Bulimic Symptomatology in College Women:
To What Degree Are Hypnotizability, Dissociation, and Absorption of Relevance?

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(ABSTRACT)

Bulimia is often viewed as an extreme expression of eating concerns and body image disturbances that afflicts many adolescent and adult women. The cognitive strategies employed by individuals to inhibit eating and facilitate bingeing and purging are thought to include disattending internal sensations of hunger and satiety while sustaining attention on food, distorted beliefs, and interoceptive experiences (e.g., Heatherton & Baumeister, 1991). To the extent that these attentional and perceptual shifts mediate bulimic symptomatology, individuals with bulimic tendencies should exhibit certain cognitive attributes. Because hypnotizability, dissociation, and absorption have each been invoked (either directly or indirectly) as explanatory constructs for clinical and subclinical bulimia, the present study evaluated the absolute and relative effects of these factors on bulimic symptomatology in a large sample of undergraduate women (N = 309) using structural equation modeling. Following 2 assessments of hypnotic susceptibility (Harvard Group Scale of Hypnotic Susceptibility, Form A [Shor & Orne, 1962] & Group Stanford Hypnotic Susceptibility Scale, Form C [Crawford & Allen, 1982]), participants completed measures of eating disorder symptomatology (Eating Disorders Inventory-2 [Garner, 1991]; Three Factor Eating Questionnaire [Stunkard & Messick, 1985]), dissociation (Dissociative Experiences Scale [Carlson & Putnam, 1986]; Dissociation Questionnaire [Vanderlinden et al., 1993]), and absorption (Tellegen Absorption Scale [Tellegen & Atkinson, 1974]; Differential Attentional Processes Inventory [Crawford, Brown, & Moon, 1993; Grumbles & Crawford, 1981]). A final model including the latent constructs Hypnotizability, Dissociation, Absorption, and Bulimic Symptomatology provided a very good fit to the data ($X^2$ (58, N = 309) = 31.09, NFI = .932, CFI = .967, & RMSEA = .053). As hypothesized, dissociation was found to have a moderate effect (Standardized coefficient = .32, p < .01) on Bulimic Symptomatology when controlling for Hypnotizability and Absorption. Moreover, contrary to past research, the path between Hypnotizability and Bulimic Symptomatology and the path between Absorption and Bulimic Symptomatology were not significant. Based on these findings, we can now speak with increased confidence of a meaningful link between dissociation and the continuum of bulimic symptomatology. A pathological dissociative style appears to contribute to the development of bulimia.
DEDICATION

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Introduction

Bulimia is often viewed as an extreme expression of eating concerns and body image disturbances that afflicts many adolescent and adult women (Lowe, Gleaves, & Murphy-Eberenz, 1998; Stice, Killen, Hayward, & Taylor, 1998b; Thompson, Berg, & Shatford, 1987). Behaviorally, bulimic symptomatology involves frequent episodes of binge eating, with a perceived loss of control, and inappropriate compensatory methods (American Psychiatric Association [APA], 1994). The cognitive strategies employed by individuals to inhibit eating and facilitate bingeing and purging are thought to include disattending internal sensations of hunger and satiety while sustaining attention on food, distorted beliefs, and interoceptive experiences (e.g., Heatherton & Baumeister, 1991; Thompson et al., 1987). To the extent that these directed attentional and perceptual shifts mediate bulimic symptomatology, individuals displaying bulimic tendencies should exhibit certain cognitive attributes. However, while much is known about the psychological dimensions of bulimia as well as the factors which precipitate binge eating (Garner, Olmstead, & Polivy, 1983; Polivy & Herman, 1985; Thompson et al., 1987), little consideration has been given to the cognitive factors that may underlie the expression of bulimia.

Clinical research studies and observations have identified several cognitive/attentional traits that appear to be relevant to bulimic symptomatology. Women identified to exhibit binge eating and other bulimic symptoms (e.g., body dissatisfaction, disinhibited eating) have been found to be higher in hypnotic susceptibility (e.g., Brown & Crawford, 1986; Covino, Jimerson, Wolfe, Franko, & Frankel, 1994; Pettinati, Horne, & Staats, 1985; Vanderlinden, Vandereycken, van Dyck, & Vertommen, 1993) and report more dissociative experiences (e.g., M. Barabasz, 1991; Covino et al., 1994; Everill, Waller, & Macdonald, 1995; Rosen & Petty, 1994; Sanders, 1986; Vanderlinden et al., 1993a) than matched control groups and published norms. Based on these findings, several authors have hypothesized that a high capacity for hypnosis or dissociation may predispose women to bulimia (M. Barabasz, 1991; Butler, Duran, Jasiukaitis,
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Koopman, Spiegel, 1996; Covino et al., 1994; Reto, Dalenberg, & Coe, 1993). Moreover, by implication, hypnotic and dissociative processes have been invoked to explain why the binge-purge cycle is often out of awareness and intractable (Vanderlinden & Vandereycken, 1997).

Bulimic symptomatology is heterogeneous and influenced by a myriad of intrapersonal and psychosocial factors (Smolak, Levine, Striegel-Moore, 1996; Thompson et al., 1987); however, it is not the goal of this investigation to examine all of these influences. Rather, the present study seeks to build on the existing literature by investigating the question of which cognitive/attentional capacities are of relevance to bulimic symptomatology. Because hypnotizability, dissociation, and absorption have each been implicated (either directly or indirectly) in the etiology of clinical and subclinical bulimia, the present study applied structural equation modeling to evaluate the absolute and relative effects of these factors in a large sample of college women. To date, most research on these relationships has been limited to the exploration of univariate relationships and, despite the fact that measures of hypnotizability, dissociation, and absorption are often correlated (Smyser & Baron, 1993; Spiegel, 1990), a unified model relating these constructs to bulimic symptomatology has not been developed and tested. The theoretical basis for this study can best be understood through a review of past studies supporting the hypothesized relationships among these constructs.

1) Hypnotizability and Bulimic Symptomatology

Based on data linking hypnotizability\(^1\) with bulimic symptoms, several investigators have hypothesized that bulimia may represent a type of auto-hypnotic phenomenon (Bliss, 1986). Griffiths and Channon-Little (1993) proposed that bingeing and purging may take place in a hypnotic state. Other researchers (Covino et al., 1993; Groth-Marnat & Schumaker, 1990; Schumaker, Warren, Schreiber, & Jackson, 1994) have hypothesized that bulimic symptoms are

\(^1\) As noted by Piccione, Hilgard, & Zimbardo (1989), hypnotizability and hypnotic susceptibility — can be considered synonyms of a person’s measured talent or ability to produce behaviors and experiences falling within the complex domain of hypnosis (p. 289).
more likely to occur in hypnotizable persons because of greater susceptibility to body distortions and/or sociocultural influences.

Pettinati and colleagues (1985) first reported that a sample of female bulimics (N = 21) scored higher in hypnotic susceptibility (Stanford Hypnotic Susceptibility Scale, Form C [SHSS:C]; Weitzenhoffer & Hilgard, 1962) than anorexics and age-matched controls. Kranhold, Baumann, and Fichter (1990) reported comparable findings with bulimic patients using the Harvard Group Scale of Hypnotic Susceptibility, Form A (HGSHS:A; Shor & Orne, 1962). M. Barabasz (1991) found significantly higher levels of hypnotic susceptibility (SHSS:C) in 42 university women diagnosed with bulimia as compared to nonbulimic college women. Likewise, Covino et al. (1994) reported that bulimic patients were significantly more hypnotizable (SHSS:C) than healthy controls. In a larger sample (N = 113) of female bulimics, Griffiths (1993) observed elevated levels of hypnotic susceptibility (HGSHS:A). Finally, Vanderlinden et al. (1995) found patients (N = 18) with bulimia to be significantly more hypnotizable than normal controls (N = 88) on the Dutch version of the Stanford Hypnotic Clinical Scale (Hilgard & Hilgard, 1983).

Several studies have observed similar relationships between hypnotic susceptibility and bulimic tendencies/symptoms in non-clinical samples. Brown and Crawford (1986) found college women who endorsed abnormal eating concerns and behaviors on the Eating Attitudes Test (EAT; Garner, Olmstead, Bohr, & Garfinkle, 1982) to be higher in hypnotic susceptibility (HGSHS:A). Similarly, Groth-Marnat and Schumacker (1990) reported a significant correlation of .30 between the HGSHS:A and two self-report measures of pathological eating behaviors and attitudes. Frasquilho and Oakley (1997) observed a strong correlation (r = .66, p < .001) between cognitive restraint on the Three Factor Eating Questionnaire (Stunkard & Messick, 1985) and "waking hypnotizability" as indexed by the Creative Imagination Scale (Wilson & Barber, 1978) in British college women (N = 37).

In terms of understanding these relationships, Pettinati et al. (1985) and others (Brown & Crawford, 1986; Covino et al., 1993) have theorized that the same cognitive and attentional skills utilized by women to enter a hypnotic state (e.g., absorptive attention) may be involved in the
acquisition of bulimic psychopathology. In other words, hypnotizability and bulimic symptomatology may be tapping into similar underlying cognitive/attentional abilities (Crawford & Barabasz, 1993; Brown & Crawford, 1986). Hence, to the extent that higher hypnotic susceptibility reflects an increased capacity to become absorbed in the focal experience of overeating, purging, or other dietary rituals, the relationship between measures of hypnotizability and bulimia may result from a shared correlation with absorption.

2) Dissociation and Bulimic Symptomatology

Dissociation is a disruption or separation of mental processes (e.g., thoughts, emotions, memory, and identity) that are normally integrated (APA, 1994; Ludwig, 1983; Spiegel & Cardena, 1993). Dissociation was once conceptualized as a continuous construct ranging from mundane phenomena such as day-dreaming and lapses in attention to pronounced disruptions in perception, memory, and control (e.g., Bernstein & Putnam, 1986). However, investigators have recently favored a typological model which differentiates between pathological and nonpathological dissociative states (Waller et al., 1996; Waller & Ross, 1997). Indeed, nonpathological episodes of absorption and extremely focused attention are considered "normative" (e.g., Kihlstrom et al., 1994; Sandberg & Lynn, 1992), while dissociative phenomena such as psychogenic amnesia and multiple personality states are symptomatic of psychopathology (APA, 1994; Waller et al., 1996).

Researchers and clinicians have increasingly proposed that bulimia is a type of dissociative phenomena (e.g., Conner, 1994; Butler et al., 1996; Reto et al., 1993; Torem, 1986). This hypothesis is consistent with the possible shared origins of dissociative pathology and bulimic symptomatology in trauma or childhood sexual abuse (Everill et al., 1995a; Vanderlinden & Vandereycken, 1997); however, abuse and trauma are neither sufficient nor necessary conditions for the development of pathological dissociation and bulimia (Waller et al., 1996; Welch & Fairburn, 1994). Empirical support for the theory that dissociative mechanisms contribute to bulimic symptomatology comes from studies that have assessed dissociation and bulimic symptoms in both college students and eating disordered patients.

Sanders (1986) found that college women classified as binge eaters reported significantly
more dissociative experiences on the Perceptual Alterations Scale (PAS) than controls. Rosen and Petty (1994) reported a correlation of .40 between the Dissociative Experiences Scale 2 (DES; Bernstein & Putnam, 1986), the most widely used self-report instrument for quantifying dissociation, and two measure of bulimic tendencies in college women. Kisler and Schill (1995) and Frasquilho and Oakley (1997) each reported significant correlations between bulimic symptoms and the DES in university women. In a multiple regression paradigm, Reto, Dalenberg, and Coe (1993) found that "extreme" DES items (i.e., highly skewed items) significantly predicted bulimic symptomatology on the Eating Disorders Inventory (EDI-2; Garner, 1991) in university women. Similarly, McManus (1995) reported a significant correlation (.44) between the EAT Bulimia scale and the DES Depersonalization/Derealization subscale among women with bulimia and anorexia.

Demitrack et al. (1990) found that female bulimics reported significantly more dissociative experiences on the DES than age-matched female controls. Vanderlinden and colleagues (1993) found that a heterogenous sample of eating disorder patients (N = 98) in the Netherlands scored significantly higher than controls on the Dissociation Questionnaire (DIS-Q; Vanderlinden et al., 1993). In another comparative study of bulimic women with female undergraduates, Everill et al. (1995a) reported a significant correlation (r = .17) between the EAT Bulimia scale and the DES Depersonalization/Derealization subscale. This relationship was observed within the college students, but not the patient population. The lack of variability in the patient population (i.e., high EAT scores), however, may have attenuated the findings. Interestingly, Everill and colleagues (1995a, 1995b) have also demonstrated significant correlations (rs = .50 to .64) between bingeing frequency and the fullscale DES among individuals with bulimia and anorexia.

Despite these positive findings, the hypothesized relationship between dissociation and bulimic symptomatology has not been endorsed by all researchers. For instance, Valdiserri and

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2 "DES" refers to the DES fullscale score; the DES also contains three subscales which index both pathological (i.e., Amnesia, Depersonalization/Derealization) and nonpathological (i.e., Absorption) types of dissociation (Ross, Joshi, & Currie, 1991).
Kihlstrom (1995a, 1995b) found significant correlations between the DES and each of the EDI-2 subscales ($r$s = .13 to .36) in two large samples of university women ($N$s > 250). However, because the DES was more strongly associated with EDI-2 subscales indicative of "ego dysfunction" and psychopathology than "abnormal eating," the authors questioned the specificity of the relationship between dissociation and disordered eating. However, there is some question about the generality of these findings since the study involved a non-clinical population, and there were very few participants who approached the pathological range on the EDI-2 subscales.

Overall, the literature is consistent with the hypothesis that dissociation may contribute to the development and/or maintenance bulimic symptomatology. Nevertheless, the nature of the relationship remains unclear because the measures of dissociation utilized (e.g., DES, DIS-Q) are heavily loaded with items representing "normal" dissociative experiences of absorption (see Waller et al., 1996), and few studies have empirically separated pathological and nonpathological dissociative experiences.

3) Absorption/Extremely Focused Attention and Bulimic Symptomatology

Another fruitful endeavor is the study of proposed relationships between bulimic symptoms and absorption or extremely focused attentional skills. Tellegen and Atkinson (1974) described "absorption" as a dimension of personality marked by a propensity to enter states of focused attention "during which the available representational apparatus seems to be entirely dedicated to experiencing and modeling the attentional object" (p. 274). Although little empirical research has examined the relationship between measures of absorption and bulimic symptomatology, it is possible that absorption may mediate binge eating. Indeed, consistent with clinical observations (e.g., Conner, 1994), Heatherton and Baumeister (1991) proposed that binge eating involves a cognitive shift "to narrow the focus of attention to the present and immediate stimulus environment" (p. 89), a process seemingly analogous to absorption. However, research directly testing this hypothesis is presently very limited. Brown and Crawford (1986) reported higher than normal absorptive abilities in women with eating disorder patterns on several measure of focused attention. Likewise, Everill et al. (1995a) found a significant correlation ($r$ = .19) between absorption, as measured by the DES Absorption subscale, and bulimic
symptomatology in female undergraduates. Moreover, because hypnotizability is now believed to represent an array of cognitive/attentional skills (e.g., Crawford et al., 1993; Kihlstrom, 1985; Tellegen & Atkinson, 1974), including the ability to sustain focused attention and resist distractions, it stands to reason that absorption may be a cognitive trait of importance to bulimia.

**Purpose**

The purpose of this study was to examine hypothesized links between hypnotizability, dissociation, and absorption and bulimic symptomatology in a large sample of university women. First, the independent influences of hypnotizability, dissociation, and absorption on bulimic symptomatology were tested by comparing three a priori models via structural equation modeling (SEM). Subsequently, the three models were combined to examine the simultaneous and relative effects of these cognitive/attentional constructs (i.e., hypnotizability, dissociation, and absorption) on bulimic symptomatology. There are several advantages to this approach over past univariate research. Namely, latent variable SEM allows one to utilize multiple measures for each construct, to empirically compare the fit of each model to the data, and to separately model the error variance, thus controlling for measurement error in examining hypothesized structural relations (Crowley & Fan, 1997; Hatcher, 1995).

Because eating disorders are highly prevalent in college women (Hart & Ollendick, 1984; Katzman, Wolchik, & Braver, 1984; Russ, 1998) and as many as 80% binge on a regular basis (Hawkins & Clement, 1980; Katzman et al., 1984), college women represent a group at high-risk for developing bulimia. In fact, the modal age of onset for bulimia is 18-19 years of age (Stice et al., 1998a). Furthermore, among those diagnosed with bulimia nervosa, there is a wide range of symptomatic severity (Thompson et al., 1987). Therefore, since bulimic symptomatology exists on a continuum (Lowe et al., 1998; Russ, 1998; Stice et al., 1998b), relationships between hypnotizability, dissociation, or absorption and bulimic symptomatology should be apparent in college women if these constructs are associated with bulimia.

In the present study hypnotizability, absorption, and dissociation are conceptualized as separate, albeit correlated, constructs. Unlike most prior research, the present study empirically addressed the overlap among these constructs often found in the literature (Frankel, 1990;
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Kihlstrom, Glisky, & Angiulo, 1994) and, in particular, the distinction between different types of dissociation (i.e., pathological vs. nonpathological; Waller et al., 1996). Hence, dissociation was operationalized to include only symptoms of extreme discontinuity in awareness, memory, perception, or identity (e.g., amnesia, depersonalization/derealization, multiple personality states). Conversely, nonpathological dissociative experiences were incorporated into the construct of absorption. Finally, bulimic symptomatology was defined by the major putative dimensions of bulimia described in the literature. In addition to bingeing and purging, research indicates that dietary restraint and body/weight dissatisfaction represent the other fundamental components of bulimia (Laessle, Tuschl, Waadt, & Pirke, 1989; Thompson et al., 1987).

Hypotheses

1. In the three preliminary models, significant paths will be found between the latent construct Bulimic Symptomatology and the latent constructs Hypnotizability, Dissociation, and Absorption.

2. In the final model, when controlling for covariation among the latent constructs:
   a) a significant path will be found between Dissociation and Bulimic Symptomatology;
   b) a significant path will not be found between Hypnotizability and Bulimic Symptomatology; rather, the relationship between Hypnotizability and Bulimic Symptomatology found in the literature is due to a shared correlation with absorption;
   c) a significant path will be found between Absorption and Bulimic Symptomatology.

Method

Participants

Participants were 342 undergraduate women, aged 18 and older, taking psychology courses at a large mid-Atlantic university. Participants received 5 hours of extra credit for participating in the study. Men (N = 160) also participated in the study, but this data will not be reported in this document. Among the present sample, the mean age was 19.27 (SD = 2.19; Range = 18 - 39). With regard to eating disorder symptomatology, 17.5% of the sample (N = 54) scored 14 or greater (transformed responses; see Garner, 1991) on the EDI-2 Drive for Thinness Subscale. Hence, a substantial number of the participants endorsed items suggestive of
"subclinical" eating syndromes or "high risk" for an eating disorder (Garner, 1991).

Procedure

Participants were administered two standardized measures of hypnotizability within a 2 hour period. After the second hypnosis session, participants were asked to participate in a further study described as "a study of individual differences in attentional processing, personality styles, eating styles, and hypnotic responsiveness" (see Appendix A). This research represents one part of a larger project on the correlates of hypnotizability currently underway in the Neurocognition Laboratory of Professor Helen J. Crawford. The larger study includes several additional measures which are not part of this dissertation.3

Interested participants received a packet of questionnaires to be completed at home. The entire packet of questionnaires required approximately 2 to 3 hours to complete. Participants were asked to complete the measures in a quiet place while not under the influence of alcohol or drugs, and to return the packets within 3-5 days. Participants who did not return packets within 2 weeks were contacted by phone to encourage return of the questionnaires; however, participants were given ample opportunity to decline participation in the study and assured that they may withdraw at any time without penalty. Overall, 90% of the distributed questionnaire packets were returned.

Hypnotic susceptibility assessment. Participants were administered the Harvard Group Scale of Hypnotic Susceptibility, Form A (HGSHS:A; Shor & Orne, 1962) and the Group Stanford Hypnotic Susceptibility Scale, Form C (GSHSS:C; Crawford & Allen, 1982) in small groups (less than 20 students) in a university classroom by the investigator, Dr. Helen J. Crawford, or a trained graduate student. Both scales included a hypnotic "induction" in which participants were asked to gradually relax and close their eyes. Subsequently, participants were given a series of tasks including imagining motor movements (e.g., heavy arm) and cognitive events (e.g., thinking of something not present). At the end of each scale, participants were

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3 The Vividness of Visual Imagery Questionnaire (Marks, 1973), Personality Syndrome Questionnaire (Gruzelier, 1995), and Behavioral Inhibition/Behavioral Activation Scales (Carver & White, 1994) were also administered.
asked to open their eyes, become alert, and answer questions about their experience.

Measures and Model Indicators

The self-report instruments included in this study are described below. The measures were used to identify and derive indicators for the SEM analyses. Cronbach's alphas for the indicators used in this study are presented in Table 1.4

1) Hypnotic Susceptibility As noted above, participants received the HGSHS:A and the GSHSS:C. The GSHSS:C contains the identical hypnotic suggestions (i.e., items) as the Stanford Hypnotic Susceptibility Scale, Form C (Weitzenhoffer & Hilgard, 1962), but is revised for group administration (Crawford & Allen, 1982). Both standardized measures are commonly used in hypnosis research to screen for hypnotizability; however, the GSHSS:C contains more cognitive items (which tend to be more difficult) than the HGSHS:A. Based upon the number of items "passed," each participant received a hypnotic susceptibility score from 0 to 12, with higher scores indicative of greater hypnotic responsiveness (for a review, see Hilgard, 1965). The coefficients of reliability for both scales have been found to be satisfactory (Hilgard, 1965). For instance, Hilgard (1965) reported a Kuder-Richardson estimate of .85 for the SHSS:C. Likewise, McConkey, Barnier, Maccallum, and Bishop (1996) reported internal reliability coefficients over .90 for the HGSHS:A. Furthermore, Piccione et al. (1989) found a test-retest reliability of .71 over 25 years for the Stanford Hypnotic Susceptibility Scale, Form A (Weitzenhoffer & Hilgard, 1959), an individually administered scale on which the HGSHS:A is based.

Factor analytic research has suggested that these hypnotizability scales are not unidimensional (for a review, see Balthazar & Woody, 1985). Using principal components analysis, Hilgard (1965) derived a three factor solution to the Stanford Hypnotic Susceptibility Scale, Form C (SHSS:C) which has been replicated by other researchers (e.g., Tellegen & Atkinson, 1976). Namely, Hilgard's (1965) factors, which accounted for 66% of the total

4 The individual items for the HGSHS:A were not available; hence, the Cronbach's alpha could not be calculated
variance, included 1) Ideomotor Inhibition (IDEO); 2) Difficulty (DIFF); and 3) Positive Hallucination (HALL). Other factor solutions have been found (Balthazard & Woody, 1985); however, because several of the investigators cited above (i.e., M. Barabasz, 1991; Pettinati et al., 1989) have referred to Hilgard’s (1965) factors in their research, these were selected as indicators for the construct Hypnotizability. The full scale score on Harvard Group Scale of Hypnotic Susceptibility, Form A (HGSHS:A) was also used as a model indicator.

2) Dissociative Experiences Scale II (DES-II; Carlson & Putnam, 1992). The DES-II is a 28 item self-report questionnaire originally developed to measure a continuum of dissociative phenomena "from minor dissociations of everyday life to major forms of psychopathology" (Bernstein & Putnam, 1986, p. 728). The DES-II is the most widely studied, reliable, and validated measure of dissociation in the literature (van Ijzendoorn & Schuengel, 1996). Participants are asked to circle the percent of time (in 10% increments from 0% to 100%) they engage in each experience. The DES-II is not a diagnostic instrument; rather, a quantitative score, representing the total time an individual experiences various minor and major dissociative experiences, is derived by averaging the 28 items.

Bernstein and Putnam (1986) reported Spearman-Brown split-half (internal) reliability coefficients of .71 to .96 for eight normal and clinical groups and a test-retest reliability coefficient (Spearman rank-order) of .84 over an interval of 4 to 8 weeks. Frischholz et al. (1990) found a temporal stability coefficient of .93 over 1 month. Dubester and Braun (1995) reported a 2 week test-retest reliability of .93 with dissociative disorder patients. Cronbach's alphas over .90 have been consistently reported in both college student and clinical populations (e.g., Dubester & Braun, 1995; Ensink and Otterloo, 1989; Frischholz et al., 1990). Finally, a recent meta-analysis concluded that the DES-II shows excellent convergent validity with other dissociation questionnaires and interview schedules (van Ijzendoorn & Schuengel, 1996). Subscales representing pathological dissociative experiences of depersonalization and amnesia (DES-DEP & DIS-AMN) and absorption (DES-ABS) were used as model indicators (see Results section for derivation of indicators).

3) Dissociation Questionnaire (DIS-Q; Vanderlinden et al., 1993). The DIS-Q is a 63 item
self-report questionnaire developed in the Netherlands for the measurement of dissociative experiences. Participants are asked to indicate the extent to which each statement is personally relevant on a Likert-type scale ranging from 0 ("not at all") to 4 ("extremely"). Coefficient alpha for the total scale is over .90 (Vanderlinden et al., 1995; Vanderlinden et al., 1993b). The DIS-Q is stable with a test-retest reliability of .94 (subscales ranged from .75 to .93) over a 3 to 4 week period. In studies of normals (N = 750) and psychiatric patients, the DIS-Q demonstrates good construct validity and discriminant validity (Vanderlinden et al., 1995). For instance, Vanderlinden et al. (1995) reported a correlation of .85 (Pearson) with the DES. Subscales representing pathological dissociative experiences of amnesia and identity confusion (DISQ-AMN & DISQ-ID) and absorption (DISQ-ABS) were used as model indicators (see Results section for derivation of indicators).

4) Tellegen Absorption Scale (TAS; Tellegen & Atkinson, 1974). The TAS consists of 34 true-or-false items which describe experiences of self absorption and involvement in various internal (e.g., fantasies) and external (e.g., movies) events. Tellegen (1982) reported an internal reliability coefficient alpha of .88 and a test-retest reliability of .91. Kihlstrom et al. (1989) reported a test-retest reliability of .85. Furthermore, Frischholz et al. (1991) reported high levels of internal consistency, suggesting that "the TAS appears to be measuring one form of normal dissociative-like experiences" (p. 186). The full scale score (TAS) was used as a model indicator.

5) Differential Attentional Process Questionnaire (DAPI; Crawford, in preparation; Crawford et al., 1993; Grumbles & Crawford, 1981). The DAPI consists of 40 self-report statements related to experiences of focused attention and ignoring distractions, as well as simultaneous tasks performance (Grumbles & Crawford, 1981; Lyons & Crawford, 1997). Participants are asked to rate the degree to which they ordinarily carry out activities on a 7-point Likert-type scale ranging from 0 ("Not at all") to 6 ("Always"). Factor analytic studies have identified four reproducible sub-scales in college students (Crawford et al, 1993; Grumbles & Crawford, 1981; Yanchar, 1984). The Moderately focused attention scale has eight items (e.g., concentrate easily on reading or studying while in a noisy room) that assess the perceived ability
to sustain moderately focused attention. The Extremely focused attention scale contains 12 items (e.g., Lose yourself in thought so that you are hardly aware of the passage of time) that assess the perceived tendency to fully engage one's attentional resources in the task at hand while disattending other stimuli. The dual attention cognitive-cognitive scale comprises four items (e.g., read or study easily while at the same time easily listen to radio or TV), and the dual attention cognitive-physical scale includes five items (e.g., carrying out a motor activity easily while listening to a conversation). According to Lyons and Crawford (1997), the test-retest reliability for the DAPI over 4 weeks was .90 and Cronbach’s alpha was above .90. The DAPI extremely focused attention scale (DAPI-EXT) was used as a model indicator.

6) Eating Disorders Inventory-2 (EDI-2; Garner, 1991; Garner et al., 1983). The EDI-2 is a widely used self-report instrument designed to measure continuous cognitive and behavioral traits associated with bulimia and anorexia nervosa. The measure contains 64 items that were rated on a Likert-type scale from 0 (“never”) to 5 (“always”). In clinical samples, the EDI-2 contains eight empirically derived subscales (Garner et al., 1983; Welch, Hall, & Norring, 1990). Three subscales, Drive for Thinness (DT), Bulimia (B), and Body Dissatisfaction (BD), contain items that directly assess attitudes and behaviors concerned with weight, body image, and pathological eating (i.e., bingeing and purging). Numerous studies have documented the strong psychometric properties of the EDI-2 (e.g., Garner et al., 1983; Klemchuck et al., 1990; Norring, 1990; Welch et al., 1990; for a review, see Garner, 1991).

The EDI-2 was developed to differentiate between criterion (i.e., eating disorder) and nonclinical samples. For instance, Garner (1991) reported that discriminant function analyses were able to correctly classify up to 96% of subjects who were either normals or suspected bulimics on the basis of the subscales. Hence, despite its original purpose for use in clinical populations of eating disorders, the EDI-2 has been used successfully with college students and other nonclinical populations. A cutoff score of 14 on the EDI-2 Drive for Thinness subscale has been used to identify college women likely to possess a clinically diagnosable eating disorder (Garner, 1991).

The following subscales were used as model indicators: Bulimia (BULIM), Body
Dissatisfaction (BODY), and Drive for Thinness (DRIVE). Untransformed scores were used to provide the most variance on each subscale.

7) Three Factor Eating Questionnaire (TFEQ; Stunkard & Messick, 1985). The TFEQ is a 51 item self-report instrument developed to assess the constructs underlying dietary restraint. In part 1, the TFEQ has a true-false format and items were scored as either 0 or 1. In part 2, items are rated on a Likert-type scale ranging from 0 to 5. The three empirically derived factors of the TFEQ are 1) Cognitive Restraint of eating (i.e., intentional control over eating behavior); 2) Disinhibition (i.e., tendency to lose control over eating); and 3) Hunger (i.e., susceptibility to hunger) (Williams et al., 1996). Results from several studies demonstrate the reliability and validity of the instrument (e.g., Stunkard & Messick, 1985). For instance, Stunkard and Messick (1985) reported coefficient alphas of .92, .91, and .85, respectively, for the three scales. The Disinhibition subscale (DISIN) was used as a model indicator.

Results

Missing Data

Three-hundred and forty-two (N = 342) questionnaires packets were returned to the experimenter. On scales that were 90% or more complete, missing values were replaced by the mean score of the remaining items on the scale; those that were less than 90% complete were considered missing and deleted from the sample. In total, 17 questionnaires were eliminated from further analyses due to incompleteness or highly questionable validity (e.g., responses that were out of range for a particular measure) leaving an N of 325. Additionally, among the remaining participants, 16 hypnotic susceptibility scales were incomplete due to participant drop-out during the test administration (4 on the HGSHS:A and 12 on the GSHSS:C). Hence, hypnotizability data was only available for 309 participants. Inspection of the preliminary analyses revealed no discernible outliers with respect to multivariate kurtosis; thus, the final sample included the 309 participants with complete date on all measures (the lowest pairwise "N").

Sample Characteristics

The means, standard deviations, and ranges for the model indicators and additional
measures are reported in Table 1 and Table 2, respectively. These results are consistent with another sample drawn from the same university (Lyons & Crawford, 1997) as well as other college populations (e.g., Crawford, 1982; Glisky et al, 1993; Rosen & Petty, 1994).

Principal Components Analyses of DES-II and DIS-Q

A principal components analysis with varimax rotation was conducted on the Dissociative Experiences Scale (DES-II) to parse items into indicators for the latent constructs in the study (i.e., Dissociation & Absorption). The retention of "meaningful" components was based upon the following criteria: (1) item loadings equal to or greater than .40 on a factor and not greater than .395 on another factor; (2) eigenvalues equal to or greater than 1; (3) > 5% of the variance accounted for; and (4) interpretability (Hatcher, 1994). The labels ascribed to the derived indicators are presented in brackets below. Consistent with the literature (e.g., Ross et al., 1991), a three-factor solution was extracted for the DES-II which accounted for 48.6% of the total variance. The first factor, accounting for 19.8% of the variance, contained 11 items representing experiences of absorption (e.g., "absorbed in TV/movies") [DES-ABS]. The second factor, which accounted for 14.9% of the variance and included five diverse items, may be characterized as depersonalization/derealization [DES-DEP]. The final factor, which accounted for 12.1% of the variance, included five items describing states of amnesia [DES-AMN].

Although The Dissociation Questionnaire (DIS-Q) contains four empirically derived subscales (i.e., Identity confusion and alteration, Loss of control, Amnesia, & Absorption) (Vanderlinden et al., 1993), the factor structure in North American populations has yet to be determined. Thus, a principal components analysis with varimax rotation was conducted on the DIS-Q to parse items into indicators for the latent constructs studied. Based on the above criterion, a four-factor solution was extracted which accounted for 44.0 % of the total variance. The first factor, which accounted for 19.7% of the variance, contained 15 items describing experiences of absorption [DIS-ABS]. The second factor, accounted for 11.6% of the variance and included 14 items related to dissociative states of derealization, depersonalization, and identity confusion [DIS-ID]. The third factor, which accounted for 11.1% of the variance, included eight items describing episodes of amnesia [DIS-AMN]. Finally, the fourth factor,
which accounted for 5.1% of the variance, contained 13 items with loss of control as a common theme [DIS-LOC].

**Correlational Analyses**

The Pearson correlation between the GSHSS:C and the HGSHS:A was .72 ($p < .001$). As anticipated, the indicators within each latent construct were significantly correlated. Specifically, the zero-order Pearson correlations among the hypnotizability indicators were .37 to .59; the dissociation indicators were .32 to .63; and the absorption indicators were .44 to .63 (all $p_s < .001$). Furthermore, although not utilized in subsequent analyses, the DES and DIS-Q fullscale scores were highly correlated ($r = .58, p < .001$).

**Overview of Structural Equation Analysis**

Data were analyzed using EQS 5.5a (Bentler & Wu, 1995). The models tested were covariance structure models with a minimum of three indicators for each latent construct. Because the data on several measure were skewed (i.e., DES-DEP, DES-AMN, DISQ-ID), all models were estimated with Bentler's Robust maximum likelihood method which corrects for non-normal data (Byrne, 1994). This method is favored over transforming skewed data because such transformations can lead to changes in the covariance matrix (Byrne, 1998).

In each case, structural equation modeling followed the two-step approach recommended by Anderson and Gerbing (1988) and outlined in Hatcher (1994). First, a confirmatory factor analysis was performed to determine the adequacy of the hypothesized factor loadings, the degree of model fit, and latent construct intercorrelations. At this stage of analysis, all factor loadings were freed, factor variances were constrained to 1.00 (to identify the constructs), and all latent constructs were allowed to correlate freely. In Step two, the measurement models were modified to include the hypothesized causal paths, and a simultaneous test of each measurement model and latent structural model was conducted to estimate hypothesized relationships among constructs. Standardized path coefficients are calculated by EQS for direct effects in the models. These coefficients are analogous to beta weights, indicating the magnitude of influence of one construct (i.e., the independent variable) on another (Hatcher, 1994).

As suggested by Byrne (1994, 1998) and Hatcher (1994), models were evaluated and
compared using the following "goodness of fit" indices: Chi-square ($X^2$), comparative fit index (CFI), non-normed fit index (NNFI), & root mean square error of approximation (RMSEA). The chi-square test provides a statistical test of the null hypothesis that the model fits the data (Hatcher, 1994). If the model provides an acceptable fit to the data, the $X^2$ will be relatively small, and the corresponding $p$-value will be relatively large; however, because the $X^2$ statistic is sensitive to sample size, a reasonably large sample will often result in a significant $X^2$ even when a model adequately describes the data. Interpretively, the CFI and NFI range from 0 to 1.00, with values greater than .90 suggesting a psychometrically acceptable model fit (Crowley & Fan, 1997). The RMSEA provides an index of fit per degrees of freedom, with lower values indicative of a better fit. The standardized residuals (i.e., the difference between the actual covariance matrix and the predicted matrix) of a well specified model are expected to be symmetrical and centered on zero, with values greater than .10 considered problematic (Byrne, 1998). Finally, when competing models were nested (i.e., Models 5 and 6), comparative fit was evaluated using the chi-square difference test (Anderson & Gerbing, 1988; Pedhazur & Schmelkin, 1991).

In total, five models were developed, tested, and revised to achieve an acceptable fit to the data. The initial three models were constructed to derive the measurement models for the latent constructs of interest and to separately test the hypothesized influences of hypnotizability, dissociation, and absorption on bulimic symptomatology. Subsequently, the three models were combined to produce two models which evaluated the simultaneous influences of hypnotizability, dissociation, and absorption on bulimic symptomatology.

**Initial Models**

**Model 1.** This model examined the hypothesized relationship between Hypnotizability and Bulimic Symptomatology. Thus, a CFA was performed to test the hypothesis that Model 1 would provide an acceptable fit to the data. The chi-square value for the model was significant, $X^2 (19, N = 309) = 113.52, p < .001$. Fit indices indicated that this model provided a poor fit to the data, NFI = .90, CFI = .88, RMSEA = .127. However, inspection of the residuals showed that the model fit could be improved if correlated error was estimated between the indicators.
BULIM and BODY. This modification was defensible given that the indicators were subscales from the same measure, and method error was to be expected. Thus, the Model 1 was refitted to the data with this adjustment. In the revised model, the chi-square value was non-significant, $X^2 (18, N = 309) = 25.41, p = .11$. Furthermore, the goodness of fit indices, NFI = .973, CFI = .992, RMSEA = .036, indicated that the model provided a very good fit to the data (see Table 3). Finally, the model’s standardized residuals were symmetrical and centered on zero, with no residuals greater than .08 (mean = .018).

As expected, the standardized factor loadings for the revised model indicators were all significant ($p < .01$) and ranged from .55 to .97, supporting the convergent validity of the indicators and construct validity for the latent constructs Hypnotizability and Bulimic Symptomatology. A significant ($p < .01$) correlation of .56 was also found between BULIM and BODY. This covariation was maintained in the remaining models. Finally, the causal path between Hypnotizability and Bulimic Symptomatology was introduced, and the full structural model was tested. The structural model fit precisely as well as the CFA model, because the correlation between the Hypnotizability and Bulimic Symptomatology was replaced with a path. As Predicted, Hypnotizability was found to have a relatively small, but significant, effect (Path coefficient = .16) on Bulimic Symptomatology.

Model 2. This model investigated the hypothesized link between the latent constructs Dissociation and Bulimic Symptomatology. The chi-square value for the model was significant, $X^2 (18, N = 309) = 74.27, p = .001$. However, the goodness of fit indices, NFI = .928, CFI = .944, RMSEA = .101, indicated that the model provided an adequate fit to the data (see Table 3). Finally, the standardized residuals were relatively symmetrical and centered on zero (mean = .04). The standardized factor loadings for the model indicators ranged from .54 to 1.0, and all were significant ($p < .01$). These results support the convergent validity of the indicators and construct validity for the latent construct Dissociation. The structural model fit precisely as well as the CFA model, because the correlation between Dissociation and Bulimic Symptomatology was replaced with a path. As predicted, Dissociation was found to have a significant effect (Standardized coefficient = .41) on Bulimic Symptomatology.
Model 3. This model investigated the hypothesized relationship between the latent constructs of Absorption and Bulimic Symptomatology. The chi-square value for the model was significant, $X^2 (18, N = 309) = 31.09, p < .05$. As shown in Table 3, the NFI and CFI were greater than .9 and the RMSEA was small, indicating an acceptable fit to the data. Finally, the model's standardized residuals were all less than .08 and centered on zero. The standardized factor loadings for the model indicators ranged from .54 to .99, and all were significant ($ps < .01$). These results provide evidence in support of the convergent validity of the indicators and construct validity for the latent construct Absorption. The structural model fit precisely as well as the CFA model, because the correlation between the Absorption and Bulimic Symptomatology was replaced with a path. As hypothesized, Absorption was found to have a significant effect (Standardized coefficient = .37) on Bulimic Symptomatology.

Comparison of Models 1, 2, and 3

The above results support the first hypothesis that significant paths will be found in the initial models between Bulimic Symptomatology and Hypnotizability, Dissociation, and Absorption. Additionally, these results support the reliability and validity of the model indicators and latent constructs. Table 3 compares the goodness of fit indices for the three initial models. Based on these statistics, the model including Hypnotizability (Model 1) provided the best overall fit to the data, while the model including Dissociation (Model 2) provided a relatively inferior fit. However, because each model provided an acceptable fit and each was theoretically defensible, this led to the specification of Model 4 which combined the three initial models to evaluate the relative influence of the constructs on Bulimic Symptomatology.

Complete Models

Model 4. This model combined the four latent constructs, as defined above, to examine the hypothesized influences of Hypnotizability, Dissociation, and Absorption on Bulimic Symptomatology. The chi-square value for the model was significant, $X^2 (97, N = 309) = 334.82, p < .001$. However, the goodness of fit indices suggested a poor fit to the data: NFI = .853, CFI = .893, RMSEA = .089. Furthermore, review of the standardized residual matrix revealed points of model misfit involving several of the model indicators. Specifically, seven
residuals associated with DES-DEP, DES-ABS, and DISIN were greater than .10. Since Dissociation, Absorption, and Bulimic Symptomatology were each overidentified, with four indicators per latent construct, it was possible to revise Model 4 by eliminating the offending indicators (i.e., DES-DEP, DES-ABS, DISIN) from the measurement model without meaningfully changing the constructs or the structural model (i.e., relations among latent constructs). Indeed, with regard to model modification, deleting parameters (rather than adding them) is a more conservative approach to dealing with points of model misspecification because it minimizes the likelihood of capitalizing on chance characteristics of the sample data (Anderson, & Gerbing, 1988; Crowley & Fan, 1997; Byrne, 1998). The chi-square value for the revised model was significant, $X^2 (58, N = 309) = 31.09, p < .01$, and the goodness of fit indices (NFI = .932, CFI = .967, RMSEA = .053) indicated that the model provided an improved fit to the data (see Table 3). Additionally, the residuals were symmetrical and centered on zero (mean = .026), also suggesting a good fit. The standardized factor loadings for the model indicators ranged from .52 to 1.0, and all were significant ($ps < .01$). Moreover, a strong significant correlation of .77 was found between Absorption and Dissociation, a significant correlation of .16 between Dissociation and Hypnotizability, and a significant correlation of .22 between Absorption and Hypnotizability ($ps < .01$).

Because the correlations between Bulimic Symptomatology and Hypnotizability, Absorption, and Dissociation were each replaced with paths, the structural model fit precisely as well as the CFA model. In contrast to the initial models, only the path between Dissociation and Bulimic Symptomatology was significant (Standardized coefficient = .32). In other words, when controlling for the covariation among the latent constructs, the paths from Hypnotizability and Absorption to Bulimic Symptomatology were small and non-significant in the full model. Thus, these results are in partial support of the hypotheses.

Model 5. Given the high correlation between Dissociation and Absorption in Model 4 (revised), there was a real possibility that the model would be better specified if the two constructs were combined. Hence, in order to test the hypothesis that the constructs were actually independent, though correlated, a fifth model was developed and tested. In Model 5, the
measurement model in Model 4 (revised) was retained, but the structural model was modified. Specifically, the path between the latent constructs Absorption and Dissociation was fixed to 1, and the full model was rerun (i.e., the parameters were reestimated). This alteration resulted in a degradation in fit (see Table 3), thereby providing support for the independence of Dissociation and Absorption and for Model 4 (revised) as the final model. Furthermore, a chi-square difference test, $X^2_{\text{diff}}(2, \ N = 309) = 177.20, p < .001$, also indicated Model 5 significantly degraded the fit.

Discussion

The primary objective of this study was to investigate the cognitive/attentional capacities that may underlie the expression of bulimic symptomatology. Accumulated univariate research has supported hypotheses relating hypnotizability, dissociation, and absorption to bulimic symptomatology; however, a model including all of these constructs had not been developed and tested. Building on past investigations, this study utilized multiple measures for each construct and empirically differentiated between pathological dissociation and absorption (nonpathological dissociation) within a SEM paradigm. Furthermore, unlike past research utilizing college women (i.e., Rosen & Petty, 1994; Valdiserri & Kihlstrom, 1995a, 1995b), the present study included a sample with a high percentage (17.5 %) scoring in the clinical range on the EDI-2 (Garner, 1991).

Several findings from this study are of interest for understanding bulimia and treating patients with bulimic symptomatology. First, as hypothesized, the latent construct Dissociation, defined by experiences of amnesia and identity confusion, was found to have a moderately strong influence on the construct Bulimic Symptomatology (Standardized coefficient = .32). Indeed, the fact that this relationship was found in an ostensibly normative sample of university women adds to a body of literature suggesting that pathological dissociative tendencies may constitute a psychological vulnerability for bulimia. This finding is consistent with recent empirical work on dissociation indicating that only episodes of dissociative amnesia, depersonalization, derealization, and identity confusion are predictive of psychopathological functioning (Waller et al., 1996; Waller & Ross, 1997). Therefore, based on the final structural model (Model 4 -
revised), we can now speak with increased confidence of a meaningful link between pathological dissociation and the continuum of bulimic symptomatology.

Several authors have proposed that a predisposition to dissociate may make individuals more vulnerable to developing certain types of psychiatric disorders (M. Barabasz, 1991; McCallum et al., 1992; Spiegel, Hunt, & Dondershine, 1988). Specifically, it has been hypothesized that individuals with highly developed dissociative skills may utilize these abilities during periods of stress or emotional arousal to defend against intolerable feelings or memories (Spiegel et al., 1990; Vanderlinden & Vandereycken, 1997). If pathological dissociation serves such a function in clinical and sub-clinical bulimic women, this would explain why the construct is associated with bulimic symptomatology (Ludwig, 1983). Avoidance coping via pathological dissociative mechanisms could also explain the loss of control aspect of binge eating which has generally been characterized as a cognitive distortion (Thompson et al., 1987).

Speculatively, a pathological dissociative style, once developed, may reinforce binge eating by blocking awareness of negative affective states. Likewise, dissociation may serve to provide amnesia for episodes of bingeing and purging, thus allowing one to more easily engage in these normally aversive behaviors (Everill et al., 1995a; Reto et al., 1993). The dissociation of normal body cues (e.g., hunger and satiety) from memory or self-awareness may also facilitate disinhibited eating by interfering with normal homeostatic mechanisms. Nevertheless, it is possible that dissociation operates indirectly, perhaps by activating an individual's vulnerability to other risk factors more proximal to bingeing and purging such as reduced self-efficacy for managing stress or negative emotional states.

Despite these theories, the high prevalence of subclinical bulimia and the corresponding low base rate of pathological dissociation (Waller & Ross, 1997) seems to suggest that dissociative mechanisms may only contribute to bulimic symptoms in a subsample of individuals (Torem, 1986). For instance, it has been proposed by Everill et al. (1995) that dissociation and bulimic symptomatology may only be related in female victims of physical or sexual abuse. Additionally, in contrast to all of these theories, it is also possible that bulimic symptomatology "causes" dissociation. This model has been proposed by some authors who hypothesize that
Dissociation and Bulimia

Binge eating precipitates a period of dissociative "self-soothing" (e.g., Conner, 1994). Due to the correlational design of the present study, the question of cause-effect must be left to future prospective research.

The hypotheses regarding Hypnotizability and Bulimic Symptomatology were also supported. In contrast to Model 1, a relationship between Hypnotizability and Bulimic Symptomatology was not demonstrated after controlling for the covariation among the latent constructs in the final model. Hypnotizability has been proposed by some researchers as a predisposing factor for bulimia, possibly mediated by socio-cultural influences resulting in the internalization of unrealistic physical appearance ideals (Fasquilho & Oakley, 1997; Groth-Marnat & Schumaker, 1990). Indeed, the ability of moderate to highly hypnotizible individuals to generate and sustain perceptual distortions is well documented (e.g., Kihlstrom, 1985). Furthermore, binge episodes may involve a hypersensitivity to external cues (Heatherton & Baumeister, 1991), a quality phenomenologically similar to the heightened suggestibility found during hypnosis (Hilgard, 1965). However, the present study suggests that while many of the behaviors and personality factors which characterize bulimia lie on a continuum, high hypnotizability may be a trait found only among bulimic patients or eating disordered patient with bulimic tendencies (e.g., non-restricting anorexics). Indeed, prior studies which have found a relationship between hypnotic susceptibility and bulimic symptomatology in college women (Brown & Crawford, 1986; Frasquilho & Oakley, 1997; Groth-Marnat & Schumacker, 1990) should be viewed with some skepticism since they failed to use the SHSS:C (or GSHSS:C), the "gold standard" measure of hypnotizability (Hilgard, 1965; Kihlstrom, 1985).

It must be noted that this finding (i.e., lack of association between Hypnotizability and Bulimic Symptomatology) should not be interpreted as contrary to the demonstrated effectiveness of hypnosis in the treatment of bulimia and other eating disorders. In fact, a fairly robust literature attests to the clinical utility of hypnotic techniques with eating disorders (for a reviews, see Vanderlinden & Vandereycken, 1997; Pettinati et al., 1989). Many individuals with clinical and subclinical bulimia are moderate to highly hypnotizible, and hypnosis may be a beneficial component to a multifaceted treatment approach for bulimia (e.g., Griffiths, 1995;
Vanderlinden & Vandereycken, 1997).

Finally, the results did not support the prediction of a significant path between Absorption and Bulimic Symptomatology in the complete model. Based on Baumeister and Heatherton’s (1991) model of binge eating and empirical research linking hypnotizability and absorption, it was hypothesized that a significant path would be found. Although a relatively strong relationship between Absorption and Bulimic Symptomatology was found in Model 3, the path became non-significant in the final model (Model 4 - revised). This indicates that extremely focused attentional skills probably do not play a direct role in the development of bulimia. Whether absorption is a trait found in patients with bulimia, however, remains to be seen. Furthermore, the high significant correlation between the constructs Absorption and Dissociation ($r = .77$) suggests that these capacities are nonetheless closely related. This finding suggests that a capacity for absorption may be necessary for the development of a pathological dissociative style (Waller & Ross, 1997; Frischholz, 1985).

Although the present study sought to bridge some of the gaps in the extant literature, there are several important limitations to this research. First, although the final model provided an adequate fit to the data, structural equation modeling can not prove the validity of the proposed model; alternative models with equal or better fit are likely to exist (Anderson & Gerbing, 1988). The process of using the sample data in conjunction with past research and substantive theory for model generation in the early stages of theory development is well accepted in the literature (Anderson & Gerbing, 1988; MacCallum, 1995). Because the present study depended on several post hoc (i.e., data-driven) modifications to the measurement model, the findings from this investigation must be viewed as tentative until they are cross-validated in an independent sample of university women. In the meantime, it will remain unclear whether the present results capitalized on the idiosyncrasies of the sample. Relatedly, due to the relatively homogenous sample of college women included in the study, these results should be replicated with other non-clinical populations to establish the generality of the findings. Finally, future studies are needed to model relationships between dissociation and other known risk factors and to investigate the temporal association of proposed mediators of bulimic symptomatology.
In conclusion, the results of this study support past research and theory linking pathological dissociation and bulimic symptoms, cognition, and behaviors. Beyond theoretical interest, a greater appreciation for the role of dissociation in bulimia may facilitate treatment efforts. Clinicians working with eating disordered patients (or adolescent women with bulimic tendencies) should be cognizant of the possible role of pathological dissociative processes and seek to reduce the use of these maladaptive coping mechanisms. This study supports a greater treatment focus on the loss of control component of binge eating which is less amenable to change than abnormal eating patterns (Russ, 1998). Indeed, the automaticity associated with dissociative experiences may reduce an individual's sense of control and responsibility over eating behavior, and thus foster treatment resistance (McManus, 1995).
Table 1 Descriptive Statistics for Modal Indicators

<table>
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<tr>
<th>Latent Factor &amp; Indicator</th>
<th># Items</th>
<th>Mean</th>
<th>SD</th>
<th>Range</th>
<th>Alpha</th>
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<td>6.63</td>
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<td>0 - 3</td>
<td>.63</td>
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<td>DISIN</td>
<td>16</td>
<td>6.37</td>
<td>3.32</td>
<td>1 - 16</td>
<td>.79</td>
</tr>
</tbody>
</table>

Note. NA = Not available.
Table 2 Descriptive Statistics for Additional Measures

<table>
<thead>
<tr>
<th>Measure</th>
<th>Mean</th>
<th>SD</th>
<th>Range</th>
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<tbody>
<tr>
<td><strong>Hypnotizability</strong></td>
<td></td>
<td></td>
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<tr>
<td>GSHSS:C</td>
<td>5.75</td>
<td>2.81</td>
<td>0 - 12</td>
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<tr>
<td><strong>Dissociation Questionnaire (DIS-Q)</strong></td>
<td></td>
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<tr>
<td>Full Scale DIS-Q</td>
<td>2.07</td>
<td>0.50</td>
<td>1 - 3.83</td>
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<tr>
<td>Loss of Control</td>
<td>2.41</td>
<td>0.62</td>
<td>0 - 4.34</td>
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<tr>
<td><strong>Dissociative Experiences Scale (DES)</strong></td>
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<tr>
<td>Full Scale DES</td>
<td>14.49</td>
<td>10.14</td>
<td>0 - 59.64</td>
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<tr>
<td><strong>Differential Attentional Processes Questionnaire (DAPI)</strong></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Moderately Focused Attention</td>
<td>30.91</td>
<td>8.77</td>
<td>9 - 49</td>
</tr>
<tr>
<td>Dual Attention: Cognitive-Cognitive</td>
<td>10.97</td>
<td>4.34</td>
<td>4 - 27</td>
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<tr>
<td>Dual Attention: Cognitive-Physical</td>
<td>26.36</td>
<td>5.00</td>
<td>7 - 35</td>
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<tr>
<td><strong>Three Factor Eating Questionnaire (TFEQ)</strong></td>
<td></td>
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<tr>
<td>Cognitive Restraint</td>
<td>16.04</td>
<td>7.87</td>
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<tr>
<td>Hunger</td>
<td>8.47</td>
<td>3.73</td>
<td>1 - 17</td>
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*Note.* GSHSS:C = Group Stanford Hypnotic Susceptibility Scale, Form C.
### Table 3  Comparison of Structural Equation Models: Goodness of fit indices

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<tr>
<th>Model</th>
<th>Chi-square</th>
<th>df</th>
<th>NNFI</th>
<th>CFI</th>
<th>RMSEA</th>
<th>Mean Residual</th>
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<tr>
<td>Model 1</td>
<td>25.42</td>
<td>18</td>
<td>.987</td>
<td>.992</td>
<td>.037</td>
<td>.018</td>
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<tr>
<td>Model 2</td>
<td>74.27**</td>
<td>18</td>
<td>.913</td>
<td>.944</td>
<td>.101</td>
<td>.040</td>
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<tr>
<td>Model 3</td>
<td>31.09*</td>
<td>18</td>
<td>.980</td>
<td>.987</td>
<td>.049</td>
<td>.022</td>
</tr>
<tr>
<td>Model 4 (revised)$</td>
<td>107.88**</td>
<td>58</td>
<td>.955</td>
<td>.967</td>
<td>.053</td>
<td>.026</td>
</tr>
<tr>
<td>Model 5</td>
<td>261.23**</td>
<td>59</td>
<td>.823</td>
<td>.866</td>
<td>.106</td>
<td>.076</td>
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</tbody>
</table>

Note. $ Final model;  *p < .05.  **p < .001.
References

Abraham, S. F., & Beumont, P. J. (1982). How patients describe bulimia or binge eating. Psychological Medicine, 12, 625-635.


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Dissociation and Bulimia


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Gruzelier, J. H. *Personality Syndrome Questionnaire*, Unpublished measure, Department of Psychiatry, Charring Cross and Westminster Medical School, London W6 8RF, UK.


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and hypnotic experiences in eating disorder patients: An exploratory study. American Journal of
Clinical Hypnosis, 38, 97-108.

Dissociative experiences and trauma in eating disorders. International Journal of Eating
Disorders, 13, 187-193.

Dissociative and traumatic experiences in the general population of the Netherlands. Hospital
and Community Psychiatry, 44, 786-788.

Vanderlinden, J., van Dyck, R., Vandereycken, W., Vertommen, H., & Jan Verkes, R.
(1993). The dissociation questionnaire (DIS-Q): Development and characteristics of a new self-

Waller, N. G., Putnam, F. W., Carlson, E. B. (1996). Types of dissociation and
dissociative types: A taxometric analysis of dissociative experiences. Psychological Methods, 1,
300-321.

Waller, N. G., & Ross, C. A. (1997). The prevalence and biometric structure of
pathological dissociation in the general population: Taxometric and behavior genetic findings.


APPENDIX A

CONSENT FORM

TITLE OF EXPERIMENT: Cognitive Processing and Personality Styles: Relationships to Hypnotic Susceptibility

1. PURPOSE OF EXPERIMENT
You are invited to participate in a study of individual differences in cognitive processing, personality styles, and eating styles and their relationships to hypnotic susceptibility. You have already participated in our assessment of hypnotic susceptibility in our laboratory.

2. PROCEDURE TO BE FOLLOWED IN THE STUDY:
To accomplish the goals of the study, you will be asked to complete a packet of questionnaires. Please feel free to browse through the packet and ask any questions you may have. The questionnaires are about characteristics and attitudes of yourself, the degree to which you attend to some things in the environment and ignore other things, the vividness of your visual imagery, eating styles, and life experiences you may have had. This experiment will take approximately two hours. You will be provided with extra credit for a psychology course if you so desire. Please check with your course's syllabus for information about how to receive extra credit. If you decide to withdraw at any time during the experiment today, we will give full extra credit for participation.

3. ANONYMITY OF SUBJECTS AND CONFIDENTIALITY OF RESULTS:
The results of this study will be kept strictly confidential. At no time will the researchers release your results to anyone without your written consent. The information you provide will have your name removed and only a subject code will identify you during analyses and any write-up of the research.

4. DISCOMFORTS AND RISKS FROM PARTICIPATING IN THE STUDY:
There are no apparent risks to you from participation in this study. Should any of the questions raise issues you wish to discuss with someone, we suggest you contact one of the following agencies: Virginia Tech Counseling Center (231-6557); RAFT Crisis Center (552-5707)

5. BENEFITS OF THIS PROJECT:
Your participation in this project today will help advance the scientific knowledge of cognitive processes and personality styles and their relationship to hypnotic susceptibility. No guarantee of benefit has been made to induce your participation. You may receive extra credit towards one designated psychology course, if that is offered, for participation in each of the two hours of research. Please check with your instructor for alternative ways by which you may receive extra credit in the course.

6. FREEDOM TO WITHDRAW:
You are free to withdraw from this study at any time without penalty.

7. USE OF RESEARCH DATA:
The information from this research may be used for scientific or educational purposes. It may be presented at scientific meetings and/or published and reproduced in professional journals or books, or used for any other purpose that Virginia Tech's Department of Psychology considers proper in the interest of education, knowledge, or research.
8. APPROVAL OF RESEARCH:
   This research project has been approved by the Human Subjects Committee of the
   Department of Psychology and by the Institutional Review Board of Virginia Tech.

9. SUBJECT'S PERMISSION:
   I have read and understand the above description of the study. I have had an opportunity to
   ask questions and have had them all answered. I hereby acknowledge the above and give my
   voluntary consent for participation in this study. I further understand that if I participate I may
   withdraw at any time without penalty. I understand that should I have any questions regarding this
   research and its conduct, I should contact any of the persons named below:

   Primary Researcher:                   Helen J. Crawford, Ph.D.              231-6520
   Researcher                          Daniel I. Galper, M.S.               552-6033
   Chair, Human Subjects Committee:    Richard Eisler, Ph.D.                231-7001
   Chair, Institutional Review Board:   H.T. Hurd                           231-5281

   ___________________________________________ ____________________
   Participant Signature                Date
CURRICULUM VITAE
1999
DANIEL I. GALPER

PERSONAL INFORMATION

Birth Date: November 30, 1966
Birth Place: Bethesda, Maryland
Marital Status: Married; October 19, 1996

Home Address: 307 15th Street, N.W. #13
Charlottesville, VA 22903
(804) 971-1316

School Address: Department of Psychology,
Virginia Polytechnic Institute & State University,
Blacksburg, VA 24061 - 0436
(540) 231-6581

E-Mail: dg4w@virginia.edu; dgalper@vt.edu

EDUCATION

July, 1998 - Present
Pre-doctoral Residency (APA Accredited)
University of Virginia School of Medicine,
Department of Psychiatric Medicine, Charlottesville, VA
Rotations in Psychological Oncology, Behavioral Medicine, and Inpatient Psychiatry.
Training Director: Patrick C. Fowler, Ph.D.

August, 1992-Present
Virginia Polytechnic Institute and State University (VPI & SU), Blacksburg, Virginia
Specialization: Clinical health psychology
Advisor & Dir. of Clinical Training: Richard A. Winett, Ph.D.

M.S. conferred May, 1995
Ph.D. candidate (degree expected May, 1999)
Dissertation: Bulimic Symptomatology in College Women:
To What Degree are Hypnotizability, Dissociation, and Absorption of Relevance?
Co-chairs: Richard A. Winett, Ph.D. & Helen J. Crawford, Ph.D.

September, 1984-May, 1990
Duke University,
Durham, North Carolina
B.A. awarded May, 1990
Major: Psychology
PROFESSIONAL ACTIVITIES

Research

May, 1995-Present
Cooper Institute for Aerobics Research, Dallas, TX
Epidemiology and Clinical Applications
Director: Steven N. Blair, P.E.D.;
Associate Director: Andrea L. Dunn, Ph.D.
Title: Intern & Research Collaborator
Role: Cooperate in on-going National Heart, Lung, & Blood Institute (NHLBI) sponsored physical activity research. Activities have included: Focus group facilitation, participant recruitment, data collection & analysis, conference presentations, and manuscript preparation.

January, 1996-Present
Neurocognition Laboratory, VPI & SU
Title: Graduate Research Assistant
Director: Helen J. Crawford, Ph.D.
Role: Co-investigator of two studies on cognitive/personality predictors of hypnotizability & dissociation and one study on cognitive correlates of pain and pain control in college students (e.g., self-efficacy). I have been trained in (experimental) hypnotic pain control and I have administrated & scored approximately 1000 hypnotizability scales for research on cognitive functioning & hypnotic analgesia.

August, 1992-June, 1998
Center for Research in Health Behavior, VPI & SU
Title: Graduate Research Assistant
Director: Richard A. Winett, Ph.D.
Role: Activities have included: NCI & NIH grant development, project consultation, & participant recruitment.

October, 1995-April, 1997
Department of Public Health Sciences,
Bowman Gray School of Medicine,
Wake Forest University, Winston-Salem, NC
Title: Project Consultant
Primary Investigator: David G. Altman, Ph.D.
Role: Co-developer of the 4-S Program, a prototype "self-help" (cognitive-behavioral) physical activity program for senior citizens. I created the strength training & flexibility components, directed the instructional video, and field tested the program in a community sample of older adults.

August, 1993-September, 1995
Community Skin Cancer Prevention Project,
Center for Research in Health Behavior, VPI & SU
Title: Graduate Research Assistant
Supervisors: Richard A. Winett, Ph.D., David N. Lombard, Ph.D., & Bonnie S. Cleaveland, Ph.D.
Role: Assisted with intervention development, delivery, & data collection for a community skin cancer prevention project.
funded by the American Cancer Society.

**Stress Laboratory, Department of Psychology, VPI & SU**

**Title:** Graduate Research Assistant  
**Director:** Russell T. Jones, Ph.D.  
**Role:** Developed a data coding system & supervised the coding of disaster (hurricane & fire) coping data.

---

**Laboratory for Children's Health Promotion,**  
Department of Community & Family Medicine,  
Georgetown University School of Medicine, Washington, DC  
**Directors:** Ronald J. Iannotti, Ph.D. & Patricia J. Bush, Ph.D.  
**Title:** Clinical Health Researcher  
**Role:** Collected data with clinical interviews and surveys (on childrens' health behaviors & attitudes towards AIDS); Assisted with project development; Coded, entered, and managed data.

---

**Teaching**

**January, 1995-**  
**Department of Psychology, VPI & SU**  
**Title:** Graduate Teaching Assistant (GTA)  
**Supervisor:** Robert S. Stephens, Ph.D.  
**Role:** GTA for Introduction to Substance Use and Abuse (senior seminar); Aided in course organization, attended lectures, & graded papers (for 60 undergraduates).

**August, 1994-**  
**Department of Psychology, VPI & SU**  
**Title:** Graduate Teaching Assistant  
**Supervisor:** Joseph A. Sgro, Ph.D.  
**Role:** Taught Introduction to Psychology laboratories (6 Sections; 35 students/section); Conducted lectures, discussions, experiments, & quizzes.

**October, 1995**  
**Department of Psychology, VPI & SU**  
**Guest Lecturer: Introduction to Clinical Psychology**  
Invited speaker for undergraduate course  
**Topic:** Behavioral medicine.

**November, 1995**  
**Department of Psychology, VPI & SU**  
**Guest Lecturer: Behavior Modification**  
Invited speaker for undergraduate course  
**Topic:** Multiple baseline design.

**July, 1997**  
**Center for Rehabilitative Medicine, Radford, VA**  
**Lecturer: In-service training**  
**Topic:** Applications of hypnosis in pain management & rehabilitative medicine.

**October, 1998**  
**UVA Cancer Center, Charlottesville, VA**  
**Guest Lecturer: Lousia Cancer General Support Group**  
**Topic:** Can you learn to enjoy exercise?
CLINICAL EXPERIENCE

Positions held

July, 1998- Present
University of Virginia Health Sciences Center
Department of Psychiatric Medicine, Charlottesville, VA
Director of Clinical Training: Patrick C. Fowler, Ph.D.
Title: Pre-doctoral Resident in Clinical Psychology
Psychooncology (6-month Primary Rotation):
Provided individual, couples, & family therapy for inpatients and outpatients @ UVA Cancer Center; Coping skills training (e.g., relaxation, hypnosis, pain management) for patients with all stages of disease. Co-facilitated support groups for patients & caregivers.
Supervisors: Joseph R. Dane, Ph.D. & Lora D. Baum, Ph.D.

Behavioral Medicine (12-months Secondary Rotation; In progress):
Provide individual cognitive-behavioral therapy & biofeedback for a diverse medical patient population (e.g., insomnia, chronic illness & pain, diabetes, & anxiety disorders); Complete gastric bypass surgery evaluations for morbidly obese patients; Provide psychology consultation to UVA medical clinics.
Supervisors: Linda Gonder-Frederick, Ph.D. & Daniel Cox, Ph.D., ABPP

Inpatient Psychiatry (6-month Primary Rotation; In progress):
Conduct individual and group therapy with chronically mentally ill patient @ Western State Hospital. Most patients suffering from schizophrenia, bipolar disorder, Schizoaffective disorder, substance dependence, and/or a severe Axis II disorder.
Supervisor: Marc Hastings, Ph.D.

February, 1997- June, 1998
Center for Rehabilitative Medicine, Radford, VA.
Comprehensive Pain Management Program (CARF).
Title: Psychology Intern
Hours Completed: 900
Responsibilities: Assist with implementation and evaluation of the comprehensive pain management program. Activities included: Intake interviews, multimethod pain/psychosocial assessment and report writing (80 reports), individual & group psychotherapy, and coping skills training (e.g., pain/stress management, hypnosis, assertiveness, & relaxation) for both inpatients and outpatients.
Supervisor: Roy H. Crouse, Ph.D., ABPP
## Clinical Practica

<table>
<thead>
<tr>
<th>Date Range</th>
<th>Location and Description</th>
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<tbody>
<tr>
<td>August, 1996-</td>
<td>Psychological Services Center &amp; Child Study Center, Department of Psychology, VPI &amp; SU.</td>
</tr>
<tr>
<td>May, 1997</td>
<td>Hours completed: 240. <strong>Responsibilities:</strong> Outpatient assessment and treatment of adults and children exhibiting pain disorder, ADHD, &amp; conduct disorder. Supervised first (2) &amp; second (2) year practicum students. <strong>Supervisor:</strong> Cynthia A. Lease, Ph.D.</td>
</tr>
<tr>
<td>August, 1995-</td>
<td>Psychological Services Center &amp; Child Study Center, Department of Psychology, VPI &amp; SU.</td>
</tr>
<tr>
<td>May, 1996</td>
<td>Hours completed: 240. <strong>Responsibilities:</strong> Outpatient assessment and treatment of adults exhibiting personality disorders, depression, &amp; anxiety; Developed and implemented a minimal contact smoking cessation program utilizing self-hypnosis. <strong>Supervisors:</strong> Robert S. Stephens, Ph.D. &amp; Jack W. Finney, Ph.D.</td>
</tr>
<tr>
<td>May, 1995- May, 1996</td>
<td>Cooper Institute for Aerobics Research, Dallas, TX, Epidemiology and Clinical Applications.</td>
</tr>
<tr>
<td>August, 1995-</td>
<td>Hours completed: 400 <strong>Responsibilities:</strong> Helped facilitate a &quot;lifestyle&quot; (cognitive-behavioral) physical activity intervention for sedentary adults based on the stages of change model; assisted with fitness &amp; activity assessments, and developed intervention materials. <strong>Supervisor:</strong> Andrea L. Dunn, Ph.D.</td>
</tr>
<tr>
<td>December, 1994-</td>
<td>Cardiac Rehabilitation Program, VPI &amp; SU.</td>
</tr>
<tr>
<td>May, 1995</td>
<td>Hours completed: 120. <strong>Responsibilities:</strong> Counseled individual patients on stress reduction, diet management, and exercise adherence; Facilitated group activities. <strong>Supervisor:</strong> Douglas R. Southard, Ph.D., M.P.H.</td>
</tr>
<tr>
<td>August, 1993-</td>
<td>Psychological Services Center &amp; Child Study Center, Department of Psychology, VPI &amp; SU.</td>
</tr>
<tr>
<td>May, 1994</td>
<td>Hours completed: 480. <strong>Responsibilities:</strong> Outpatient assessment and treatment of adults, couples, and children exhibiting PTSD, depression, antisocial personality &amp; dissociative disorders, ODD, marital discord, &amp; relationship issues. <strong>Supervisors:</strong> George A. Clum, Ph.D., ABPP &amp; Russell T. Jones, Ph.D.</td>
</tr>
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</table>
June, 1993- August, 1993

**Psychological Services Center & Child Study Center, Department of Psychology, VPI & SU.**

Hours completed: 240.

**Responsibilities:** Outpatient assessment and treatment of families, children, and adults exhibiting depression, anxiety, personality disorders, & ADHD.

**Supervisor:** Richard M. Eisler, Ph.D.

August, 1992- May, 1993

**Psychological Services Center & Child Study Center, Department of Psychology, VPI & SU.**

Hours completed: 240.

**Responsibilities:** Outpatient assessment and psychotherapy with children, adults, and couples exhibiting depression, anxiety, substance dependence, ADHD, ODD, & LD.

**Supervisors:** Thomas H. Ollendick, Ph.D. & Ellie T. Sturgis, Ph.D.

### Other Clinical Activities

**May, 1991- August, 1992**

**PhoneFriend: Crisis Helpline for Children, Wash, DC**

**Title:** Hotline Volunteer

**Role:** Phone counselor for children in crisis; Trained new volunteers in reflective listening skills & crisis management.

(3-6 hours/week).

**March, 1989- August, 1989**

**John Umstead State Psychiatric Hospital, Butner, North Carolina**

**Title:** Volunteer Recreation Therapist

**Role:** Supervised recreational activities for severely disturbed patients on a closed psychiatric unit. (8 hours/week).

### OTHER EMPLOYMENT

**June, 1990- July, 1992**

**Personal Fitness, Washington, DC**

**Title:** Corporate Health & Fitness Counselor

**Role:** Conducted fitness testing (strength, aerobic capacity, flexibility, & bodyfat) & individualized training.

**Supervisor:** Douglas Baumgarten, M.S., Director

### SPECIALIZED TRAINING

Clinical Hypnosis 10 workshops (over 140 hours training), coursework, individual training, & supervision in clinical hypnosis.

### PROFESSIONAL CERTIFICATIONS

- Advanced Physical Fitness Specialist (Cooper Institute for Aerobic Research [CIAR])
- Biomechanics of Strength Training Specialty Certification (CIAR)
- Stress Management (American Association of Lifestyle Counselors)
- CPR (American Heart Association)
PROFESSIONAL AFFILIATIONS

Association for the Advancement of Behavioral Therapy (AABT)
American College of Sports Medicine (ACSM)
American Psychological Association (APA) & Divisions 30 & 38
American Pain Society (APS)
American Society of Clinical Hypnosis (ASCH)
Society of Behavioral Medicine (SBM)
Society for Clinical and Experimental Hypnosis (SCEH)

AWARDS & HONORS

Erika Fromm & Milla Alihan Scholarship Award, SCEH, 1998
Society of Behavioral Medicine Citation Poster, 1992
Dean's List, Duke University, 1989-1990

PUBLICATIONS

Peer-reviewed articles & book chapters


**Published abstracts (peer-reviewed)**


**Manuscripts**

Dissociation and Bulimia


Other publications (non peer-reviewed)


PROFESSIONAL PRESENTATIONS


WORKSHOPS TAUGHT


EDITORIAL SERVICES

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<tr>
<td>March, 1993-</td>
<td>Ad Hoc Reviewer, Journal of Applied Behavioral Analysis</td>
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<tr>
<td>May, 1995</td>
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COMMITTEES SERVED

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<th>Role Description</th>
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<tr>
<td>October, 1997-</td>
<td>Representative, Education &amp; Training Committee</td>
</tr>
<tr>
<td>Present</td>
<td>Society for Clinical &amp; Experimental Hypnosis</td>
</tr>
<tr>
<td>June, 1997-</td>
<td>Co-chair, Student Relations Committee</td>
</tr>
<tr>
<td>Present</td>
<td>Society for Clinical &amp; Experimental Hypnosis</td>
</tr>
<tr>
<td>September, 1997-</td>
<td>Student Representative to Clinical Psychology Faculty</td>
</tr>
<tr>
<td>May, 1998</td>
<td>Virginia Polytechnic Institute &amp; State University</td>
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