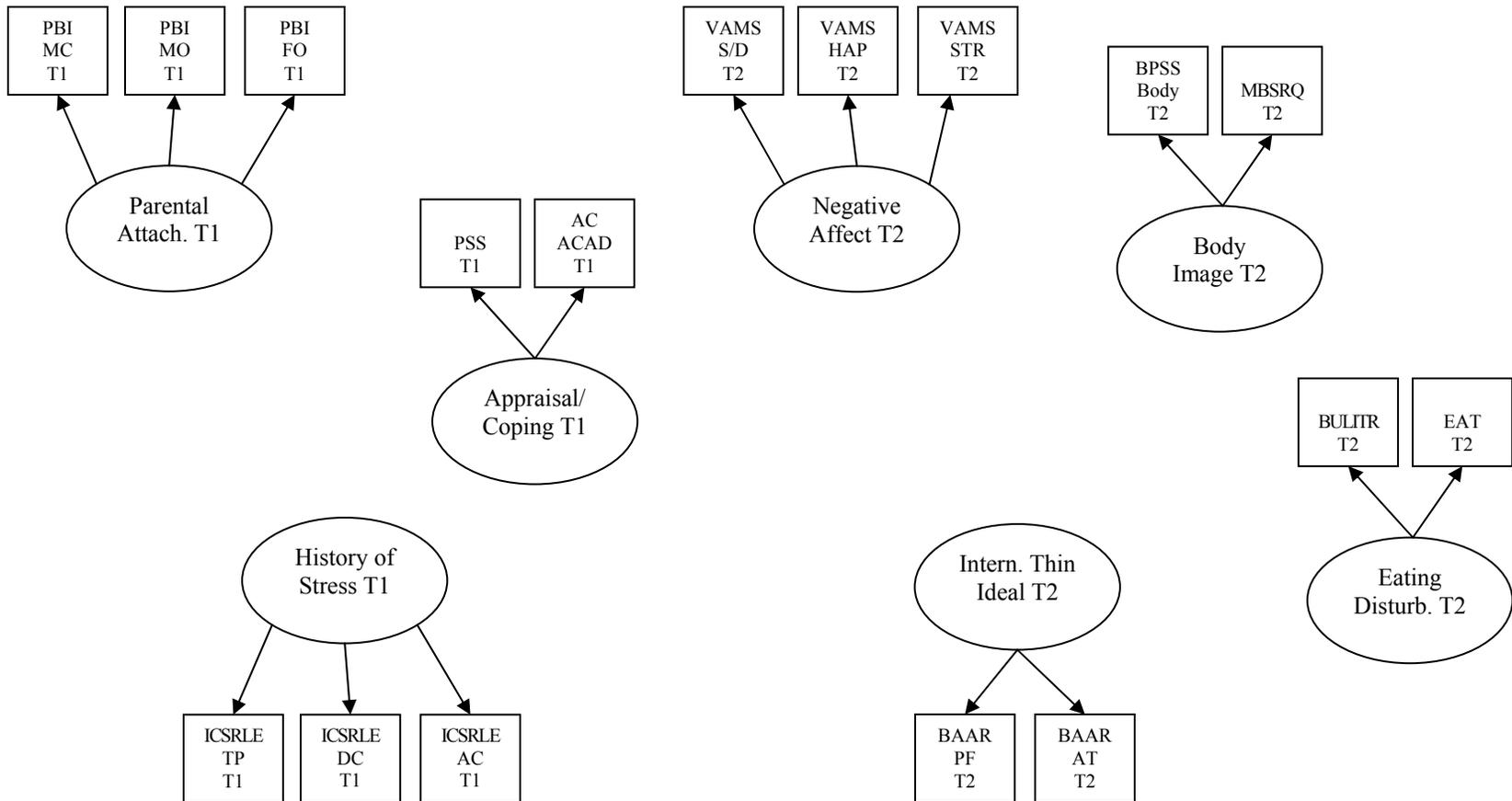


Figure 7. Basic diagram of the measurement model for factors at Time 1 and Time 2 - Parental Attachment Factor (T1), History of Stress Factor (T1), Appraisal/Coping Factor (T1), Negative Affect Factor (T2), Body Image Factor (T2), Internalization of Thin-Ideal Factor (T2), and the Eating Disturbance Factor (T2).

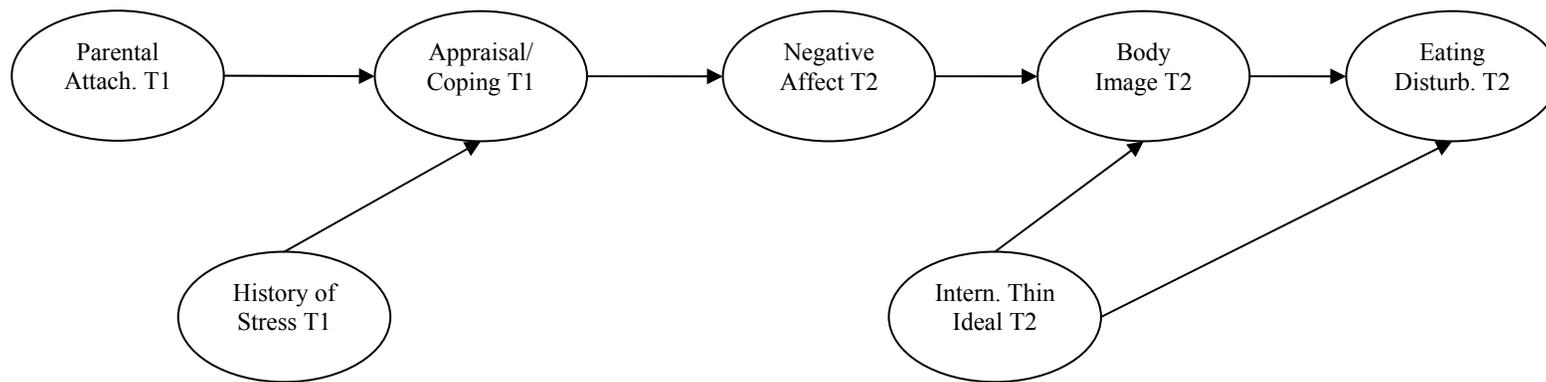


Figure 8. Basic diagram of the hypothesized structural Model A for factors at Time 1 and Time 2- Parental Attachment Factor (T1), History of Stress Factor (T1), Appraisal/Coping Factor (T1), Negative Affect Factor (T2), Body Image Factor (T2), Internalization of Thin-Ideal Factor (T2), and the Eating Disturbance Factor (T2).

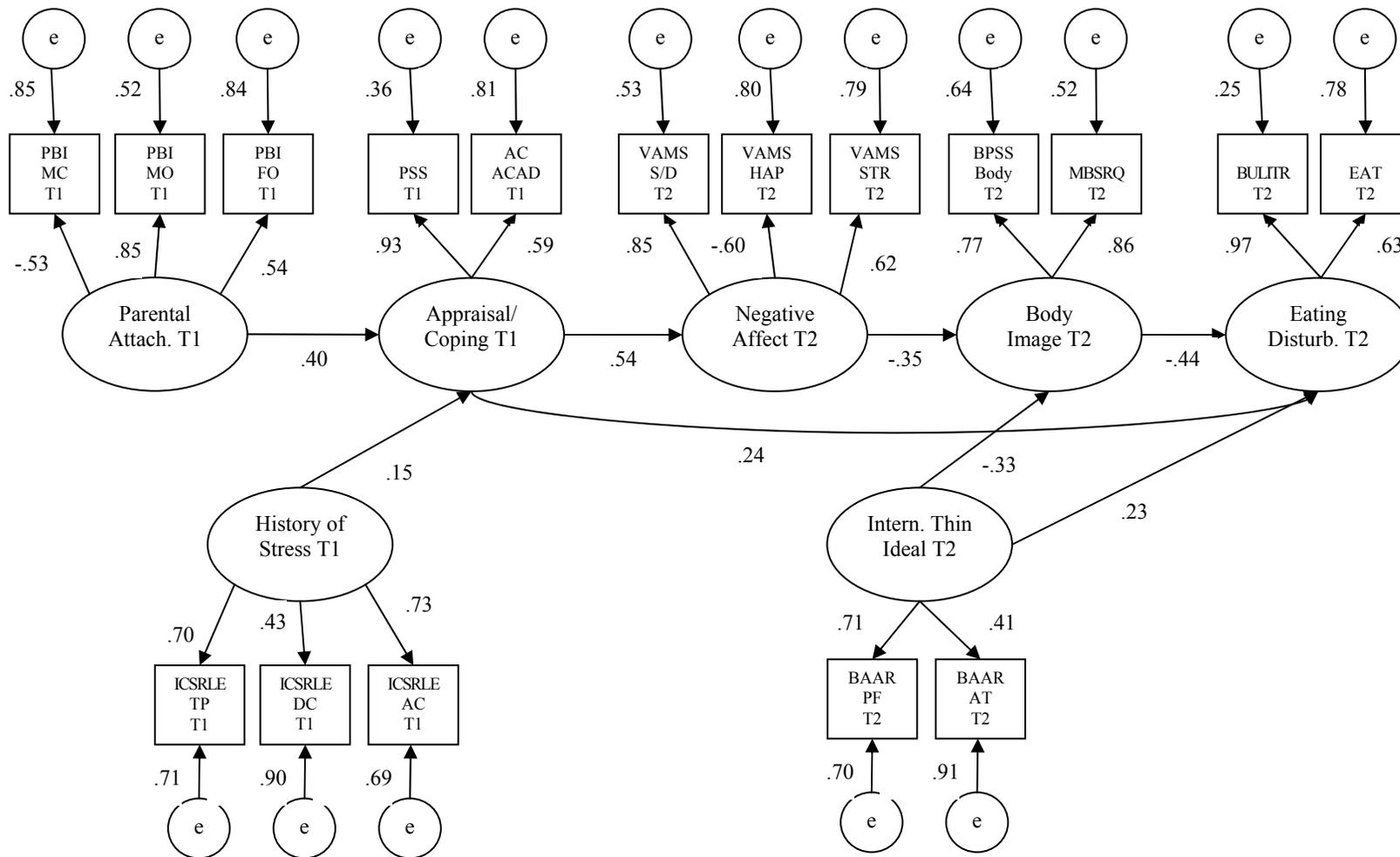


Figure 9. Diagram of structural Model A for factors at Time 1 and Time 2- Parental Attachment Factor (T1), History of Stress Factor (T1), Appraisal/Coping Factor (T1), Negative Affect Factor (T2), Body Image Factor (T2), Internalization of Thin-Ideal Factor (T2), and the Eating Disturbance Factor (T2).

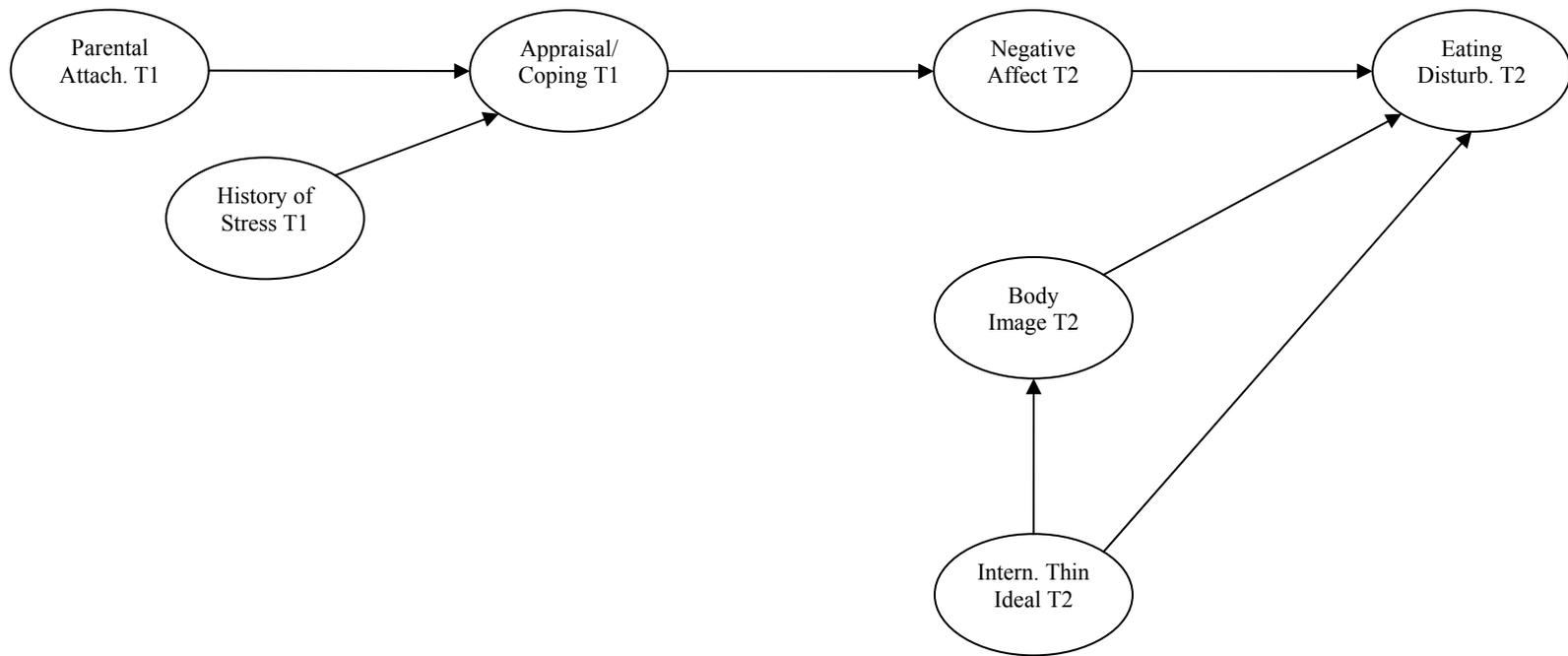


Figure 10. Basic diagram of the hypothesized structural Model B for factors at Time 1 and Time 2 - Parental Attachment Factor (T1), History of Stress Factor (T1), Appraisal/Coping Factor (T1), Negative Affect Factor (T2), Body Image Factor (T2), Internalization of Thin-Ideal Factor (T2), and the Eating Disturbance Factor (T2).

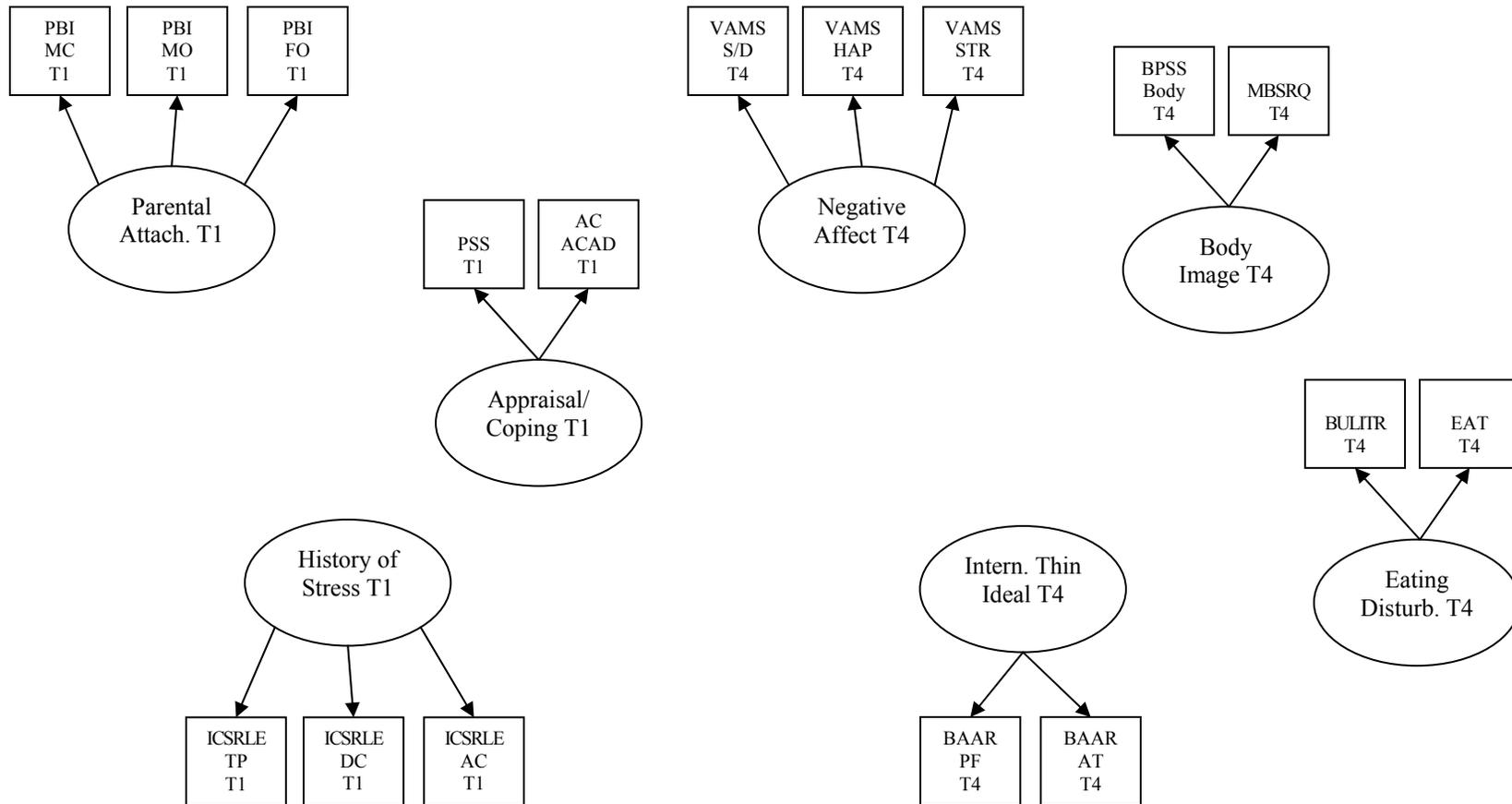


Figure 11. Basic diagram of the measurement model for factors at Time 1 and Time 4 - Parental Attachment Factor (T1), History of Stress Factor (T1), Appraisal/Coping Factor (T1), Negative Affect Factor (T4), Body Image Factor (T4), Internalization of Thin-Ideal Factor (T4), and the Eating Disturbance Factor (T4).

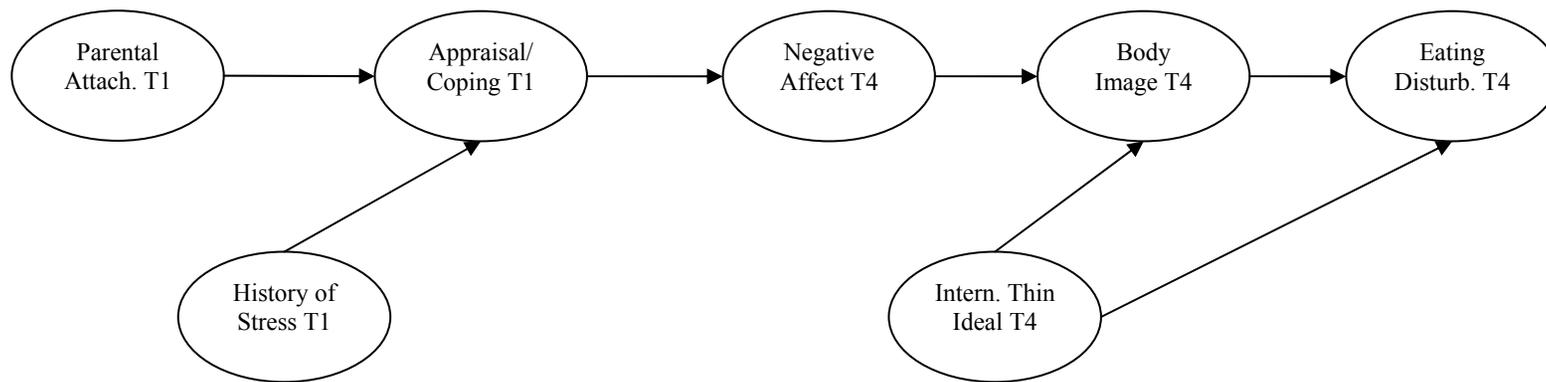


Figure 12. Basic diagram of hypothesized structural Model A for factors at Time 1 and Time 4 - Parental Attachment Factor (T1), History of Stress Factor (T1), Appraisal/Coping Factor (T1), Negative Affect Factor (T4), Body Image Factor (T4), Internalization of Thin-Ideal Factor (T4), and the Eating Disturbance Factor (T4).

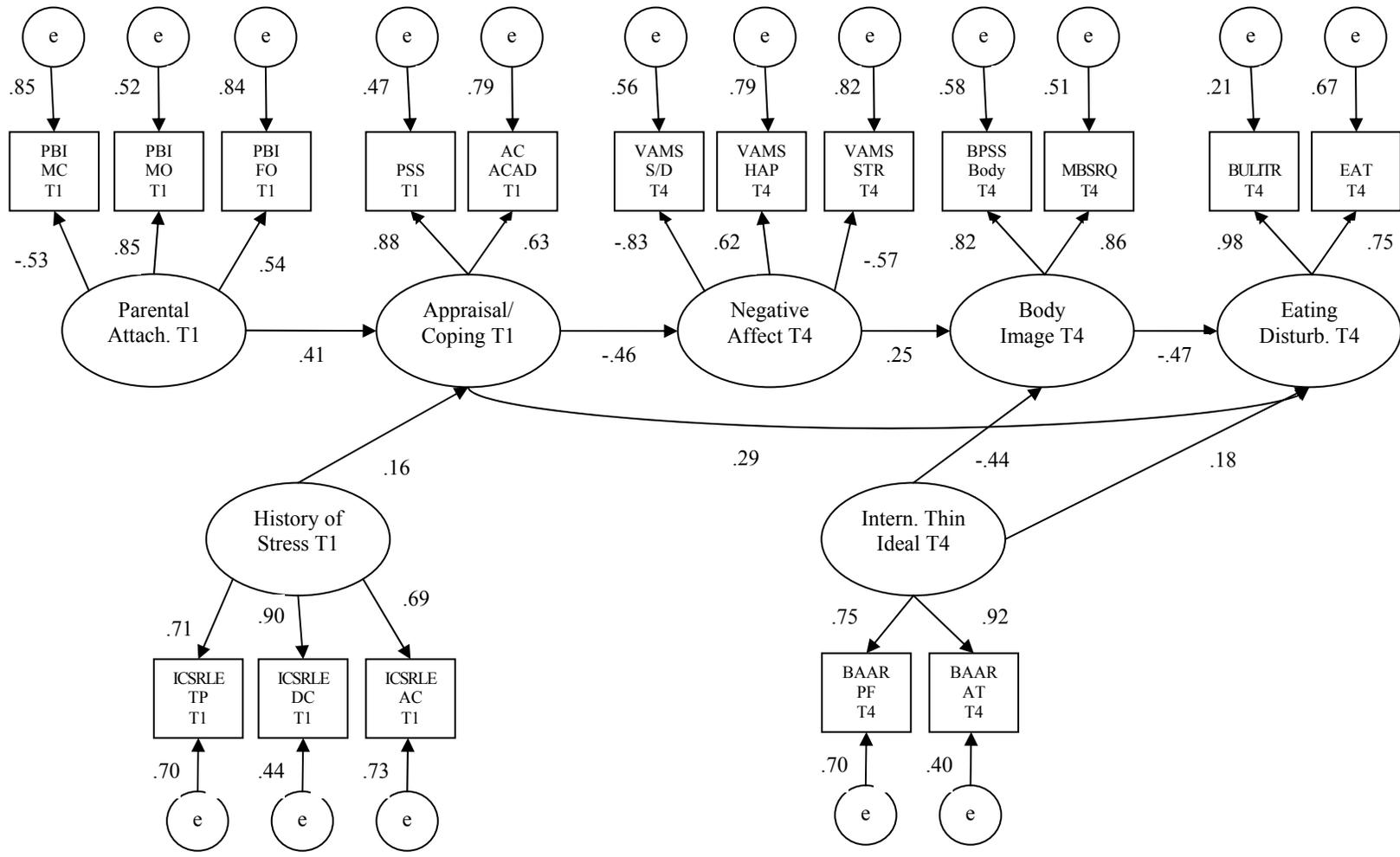


Figure 13. Diagram of structural Model A for factors at Time 1 and Time 4 - Parental Attachment Factor (T1), History of Stress Factor (T1), Appraisal/Coping Factor (T1), Negative Affect Factor (T4), Body Image Factor (T4), Internalization of Thin-Ideal Factor (T4), and the Eating Disturbance Factor (T4).

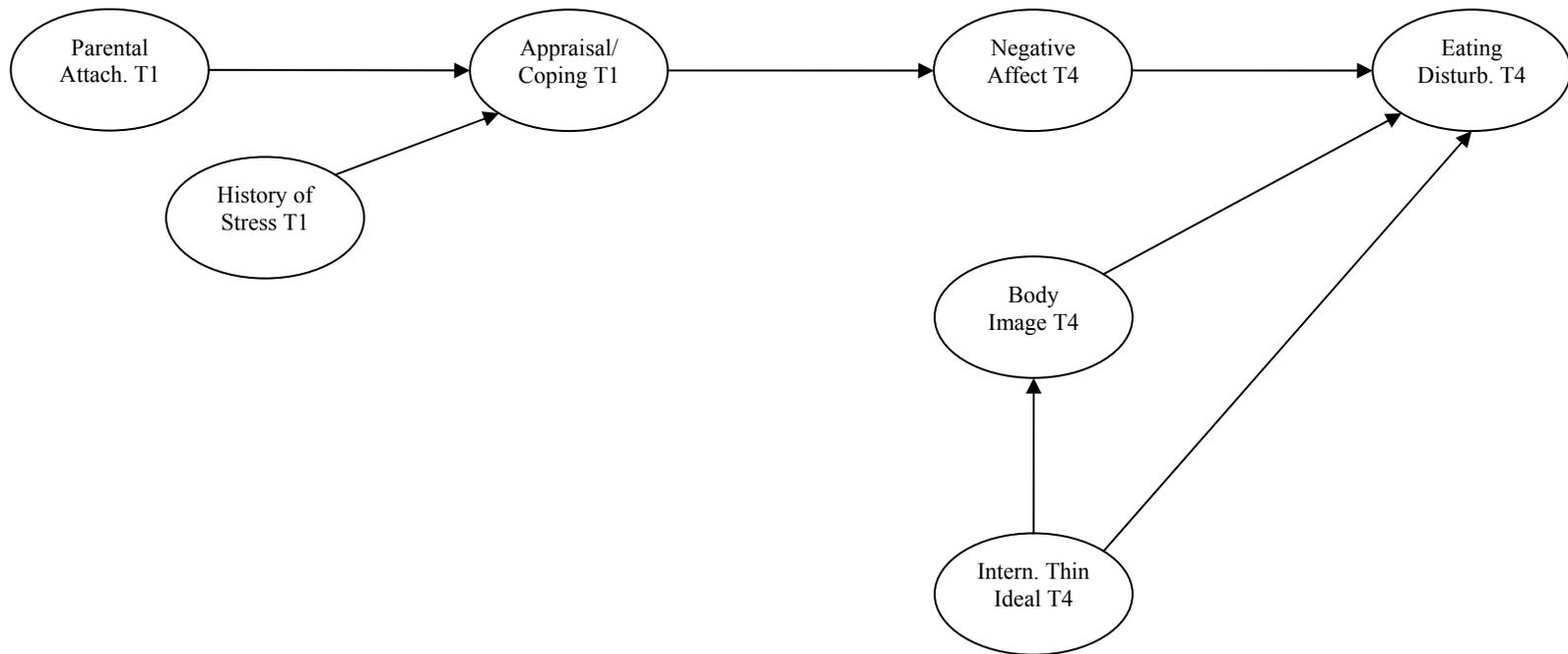


Figure 14. Diagram of structural Model B for factors at Time 1 and Time 4 - Parental Attachment Factor (T1), History of Stress Factor (T1), Appraisal/Coping Factor (T1), Negative Affect Factor (T4), Body Image Factor (T4), Internalization of Thin-Ideal Factor (T4), and the Eating Disturbance Factor (T4).

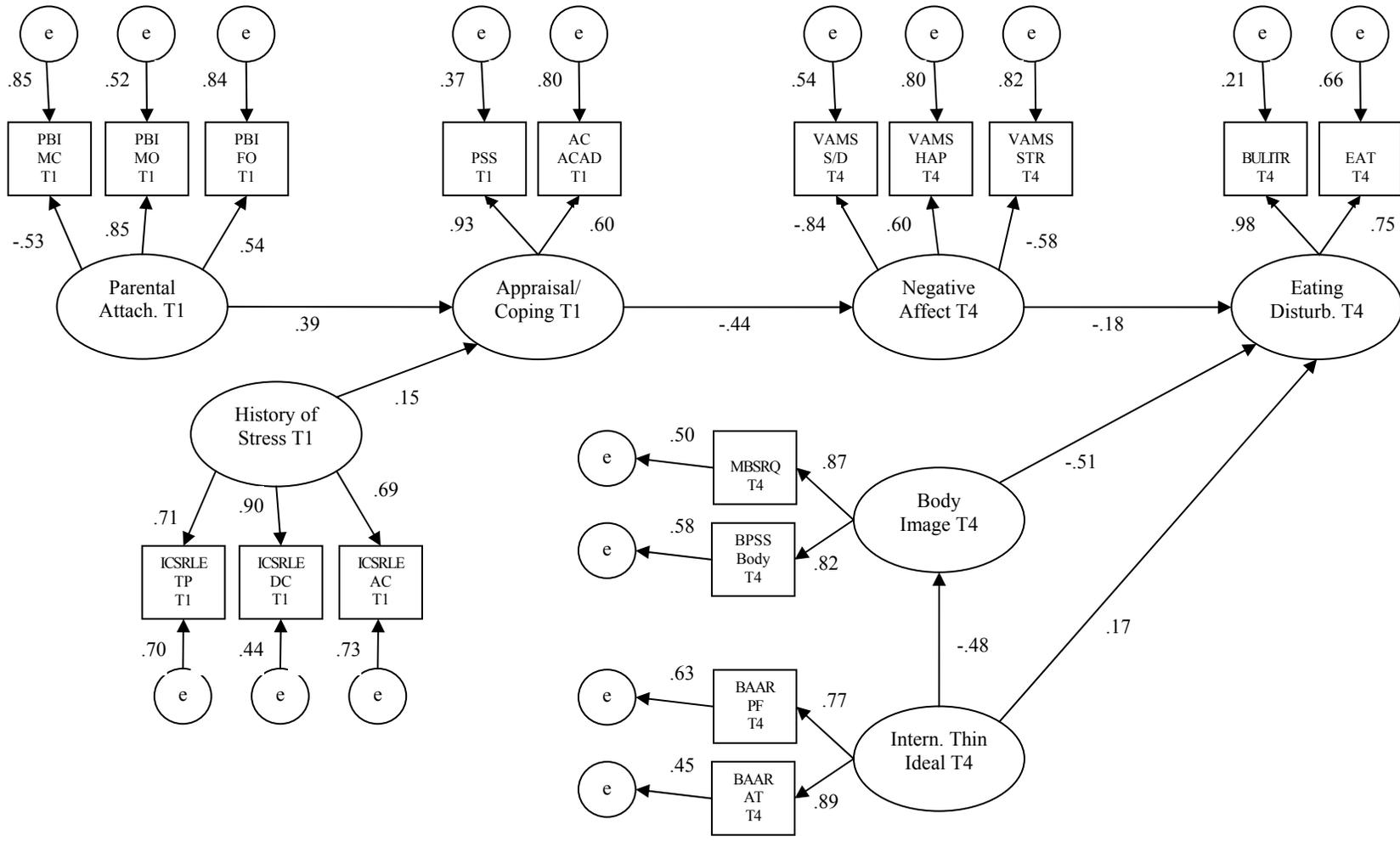


Figure 15. Diagram of structural Model B for factors at Time 1 and Time 4 - Parental Attachment Factor (T1), History of Stress Factor (T1), Appraisal/Coping Factor (T1), Negative Affect Factor (T4), Body Image Factor (T4), Internalization of Thin-Ideal Factor (T4), and the Eating Disturbance Factor (T4).

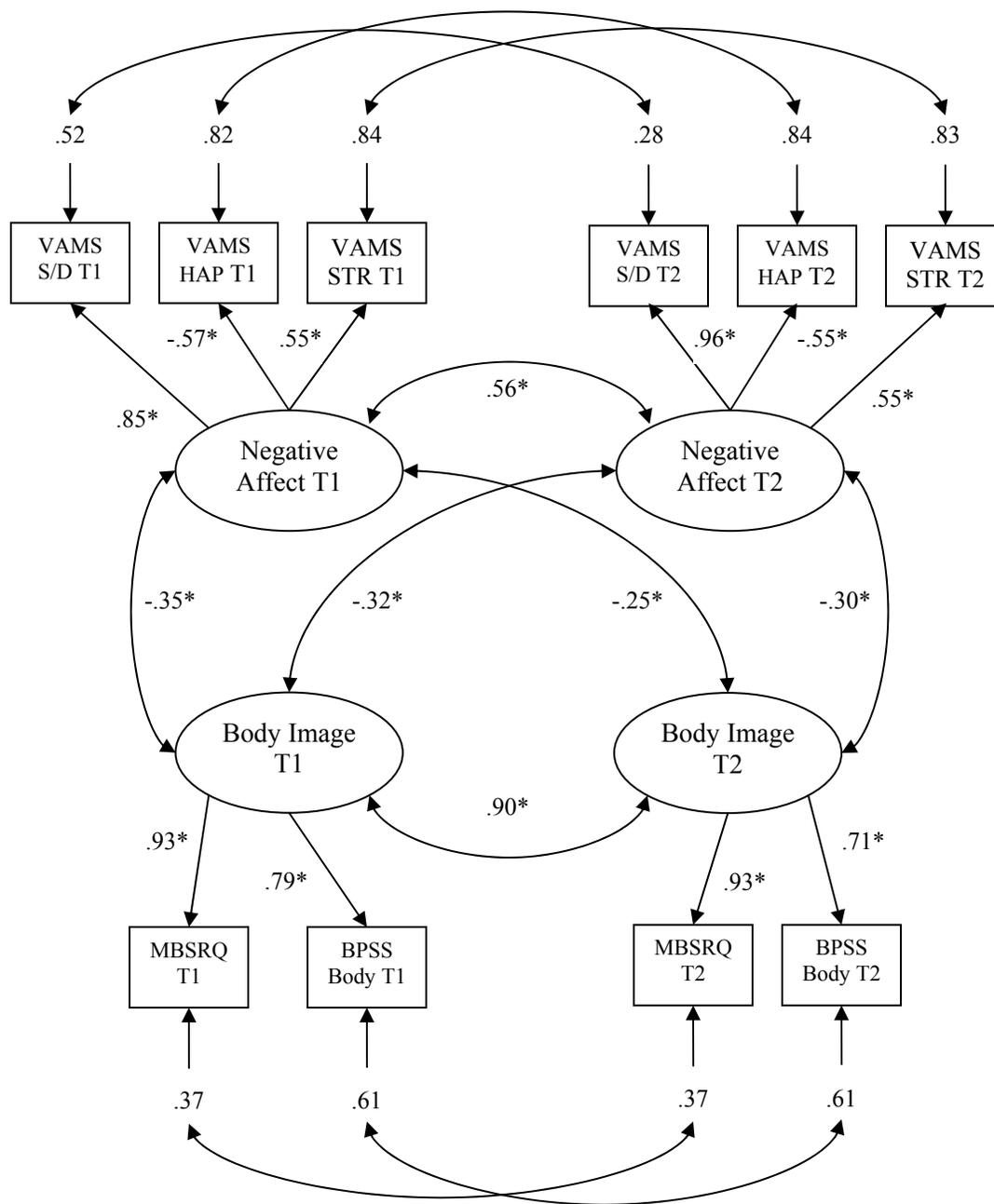
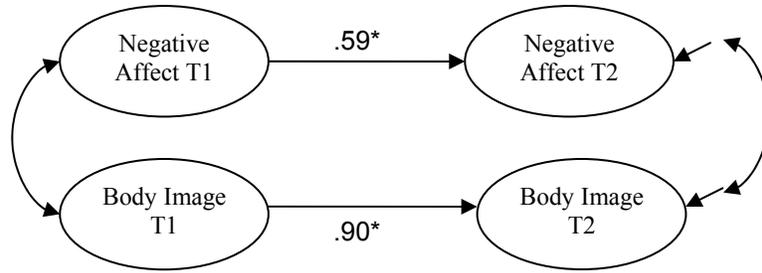


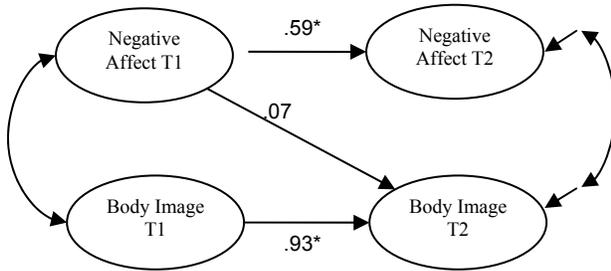
Figure 16. Measurement model for the relations between negative affect and body image at Time 1 and Time 2. * = $p < .05$.



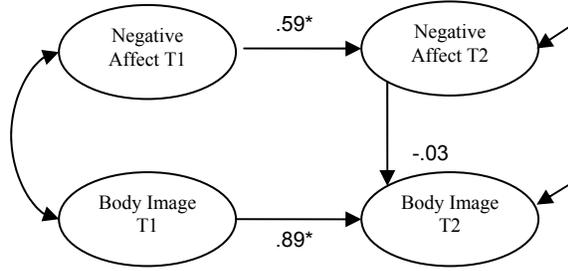
Baseline Model

Cross- Lagged Effects

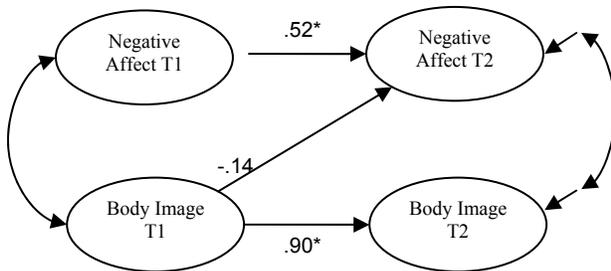
Simultaneous Effects



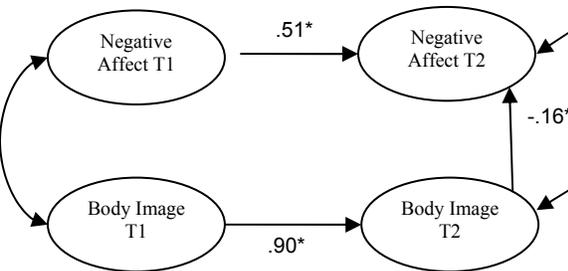
Model 1



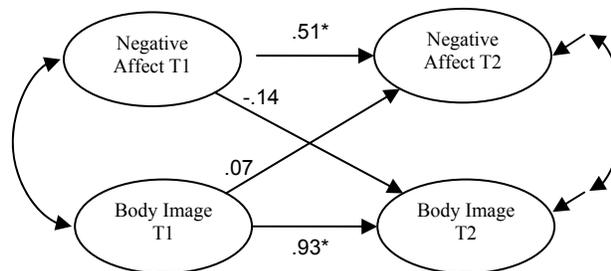
Model 4



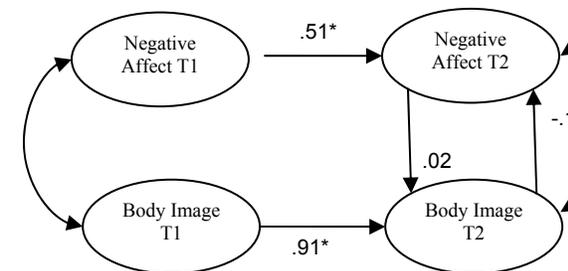
Model 2



Model 5



Model 3



Model 6

Figure 17. Structural model for the relations between negative affect and body image at Time 1 and Time 2. * = $p < .05$.

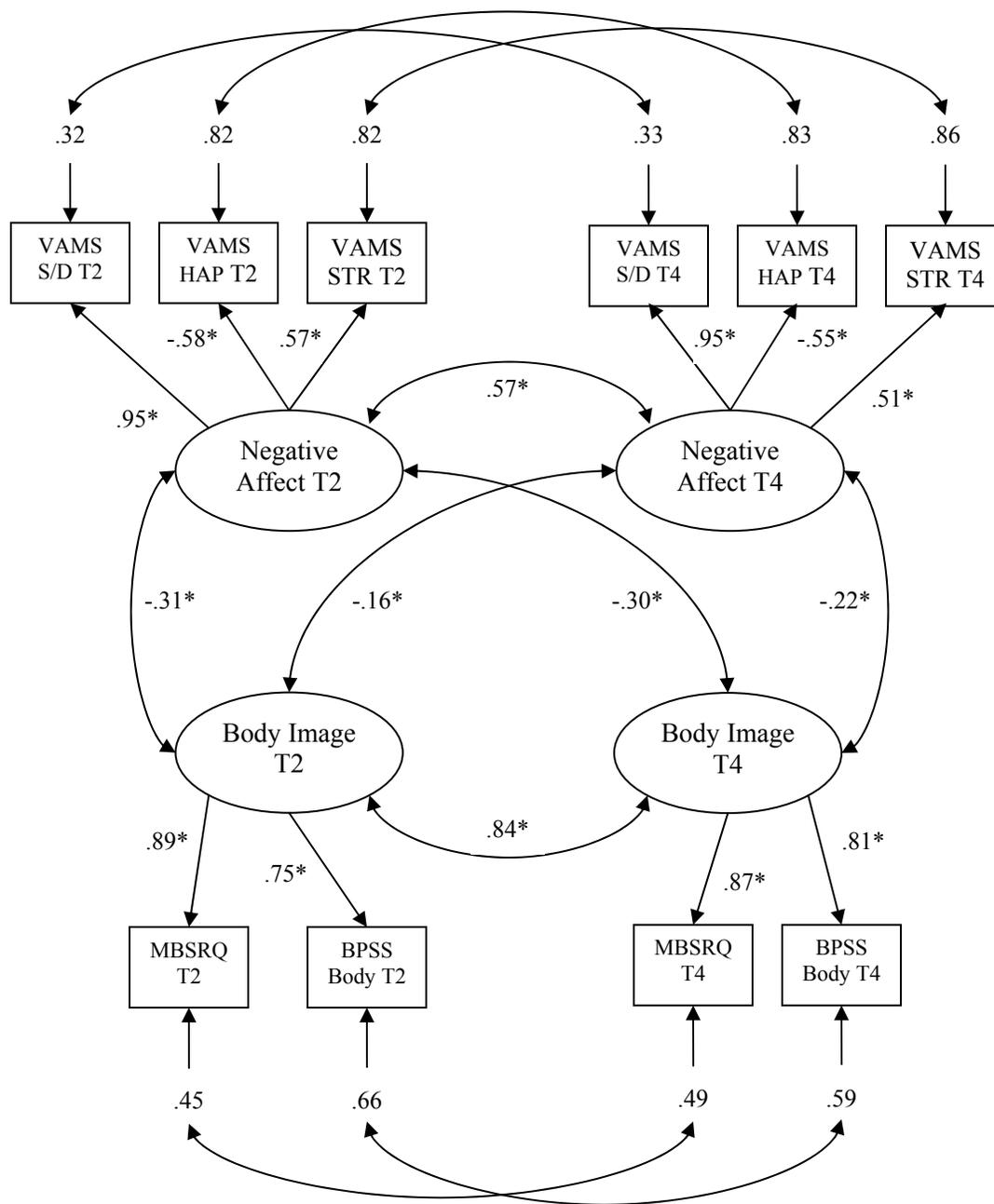
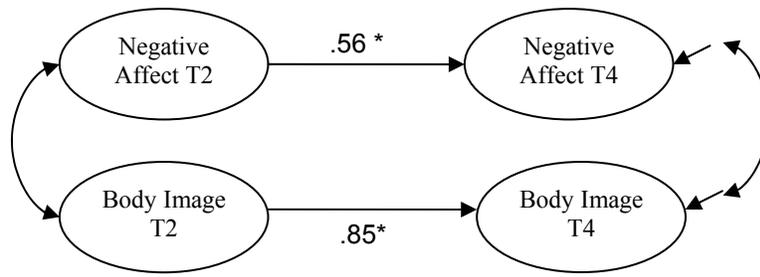


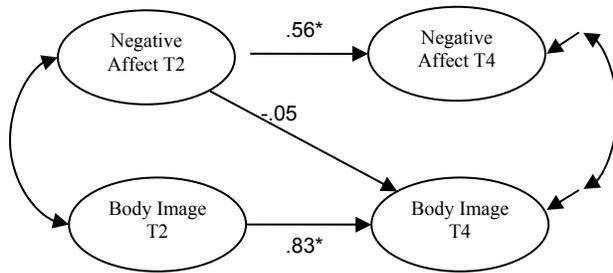
Figure 18. Measurement model for the relations between negative affect and body image at Time 2 and Time 4. * = $p < .05$.



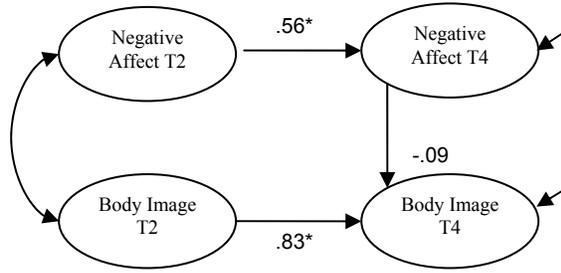
Baseline Model

Cross- Lagged Effects

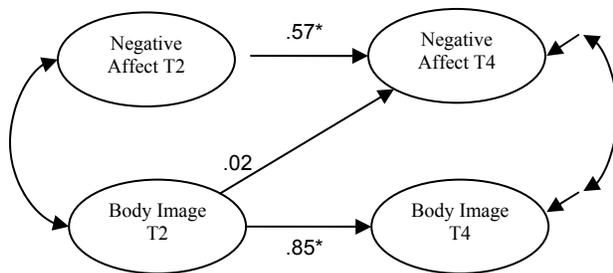
Simultaneous Effects



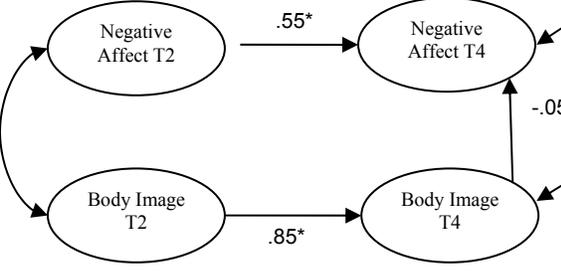
Model 1



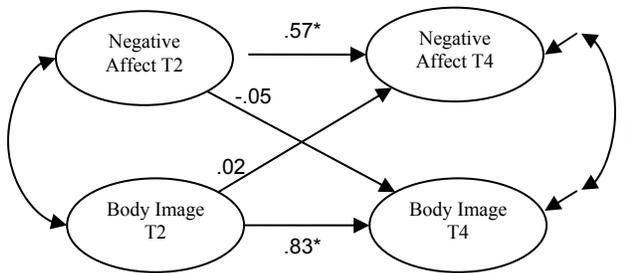
Model 4



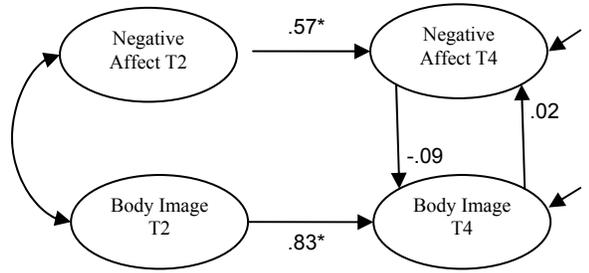
Model 2



Model 5



Model 3



Model 6

Figure 19. Structural model for the relations between negative affect and body image at Time 2 and Time 4. * = $p < .05$.

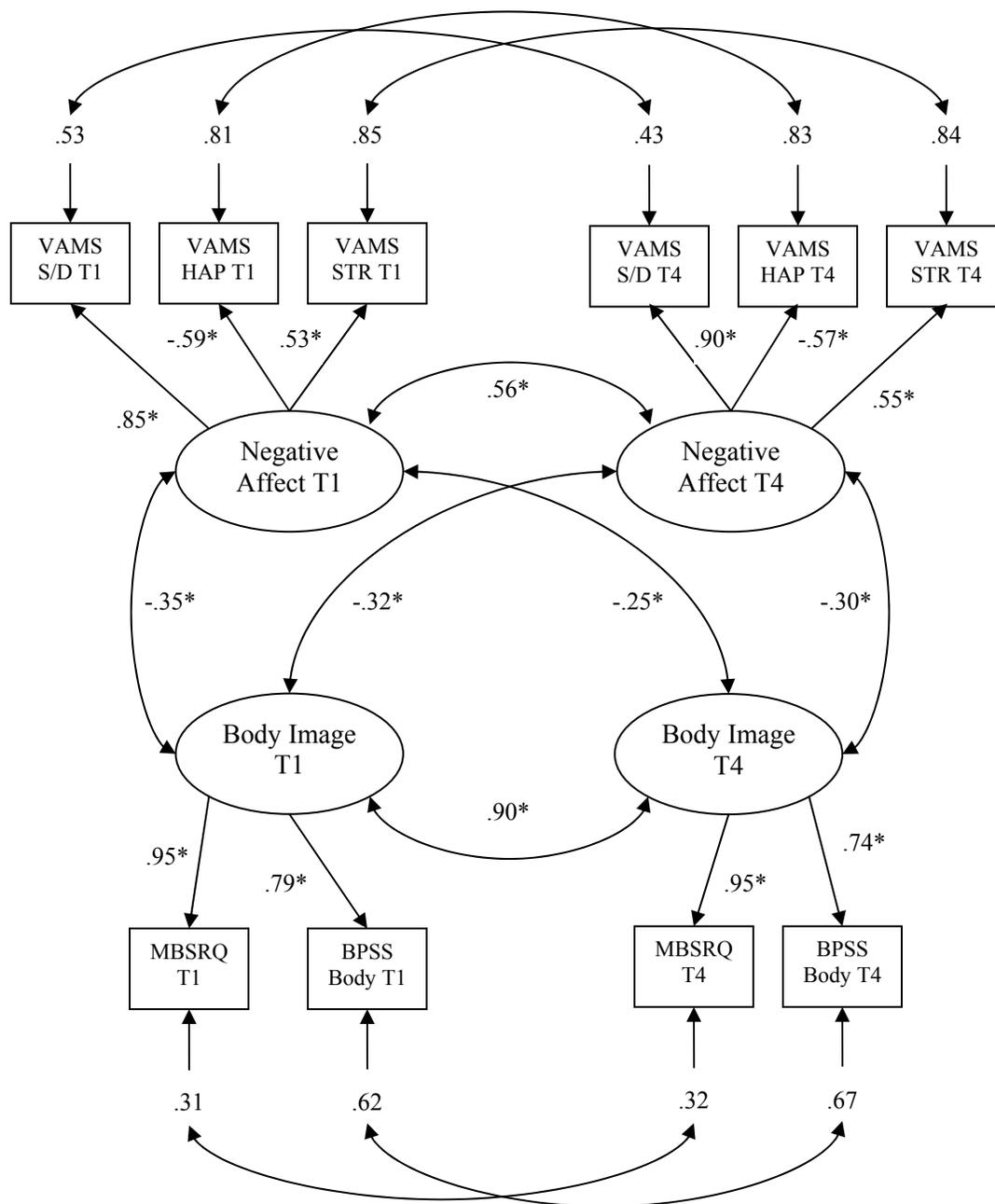
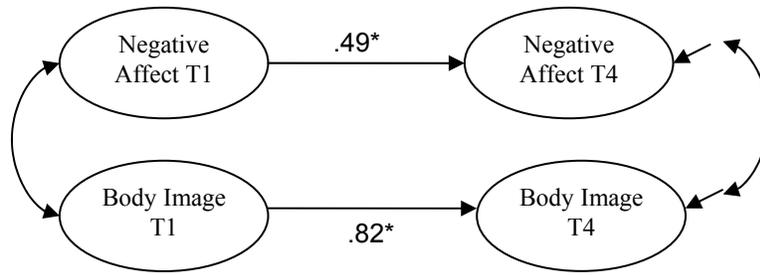


Figure 20. Measurement model for the relations between negative affect and body image at Time 1 and Time 4. * = $p < .05$.



Cross- Lagged Effects

Simultaneous Effects

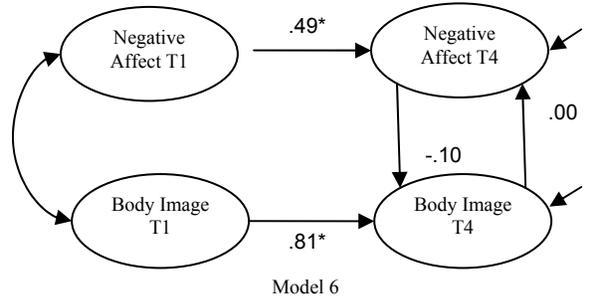
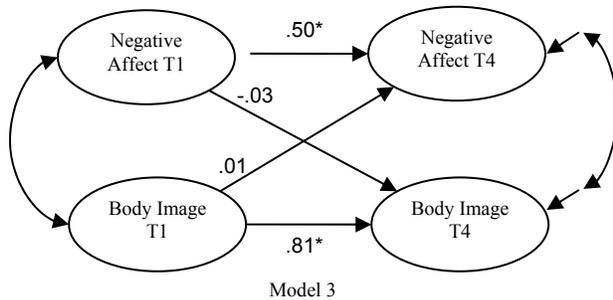
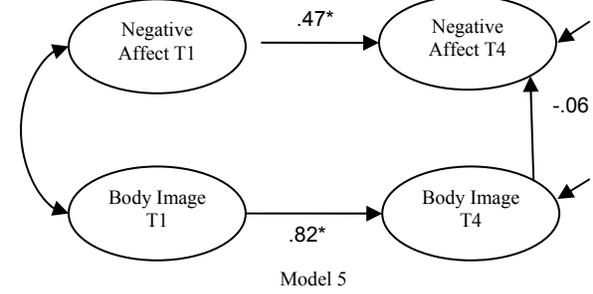
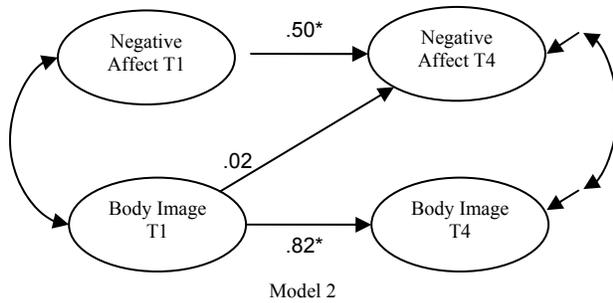
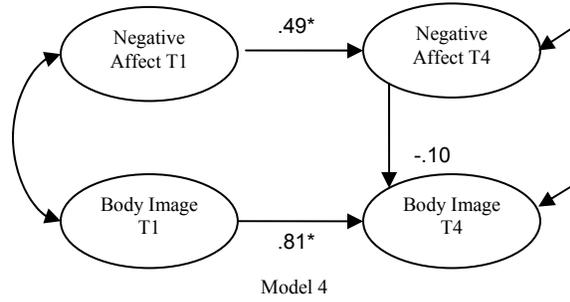
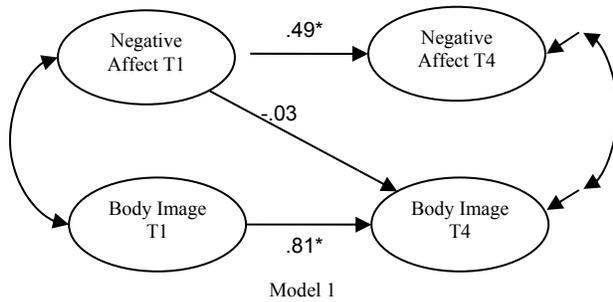


Figure 21. Structural model for the relations between negative affect and body image at Time 1 and Time 4. * = $p < .05$.

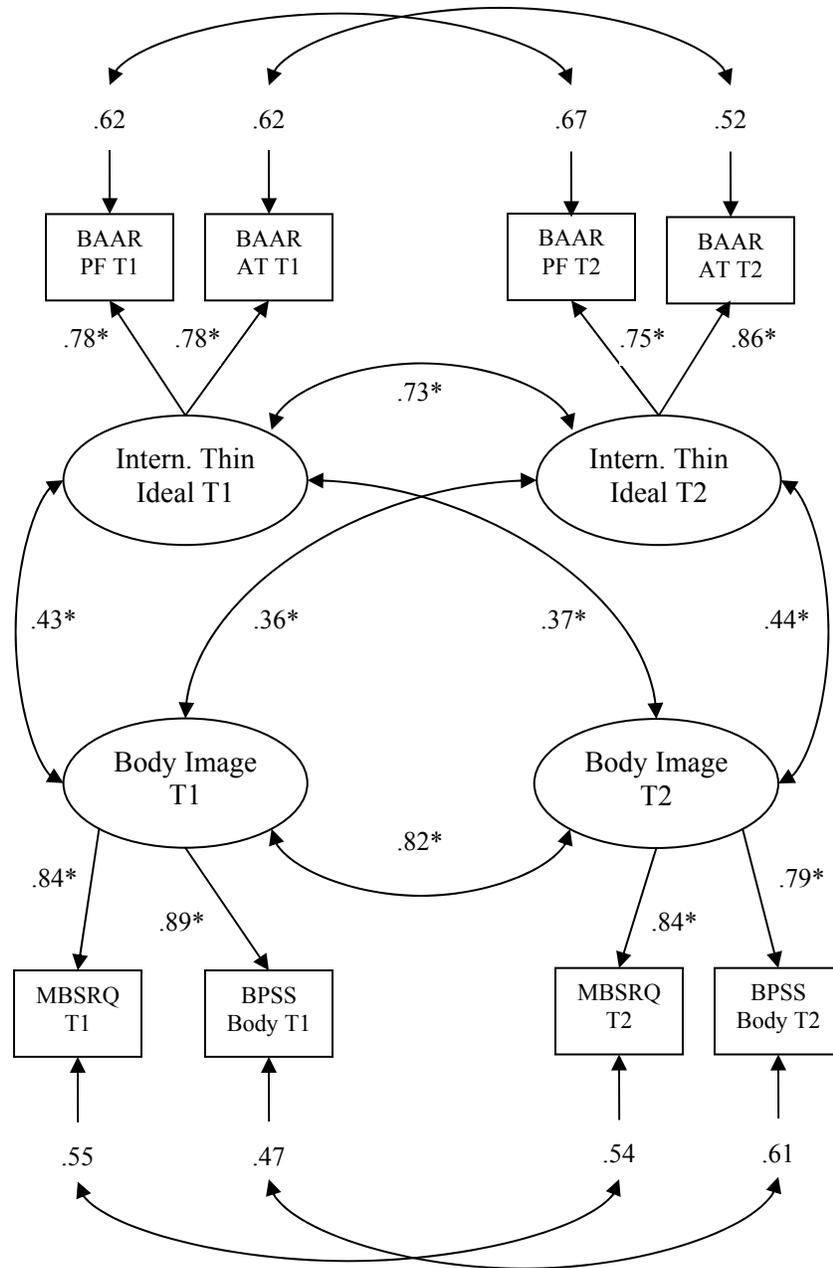
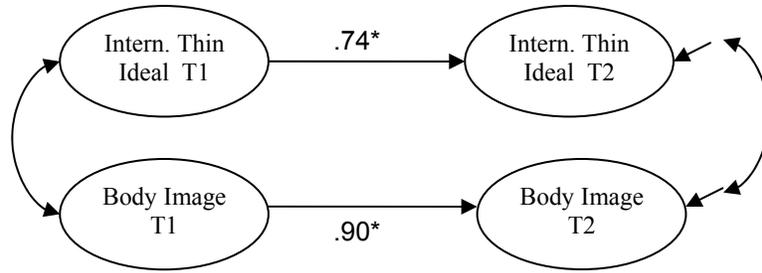
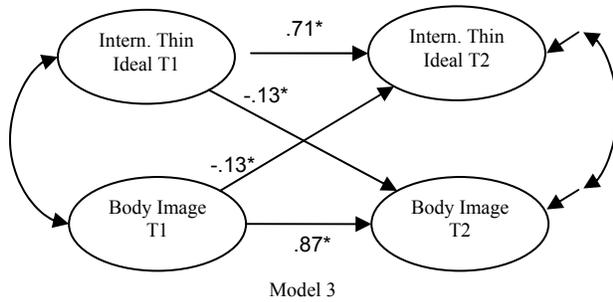
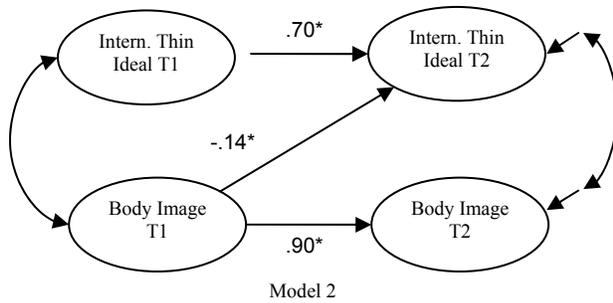
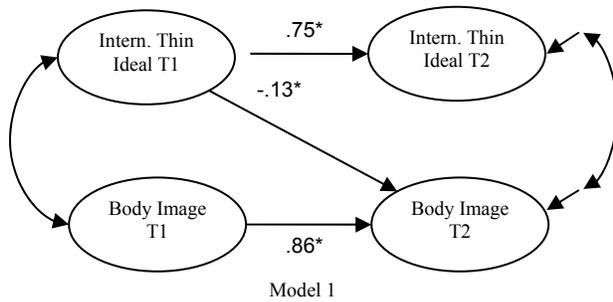


Figure 22. Measurement model for the relations between internalization of thin-ideal and body image at Time 1 and Time 2. * = $p < .05$.



Cross- Lagged Effects



Simultaneous Effects

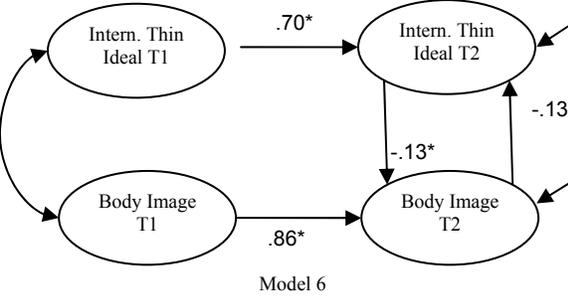
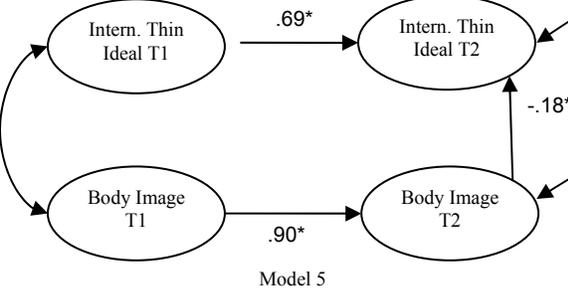
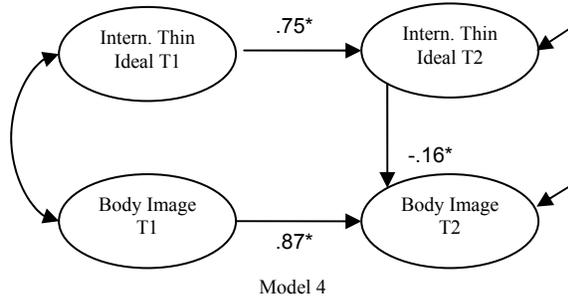


Figure 23. Structural model for the relations between internalization of thin-ideal and body image at Time 1 and Time 2. * = $p < .05$.

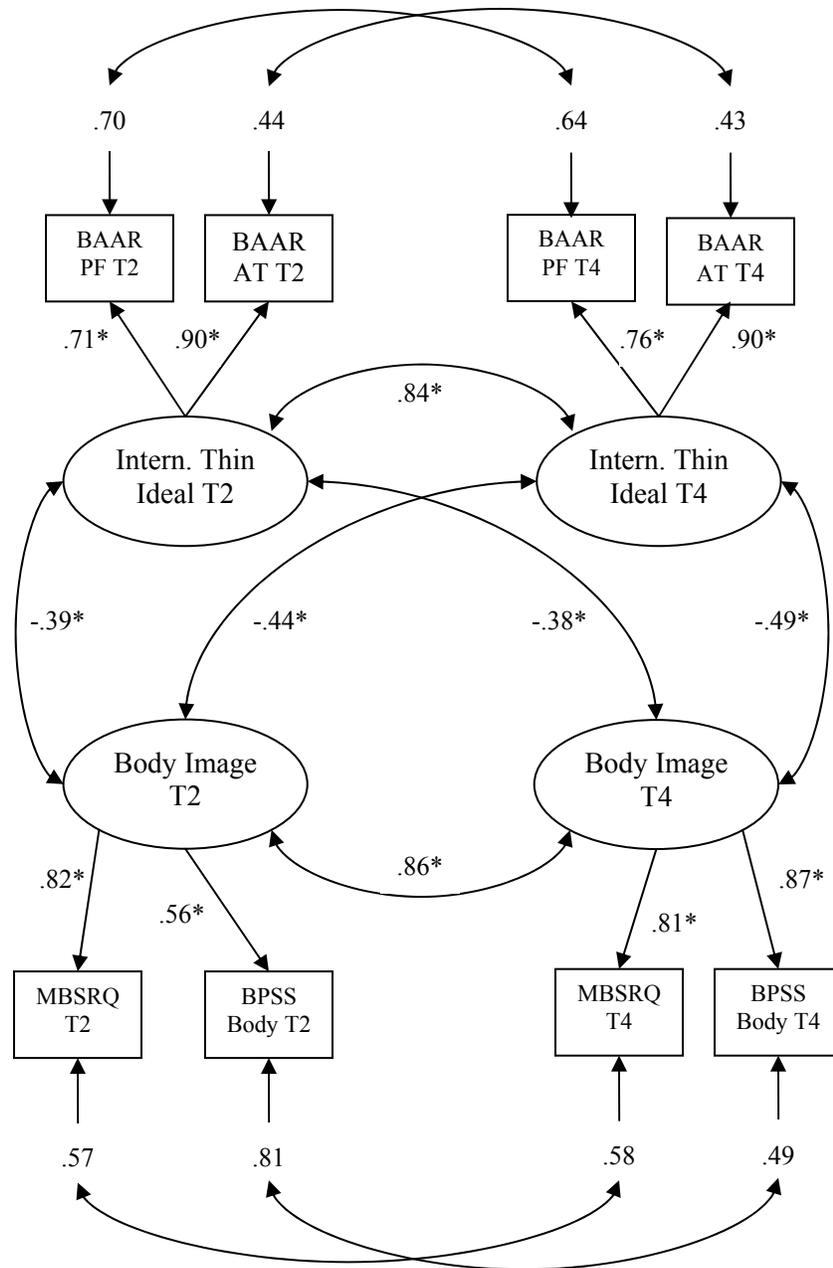
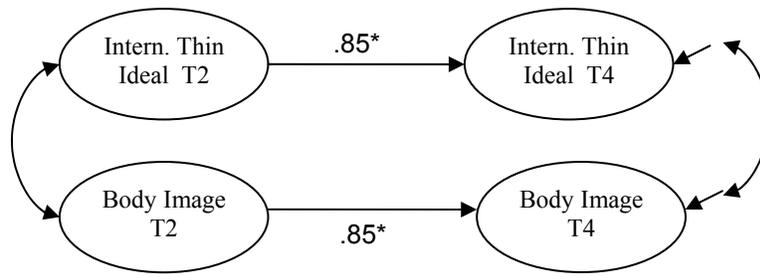


Figure 24. Measurement model for the relations between internalization of thin-ideal and body image at Time 2 and Time 4. * = $p < .05$.



Cross- Lagged Effects

Simultaneous Effects

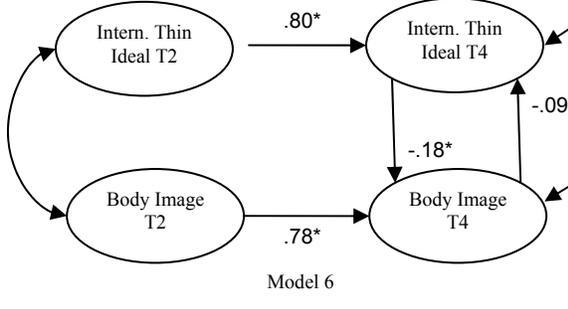
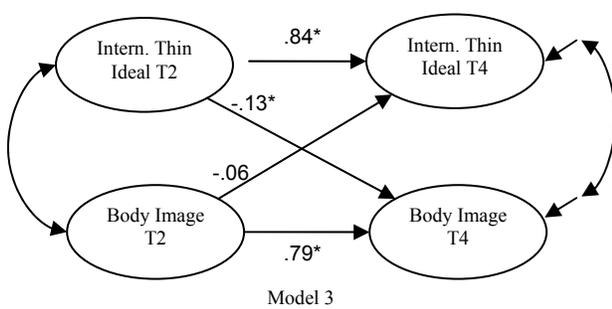
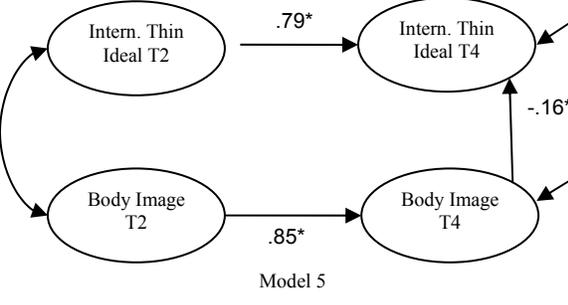
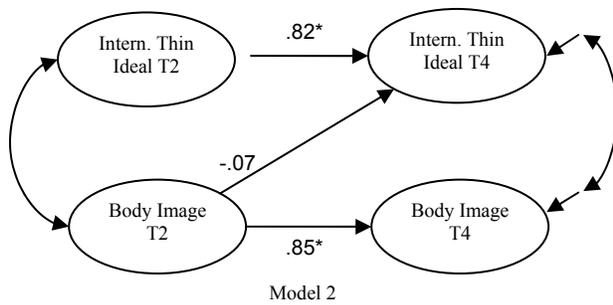
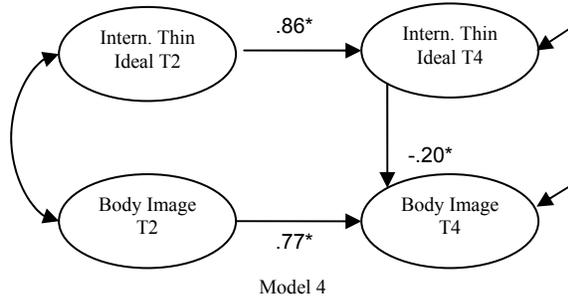
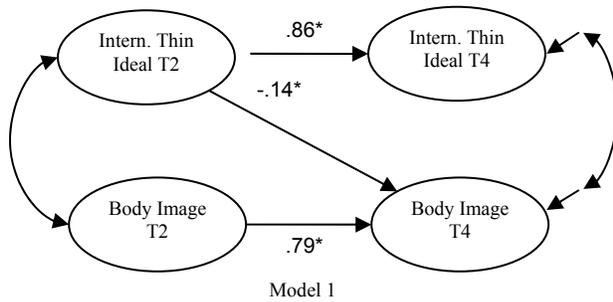


Figure 25. Structural model for the relations between internalization of thin-ideal and body image at Time 2 and Time 4. * = $p < .05$.

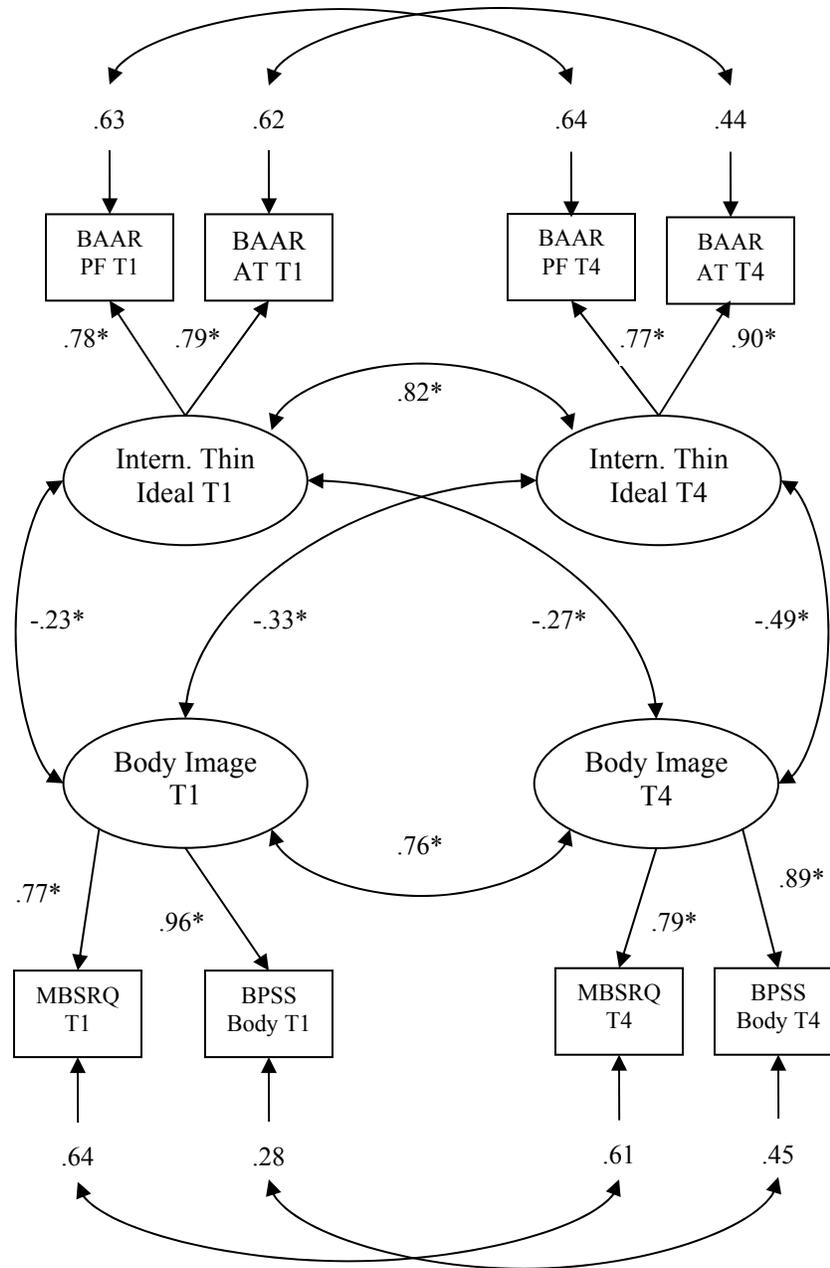
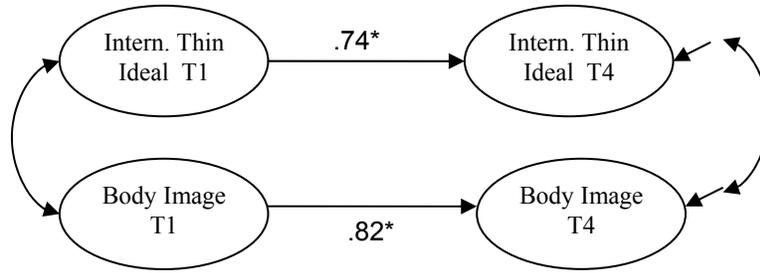


Figure 26. Measurement model for the relations between internalization of thin-ideal and body image at Time 1 and Time 4. * = $p < .05$.



Cross- Lagged Effects

Simultaneous Effects

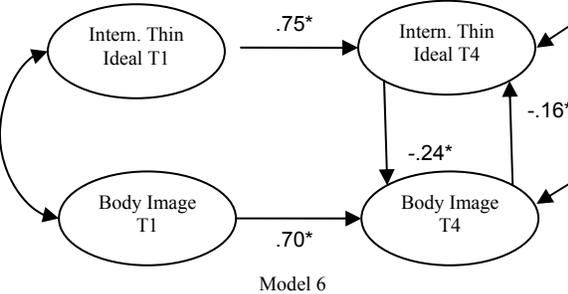
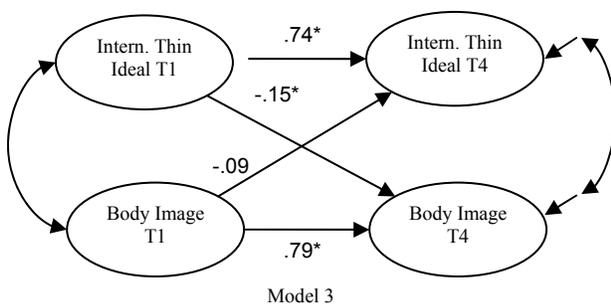
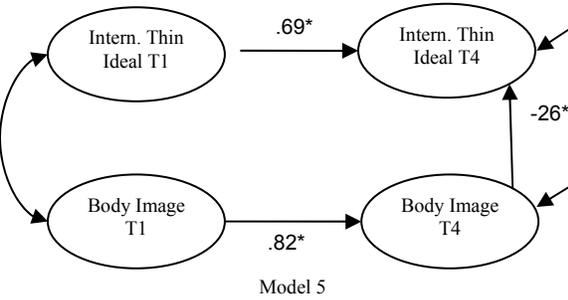
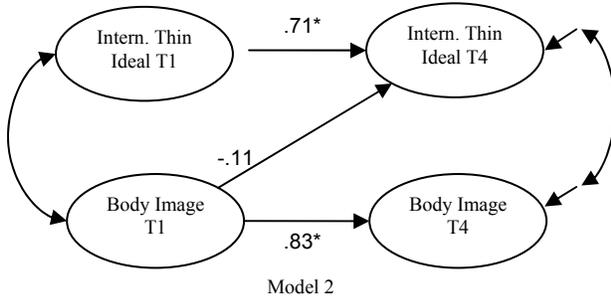
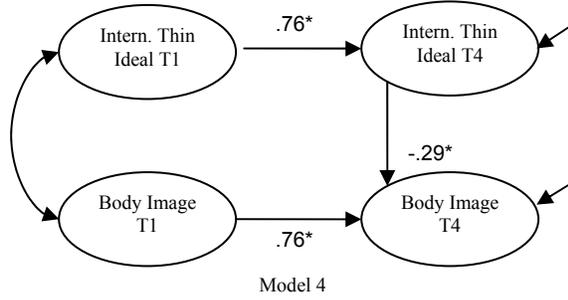
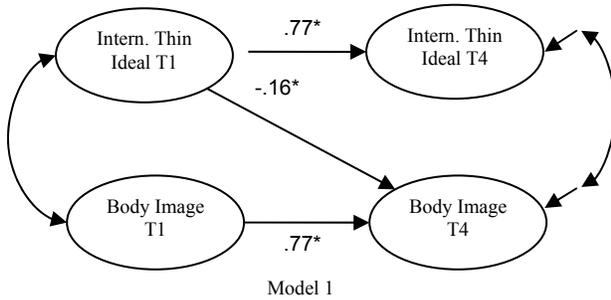


Figure 27. Structural model for the relations between internalization of thin-ideal and body image at Time 1 and Time 4. $* = p < .05$.

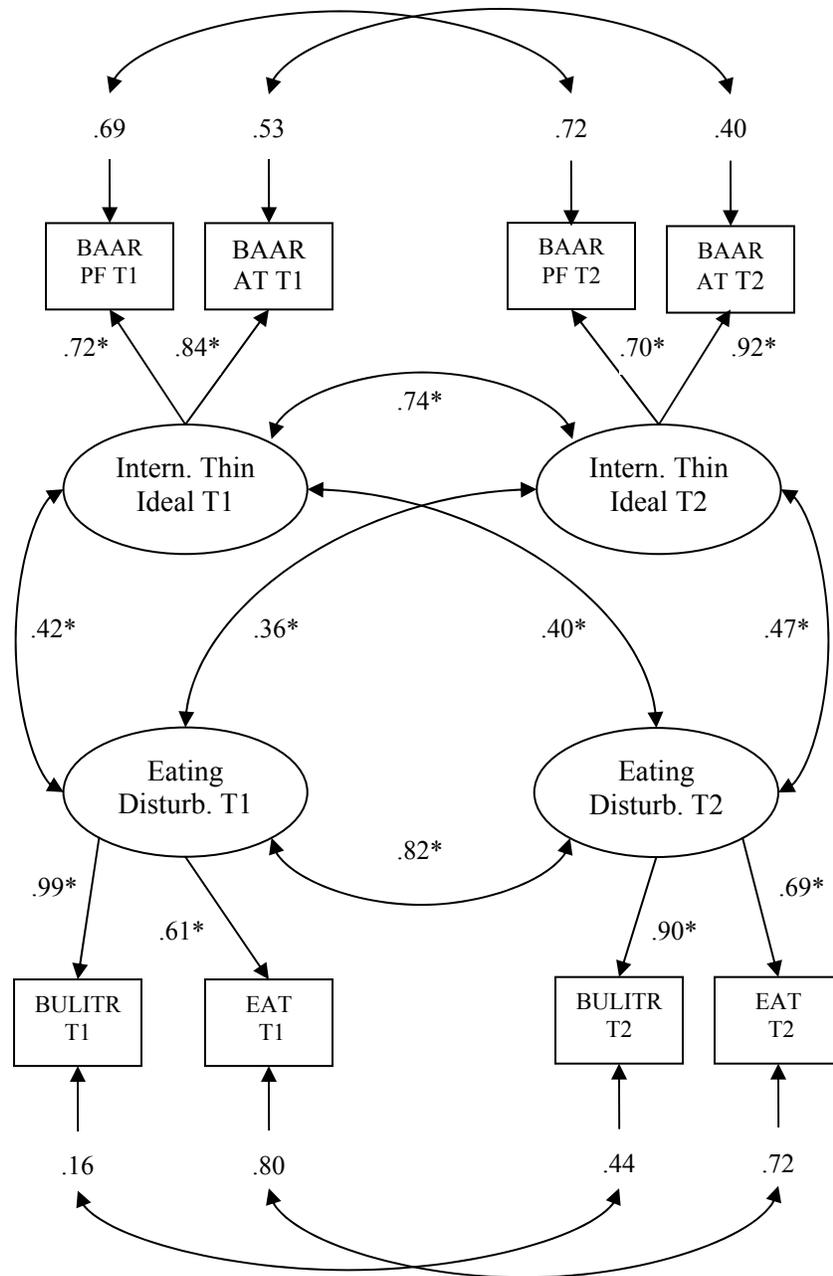
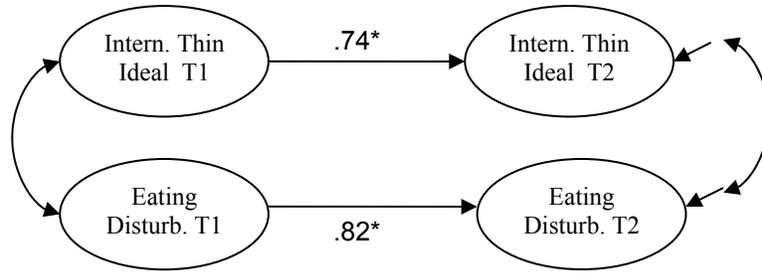


Figure 28. Measurement model for the relations between internalization of thin-ideal and eating disturbance at Time 1 and Time 2. * = $p < .05$.



Cross- Lagged Effects

Simultaneous Effects

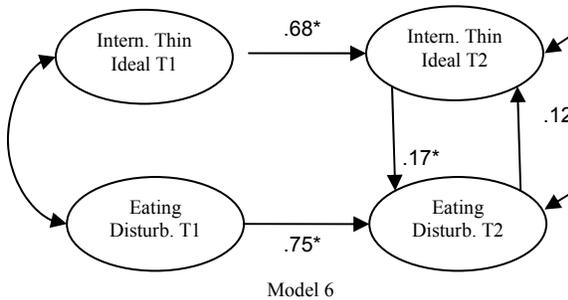
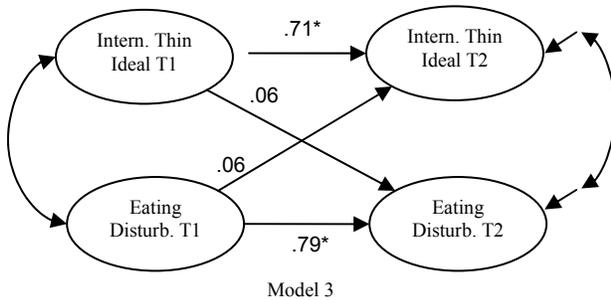
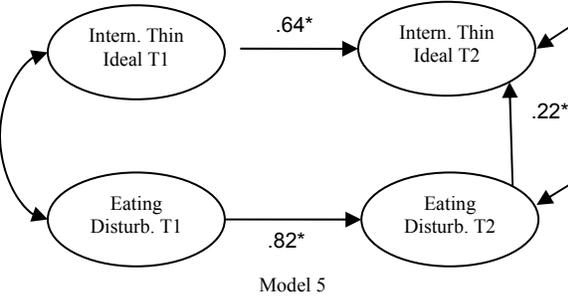
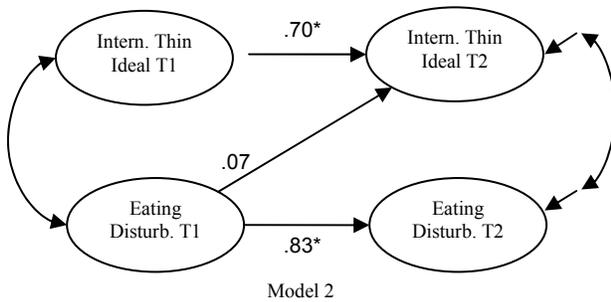
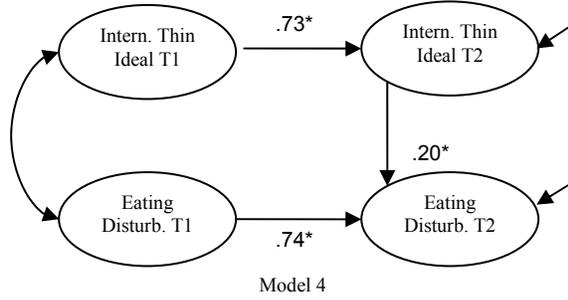
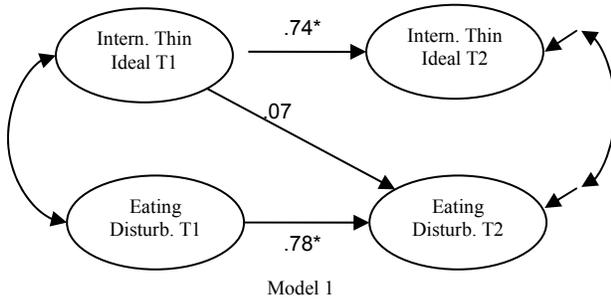


Figure 29. Structural model for the relations between internalization of thin-ideal and eating disturbance at Time 1 and Time 2. $* = p < .05$.

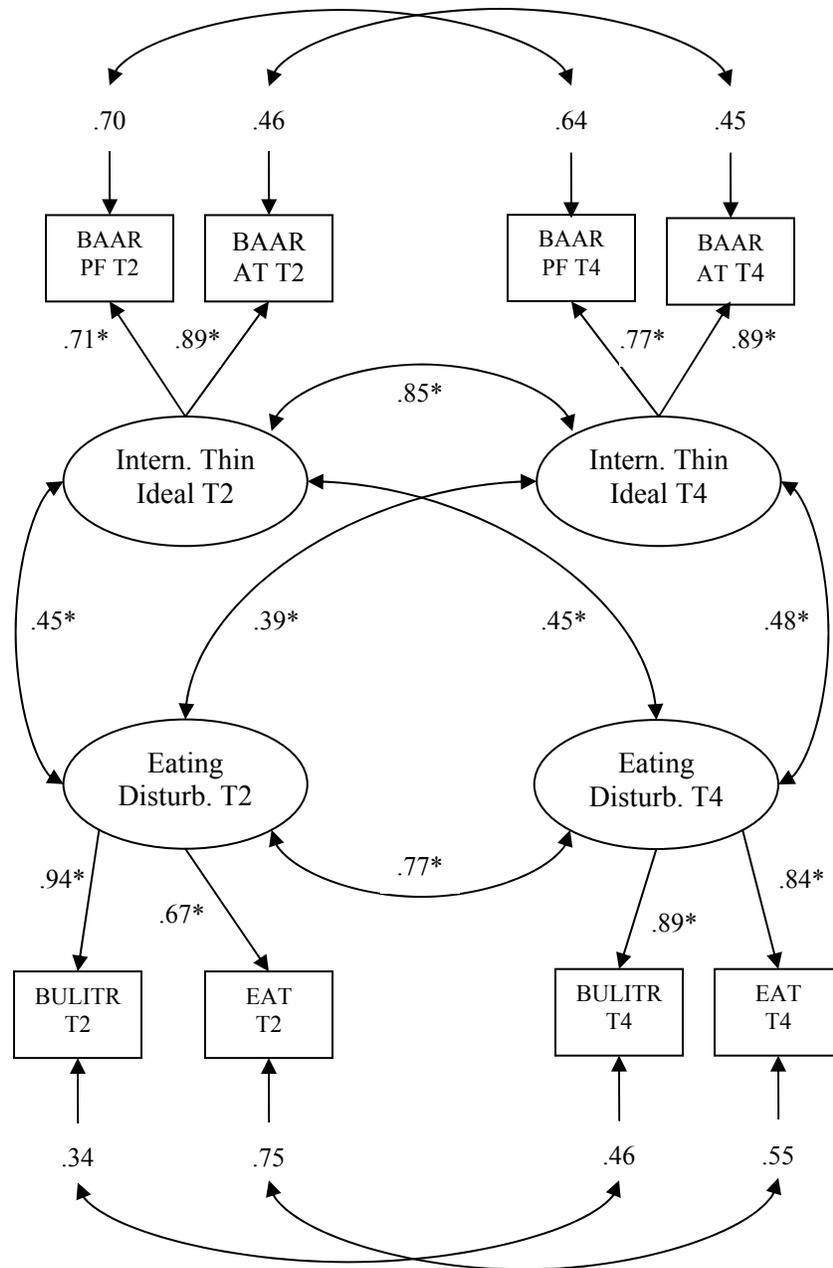
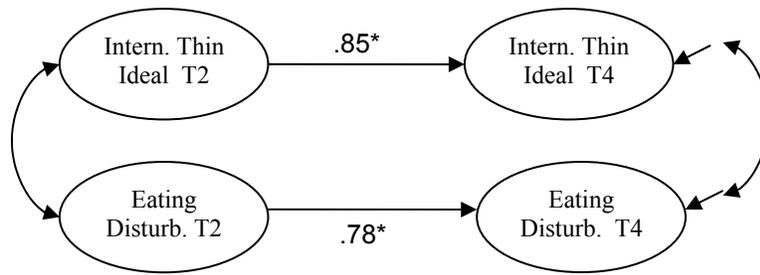
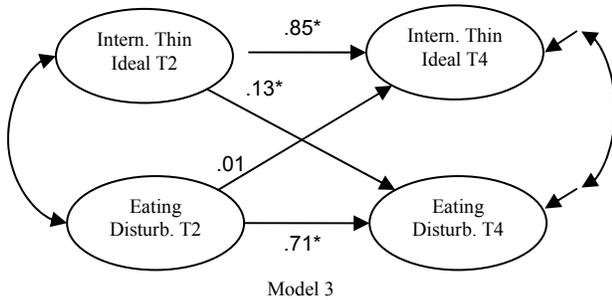
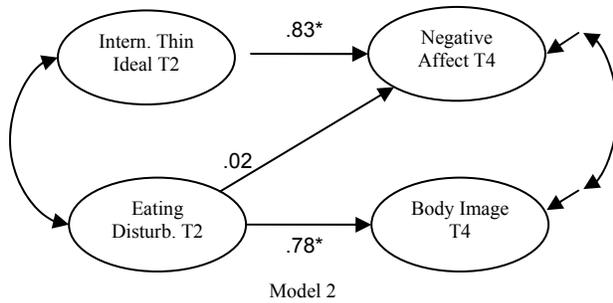
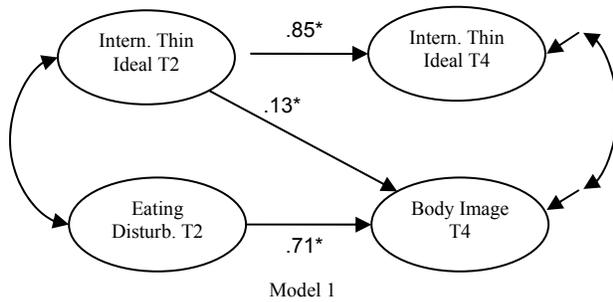


Figure 30. Measurement model for the relations between internalization of thin-ideal and eating disturbance at Time 2 and Time 4. * = $p < .05$.



Cross- Lagged Effects



Simultaneous Effects

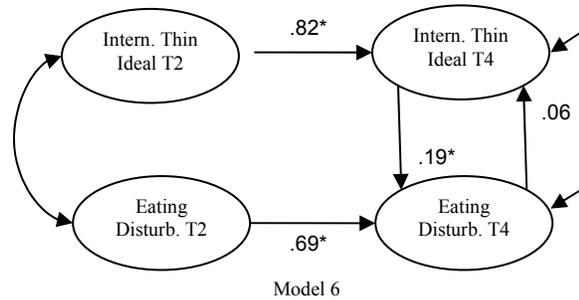
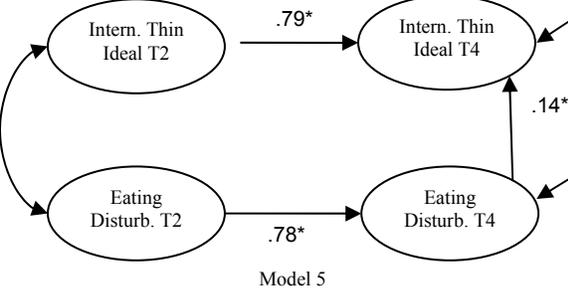
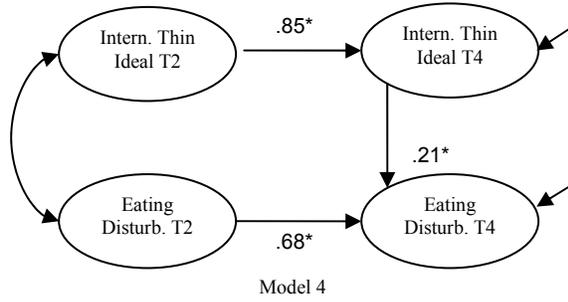


Figure 31. Structural model for the relations between internalization of thin-ideal and eating disturbance at Time 2 and Time 4. $* = p < .05$.

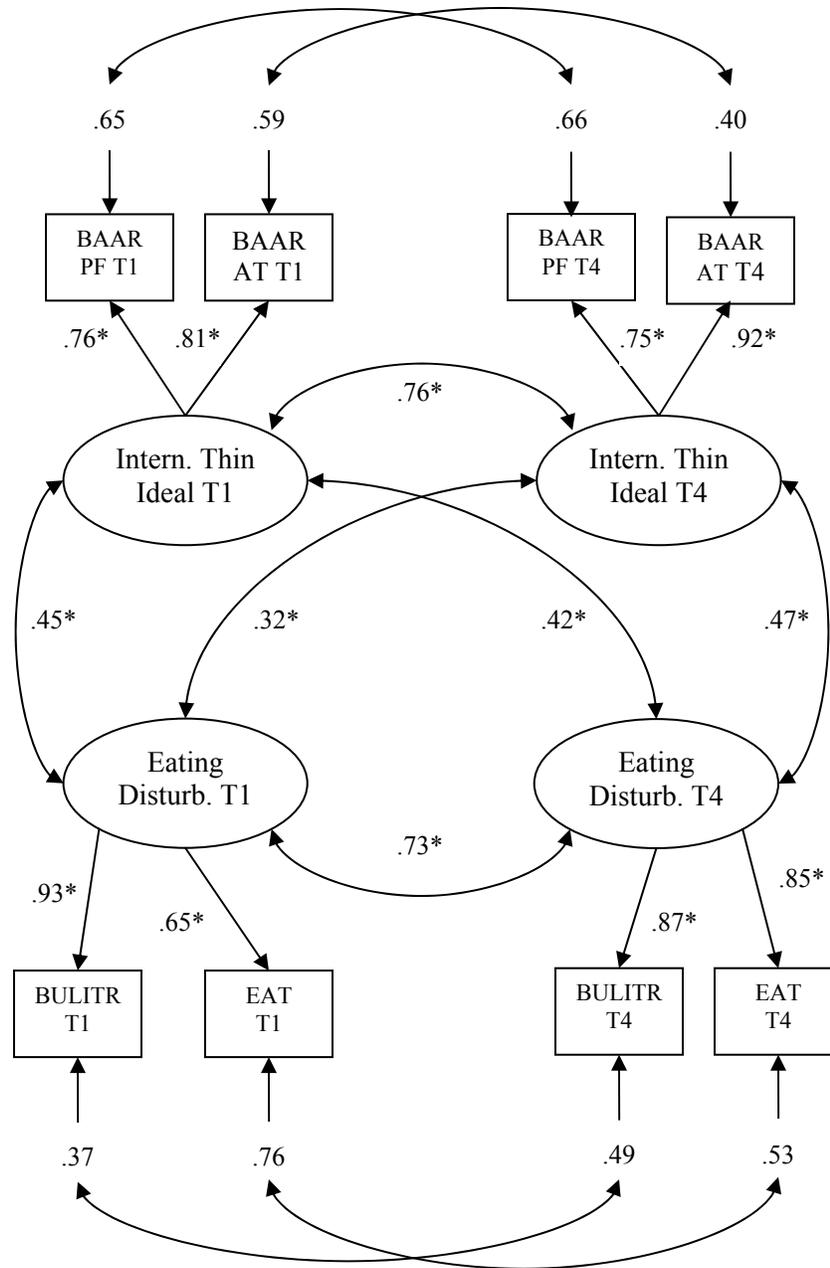
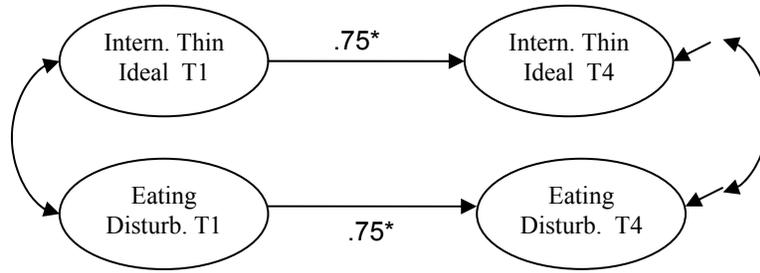


Figure 32. Measurement model for the relations between internalization of thin-ideal and eating disturbance at Time 1 and Time 4. * = $p < .05$.



Cross- Lagged Effects

Simultaneous Effects

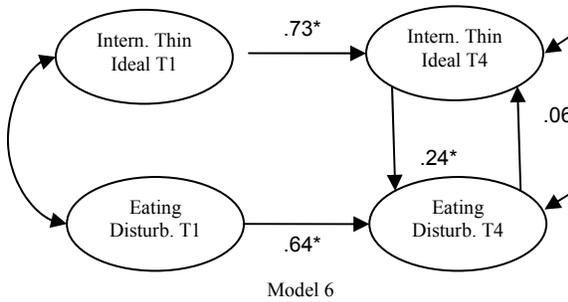
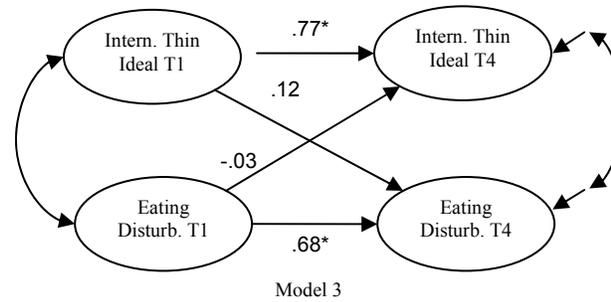
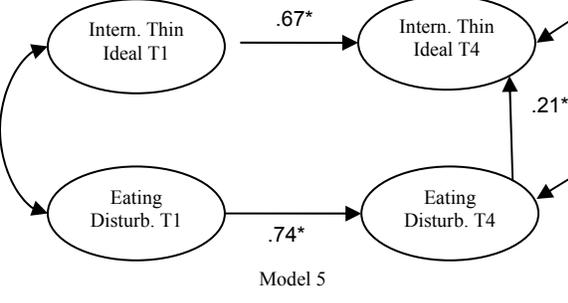
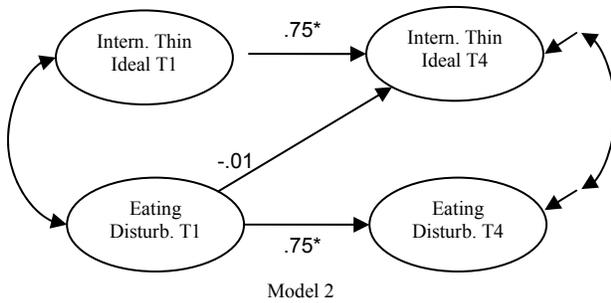
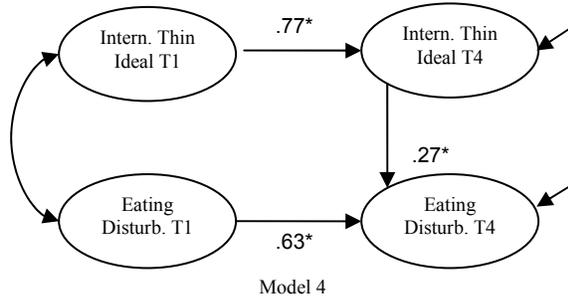
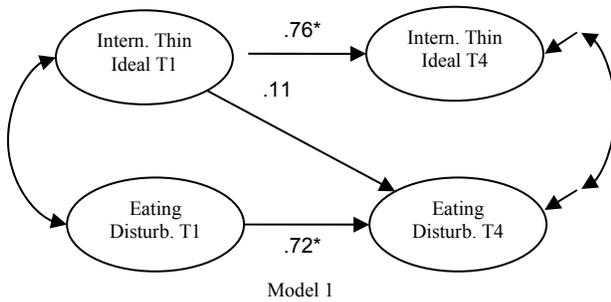


Figure 33. Structural model for the relations between internalization of thin-ideal and eating disturbance at Time 1 and Time 4. * = $p < .05$.

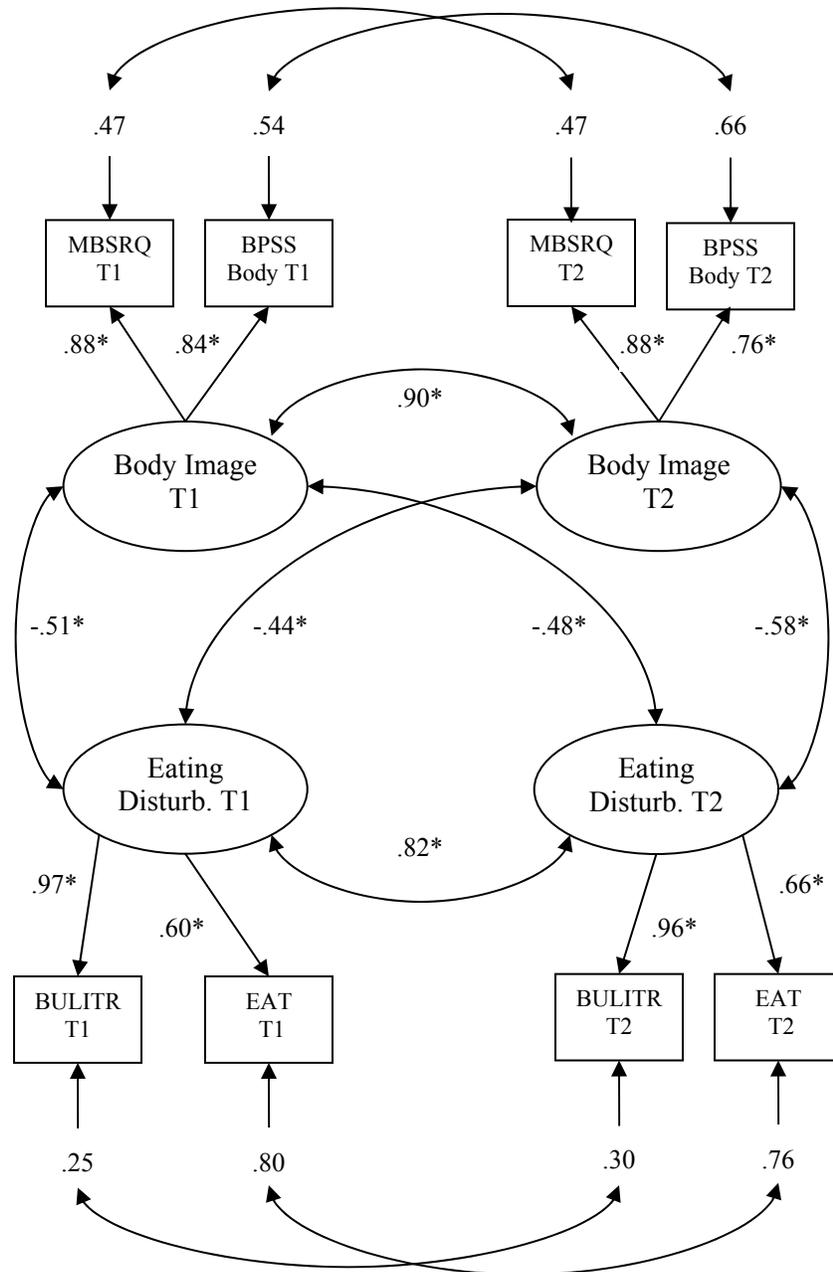
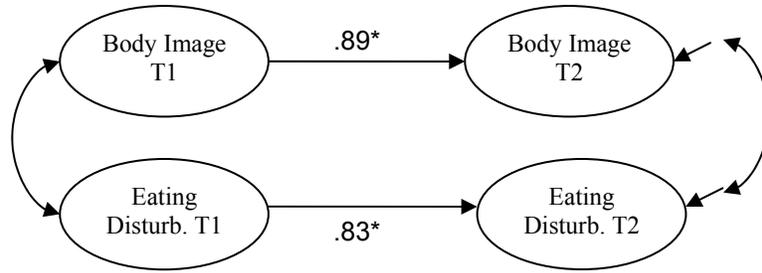


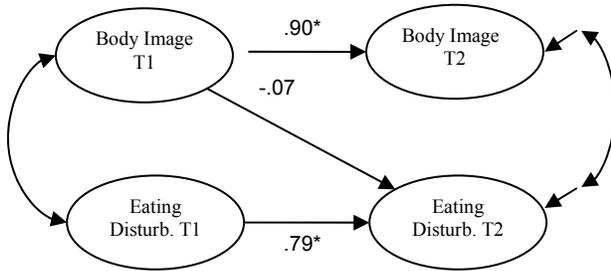
Figure 34. Measurement model for the relations between body image and eating disturbance at Time 1 and Time 2. * = $p < .05$.



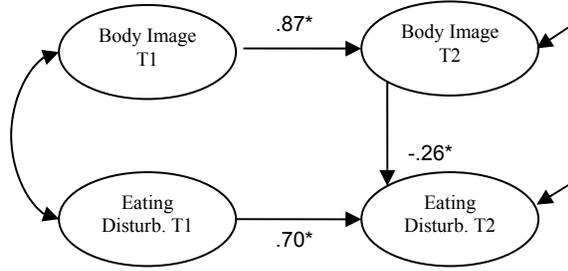
Baseline Model

Cross- Lagged Effects

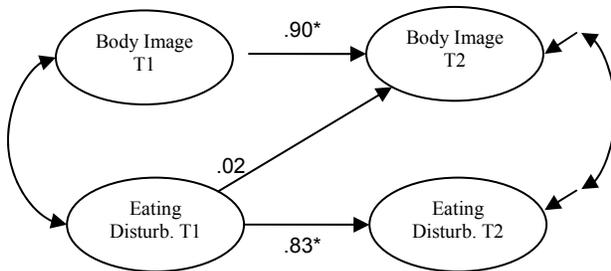
Simultaneous Effects



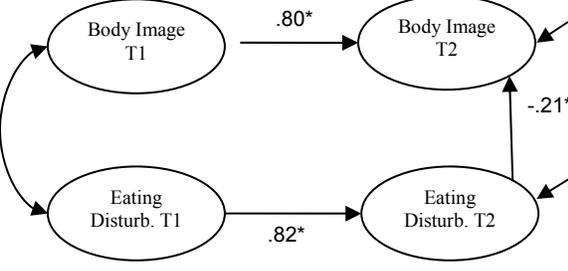
Model 1



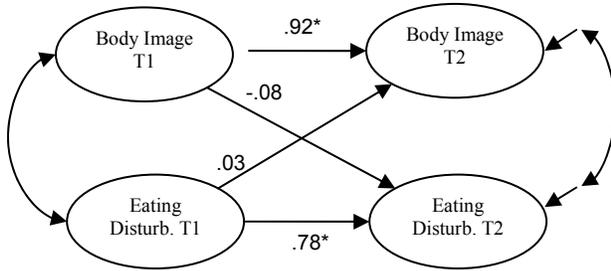
Model 4



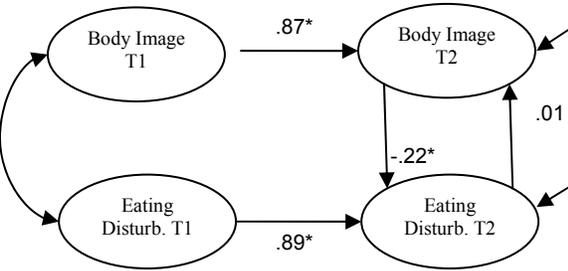
Model 2



Model 5



Model 3



Model 6

Figure 35. Structural model for the relations between body image and eating disturbance at Time 1 and Time 2. * = $p < .05$.

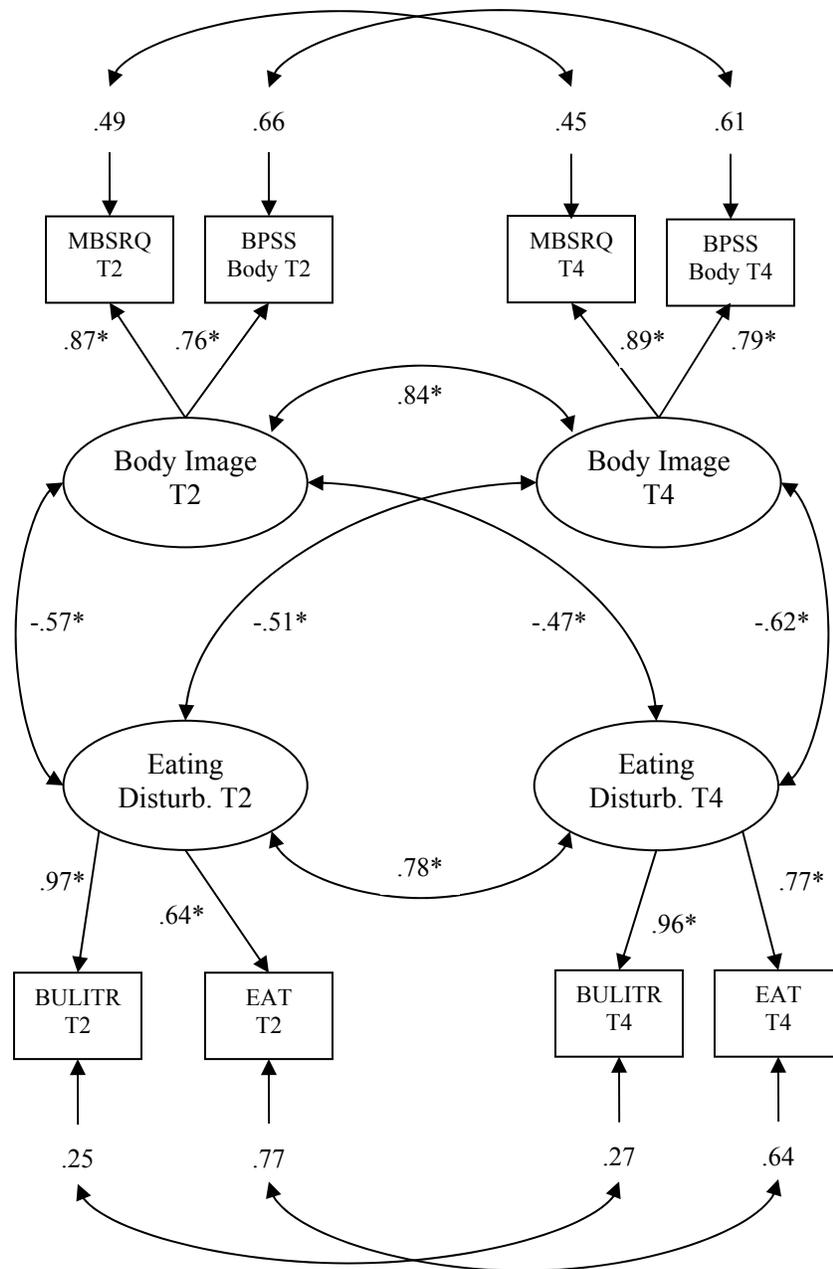
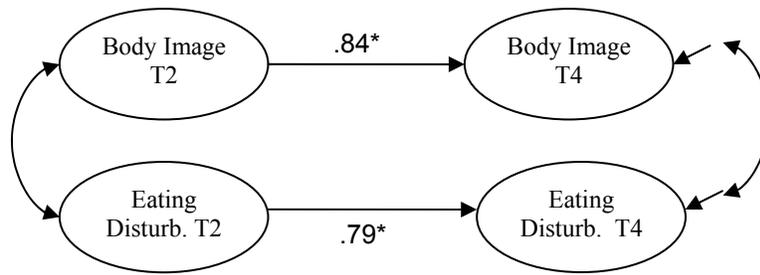


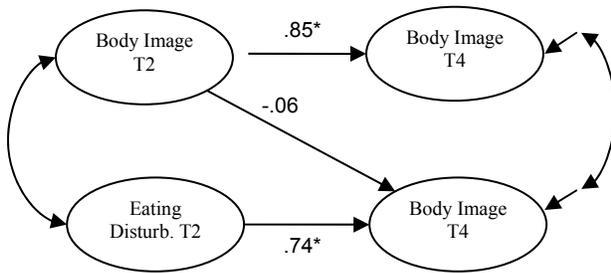
Figure 36. Measurement model for the relations between body image and eating disturbance at Time 2 and Time 4. * = $p < .05$.



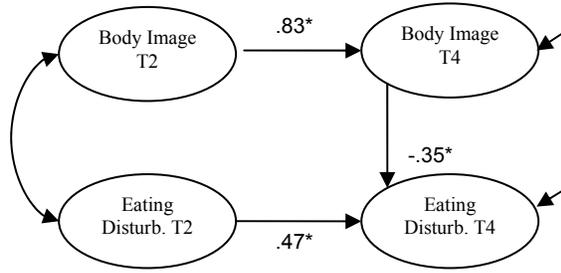
Baseline Model

Cross- Lagged Effects

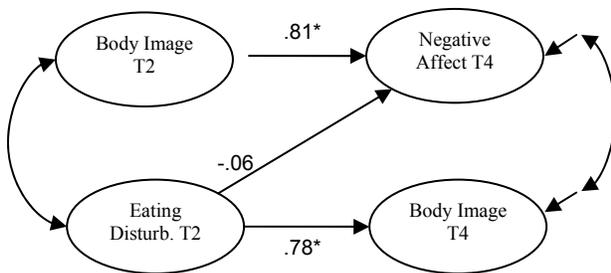
Simultaneous Effects



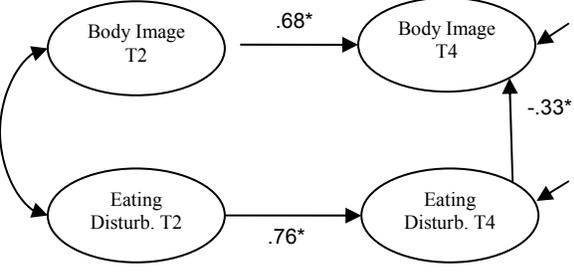
Model 1



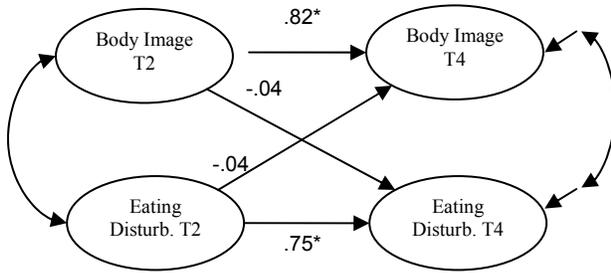
Model 4



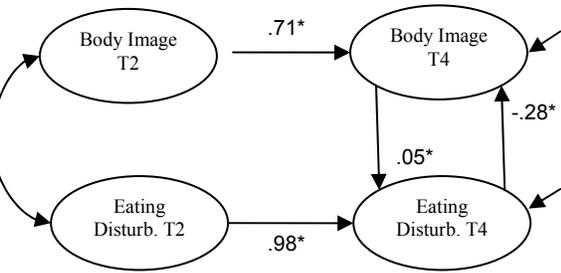
Model 2



Model 5



Model 3



Model 6

Figure 37. Structural model for the relations between body image and eating disturbance at Time 2 and Time 4. * = $p < .05$.

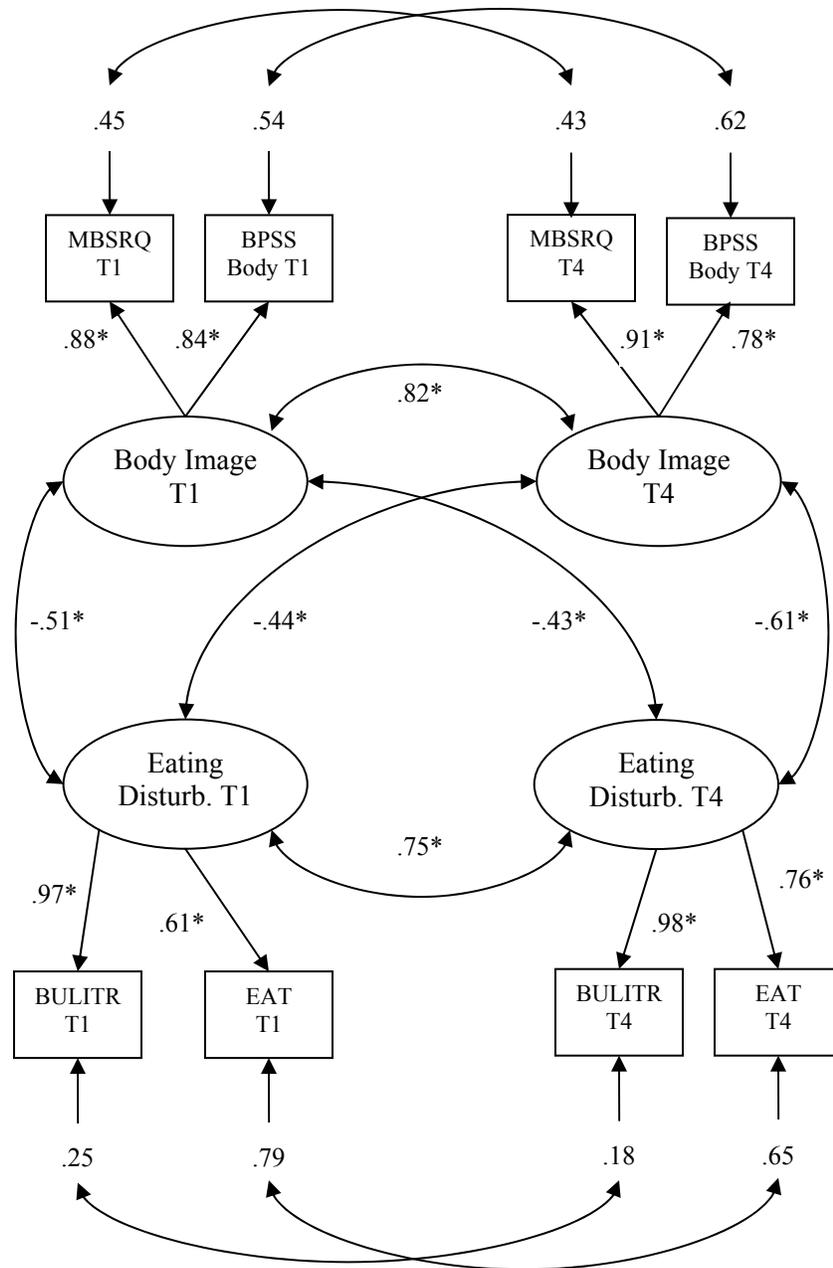
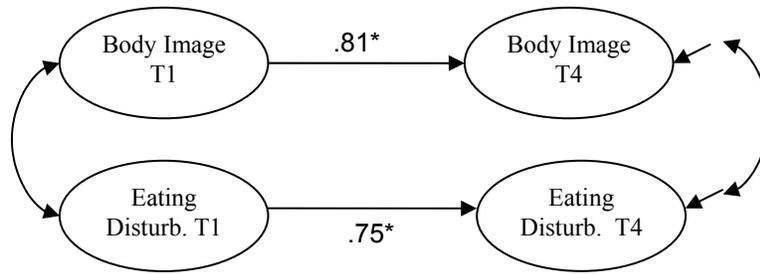
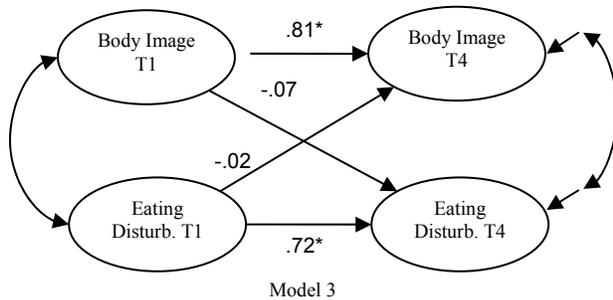
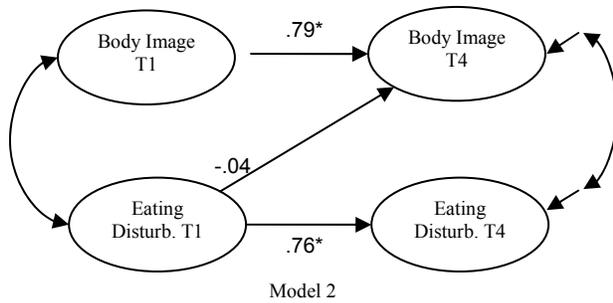
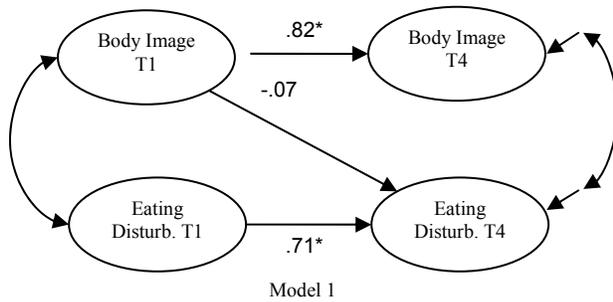


Figure 38. Measurement model for the relations between body image and eating disturbance at Time 1 and Time 4. * = $p < .05$.



Cross- Lagged Effects



Simultaneous Effects

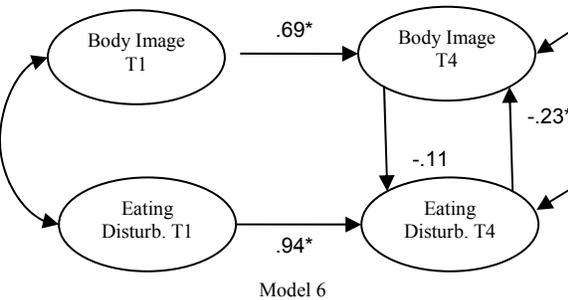
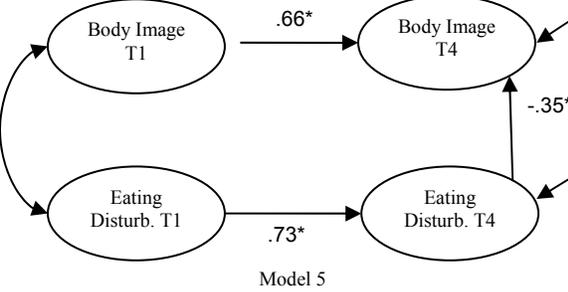
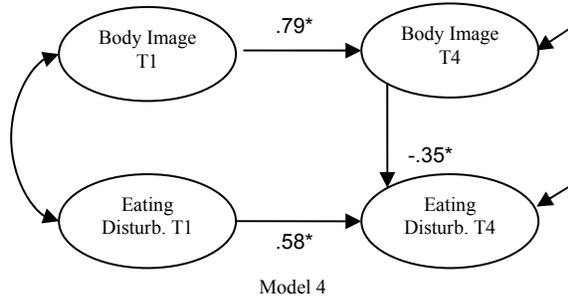


Figure 39. Structural model for the relations between body image and eating disturbance at Time 1 and Time 4. * = $p < .05$.

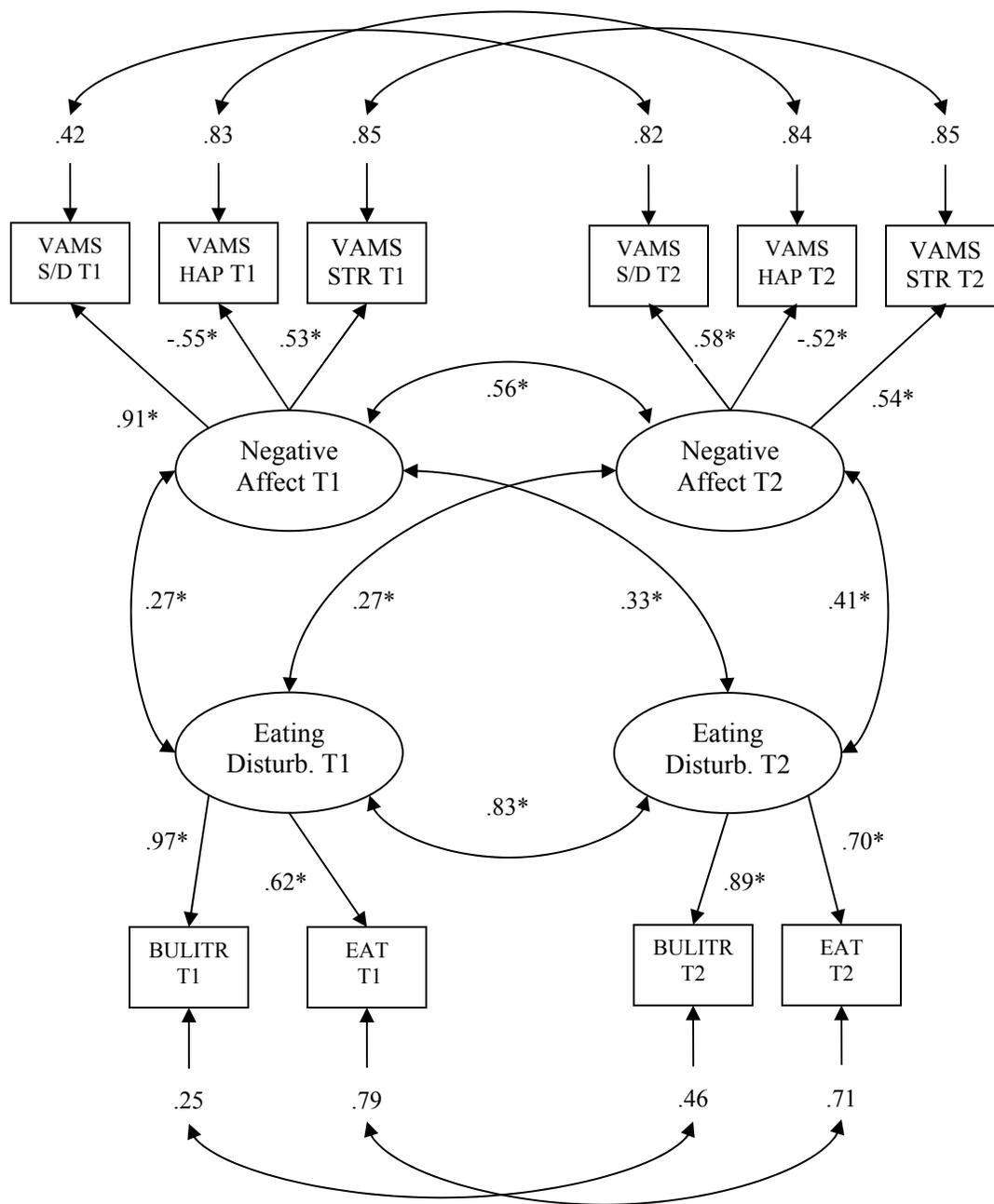
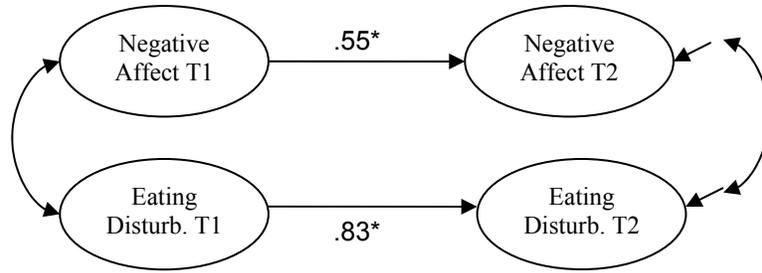


Figure 40. Measurement model for the relations between negative affect and eating disturbance at Time 1 and Time 2. * = $p < .05$.



Cross- Lagged Effects

Simultaneous Effects

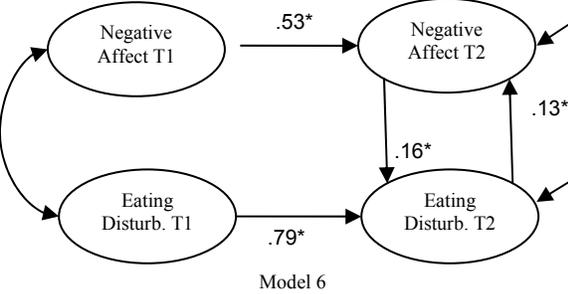
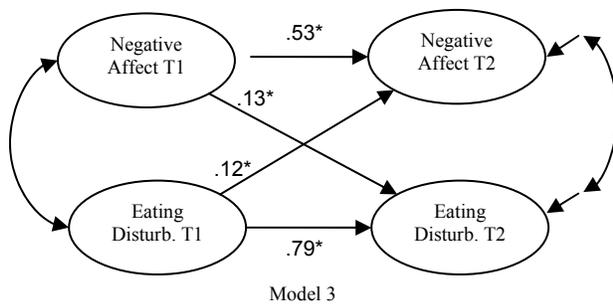
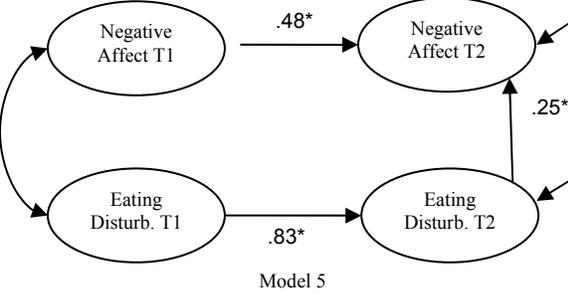
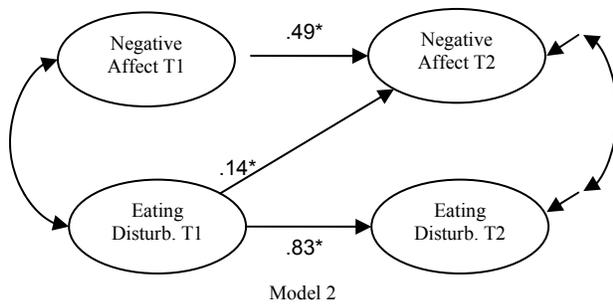
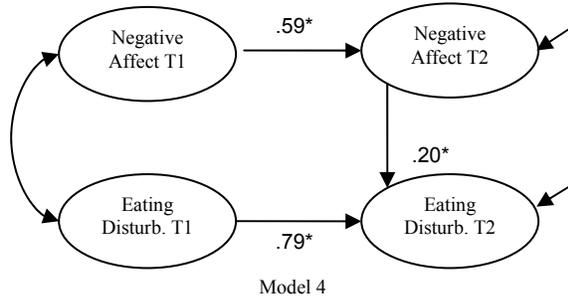
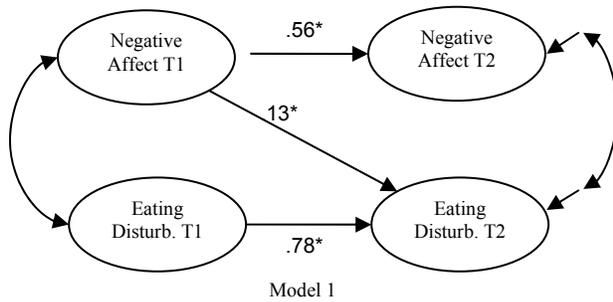


Figure 41. Structural model for the relations between negative affect and eating disturbance at Time 1 and Time 2. * = $p < .05$.

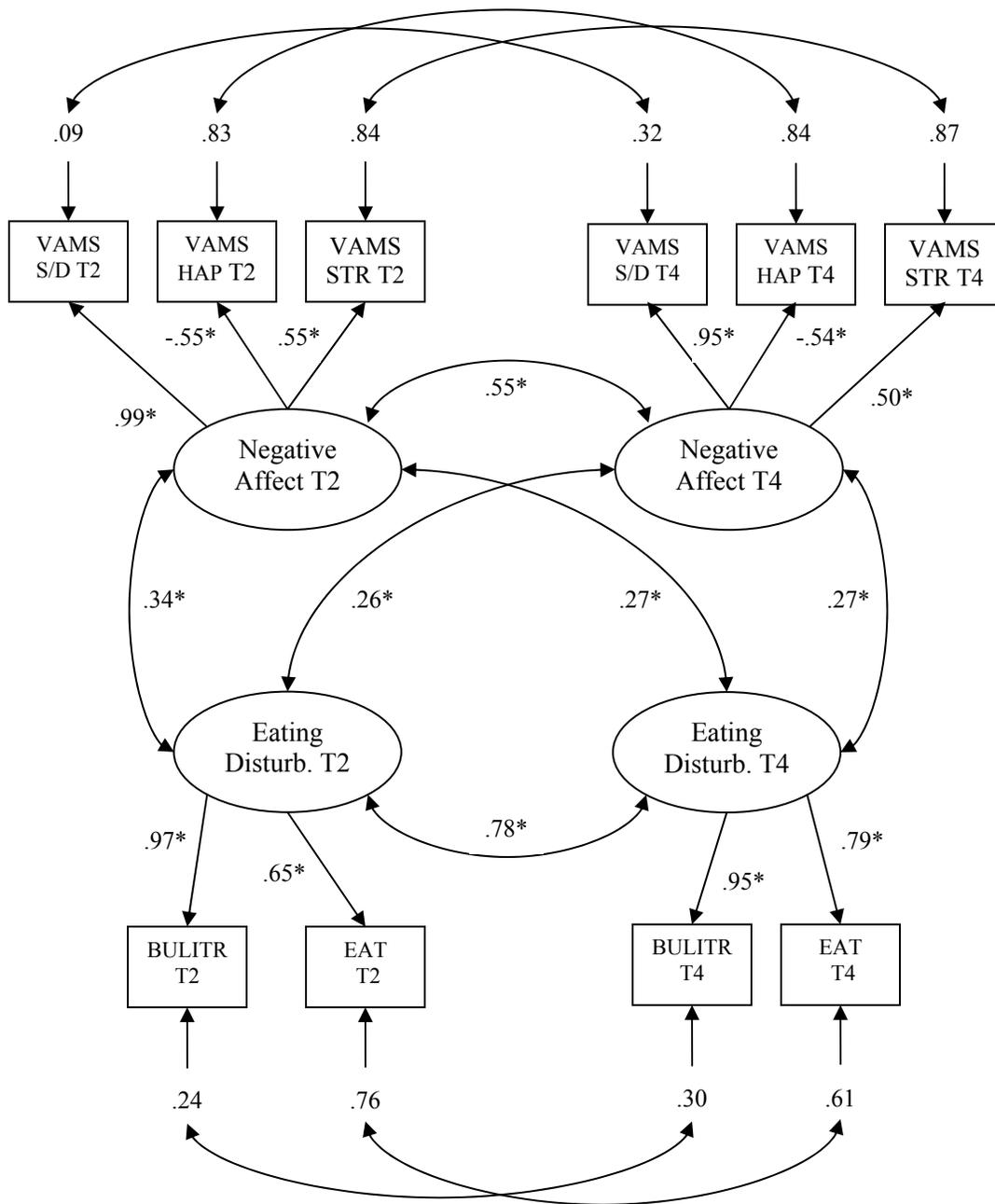
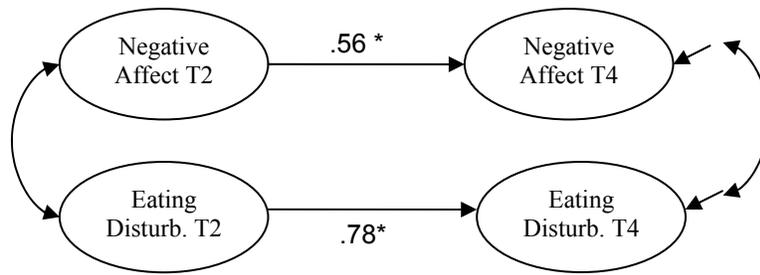


Figure 42. Measurement model for the relations between negative affect and eating disturbance at Time 2 and Time 4. * = $p < .05$.



Cross- Lagged Effects

Simultaneous Effects

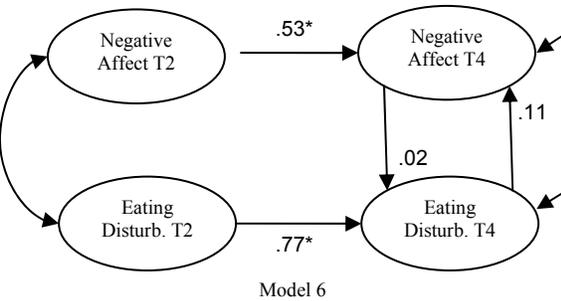
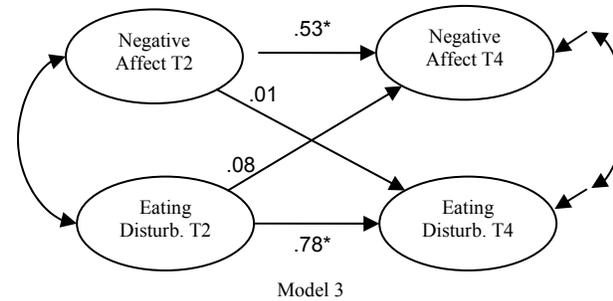
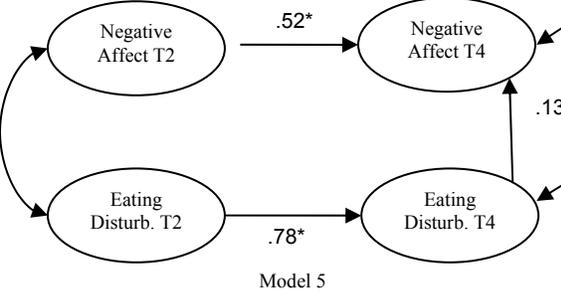
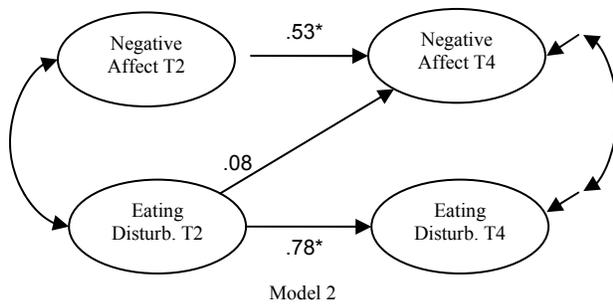
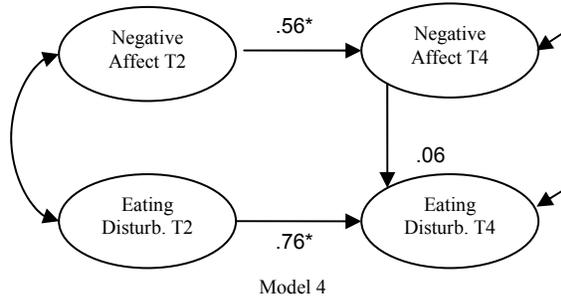
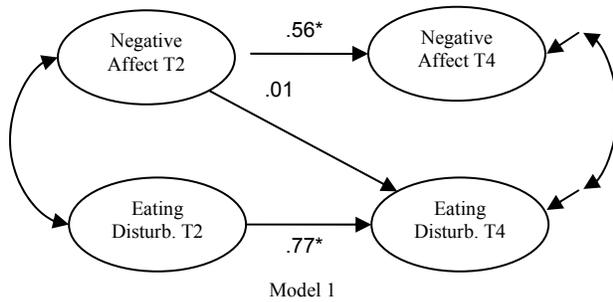


Figure 43. Structural model for the relations between negative affect and eating disturbance at Time 2 and Time 4. * = $p < .05$.

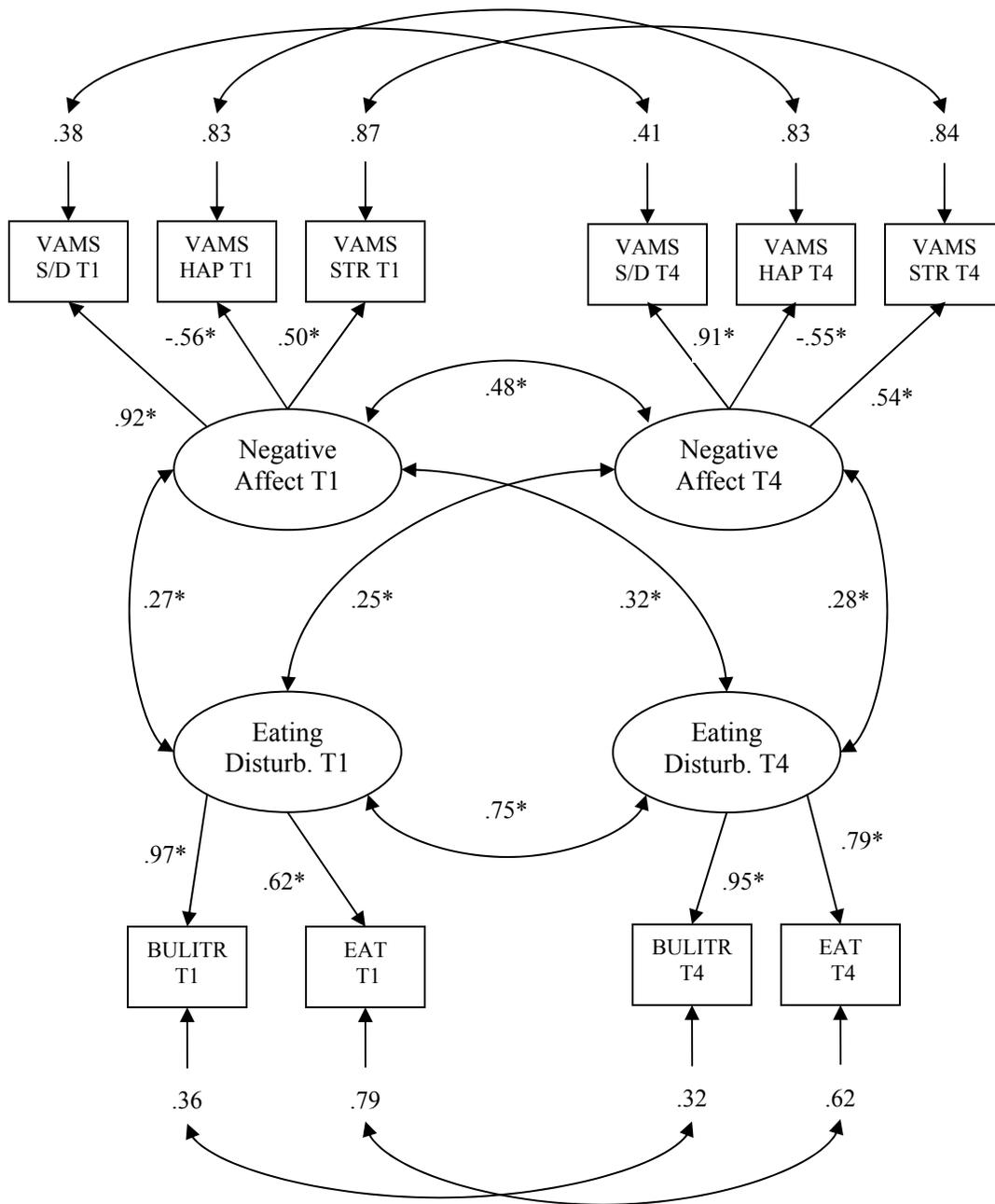
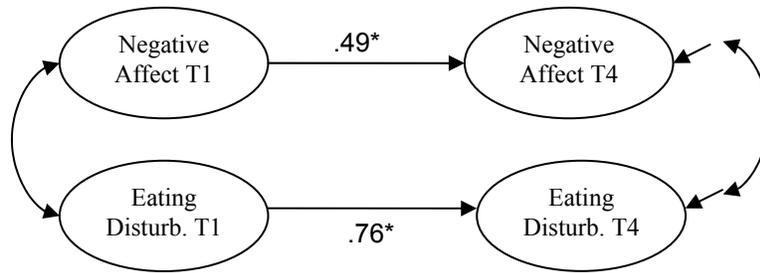


Figure 44. Measurement model for the relations between negative affect and eating disturbance at Time 1 and Time 4. * = $p < .05$.



Cross- Lagged Effects

Simultaneous Effects

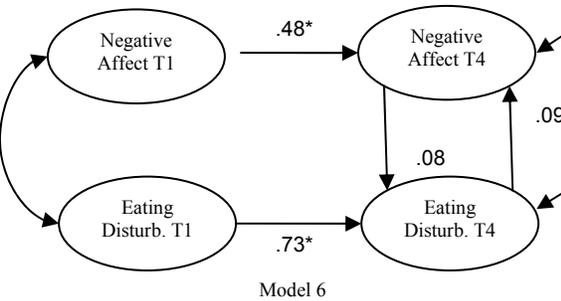
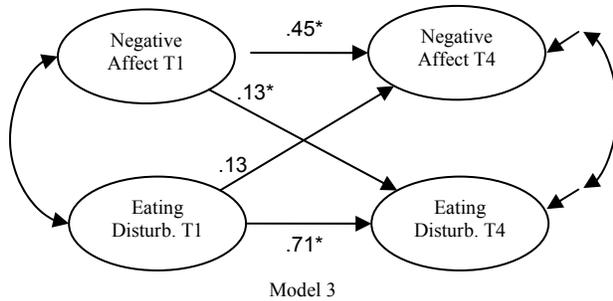
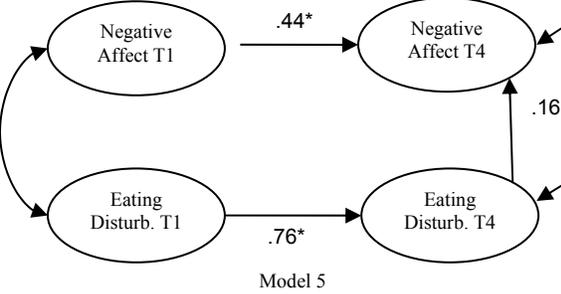
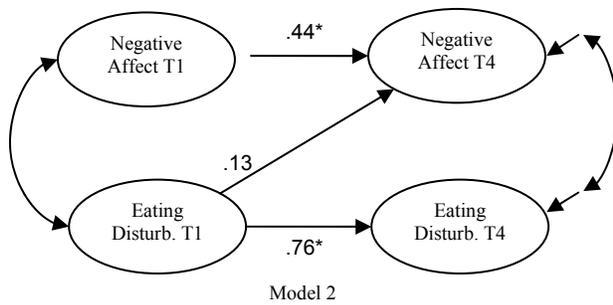
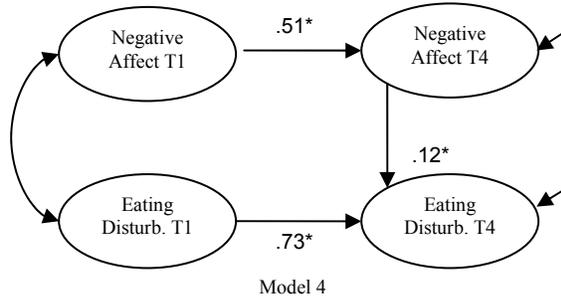
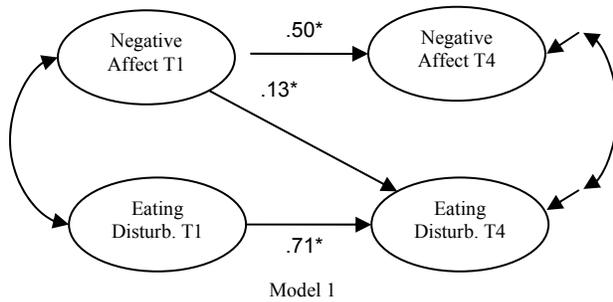


Figure 45. Structural model for the relations between negative affect and eating disturbance at Time 1 and Time 4. * = $p < .05$.

APPENDIX
DEMOGRAPHIC SURVEY

Please answer all of the following questions honestly. Some of the questions may seem repetitive to you, but it is important that you answer every question.

DEMOGRAPHIC QUESTIONNAIRE

1. Sex: _____ 1) female _____ 2) male
2. Age: _____
3. Marital Status: _____ 1) single _____ 2) married _____ 3) separated/divorced
4. Academic Rank: _____ 1) college freshman
_____ 2) college sophomore
_____ 3) college junior
_____ 4) college senior
_____ 5) other
5. Number of Years Attending and Institution of Higher Learning: _____ 1) this is my first year
(e.g., university, community college, etc.) _____ 2) this is my second year
_____ 3) more than 2 years
6. Number of hours of college credit you currently enrolled in: _____ hours
7. High School G.P.A.: _____ on a _____ scale (4.0, 5.0, 6.0, etc.)
8. Race/Ethnicity: _____ 1) Caucasian/White
_____ 2) African-American/Black
_____ 3) Hispanic American
_____ 4) American Indian
_____ 5) Asian American/Pacific Islander
_____ 6) Other: (specify) _____
9. Present Height: _____ feet _____ inches
10. Present Weight: _____ pounds
11. Ideal Weight: _____ pounds
12. Please list all of the people you lived with while growing up:

<u>Name</u>	<u>Age</u>	<u>Relationship to You</u>
_____	_____	_____
_____	_____	_____
_____	_____	_____
_____	_____	_____
_____	_____	_____

13. What was the annual income of the family that you grew up in? _____ 1) under \$20,000
_____ 2) \$20,001 - \$40,000
_____ 3) \$40,001 - \$60,000
_____ 4) \$60,001 - \$80,000
_____ 5) \$80,001 - \$100,000
_____ 6) above \$100,000

14. Current living arrangements: _____ 1) UNT dormitory
_____ 2) apartment - w/ roommate(s)
_____ 3) apartment - alone
_____ 4) at home with parents
_____ 5) other: (specify)_____

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