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Investigating the Bulimia-Depression Relationship using Sleep Deprivation

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INVESTIGATING THE BULIMIA-DEPRESSION RELATIONSHIP USING SLEEP DEPRIVATION

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APPROVAL SHEET

This thesis is submitted in partial fulfillment of the requirements for the degree of

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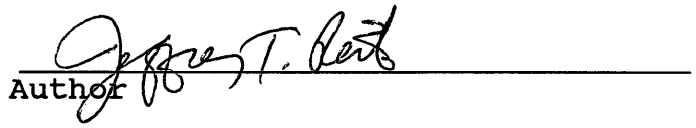

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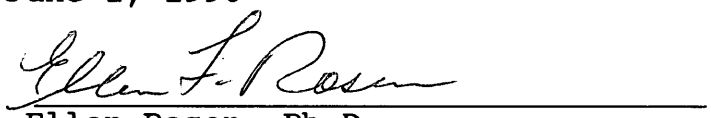

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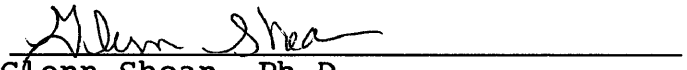

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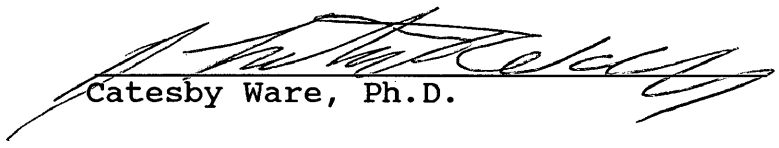

Catesby Ware, Ph.D.

DEDICATION

To my mother and father, whose support seems to know no bounds.

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ABSTRACT

Pope and Hudson (1988) recently proposed the hypothesis that bulimia is a variant form of depression. The present study tested this using a sleep deprivation procedure. One night of sleep deprivation often temporarily reduces depression (Pflug, 1976), and thus it was hypothesized that if bulimia is a form of depression, it too should be reduced by sleep deprivation.

Undergraduate women (n=27) qualifying as bulimic and depressed, bulimic and nondepressed, nonbulimic and depressed, or nondepressed and nonbulimic attended one day (control) and one night (sleep-deprivation) session. Pre- and post-measures of depressive and bulimic psychopathology were obtained at each session.

After correcting inaccurate groupings of the subjects, significant sleep-deprivation-produced increases in depression were found among nondepressed, but not depressed, subjects; bulimic symptoms, though, were unaffected. These findings are opposite those predicted by Pope and Hudson's hypothesis. The results concur with other hypotheses that sleep has a depressiogenic effect on depressed persons, and that bulimia precedes depression etiologically.

INVESTIGATING THE BULIMIA-DEPRESSION RELATIONSHIP USING SLEEP DEPRIVATION

Investigating the Bulimia-Depression Relationship Using Sleep Deprivation

Although the existence of eating disorders dates back to as early as Morton's (1689) description of an anorexic, prior to the 1970's they were considered relatively rare afflictions. Since that time, however, they have burgeoned at an alarming rate, prompting some to pronounce them as an "epidemic of the 80's" (Pope & Hudson, 1988).

Initially, the diagnostic category of anorexia nervosa was used exclusively in the classification of eating disorders, but as research grew, the disorder bulimia nervosa came to be recognized as a second distinct category. DSM-III-R (American Psychiatric Association, 1987) lists the diagnostic criteria for bulimia nervosa as follows: (1) recurrent episodes of binge eating; (2) a feeling of lack of control over eating behavior during the eating binges; (3) regular engagement in either self-induced vomiting, use of laxatives or diuretics, strict dieting or fasting, or vigorous exercise in order to prevent weight gain; (4) a minimum average of two binge eating episodes a week

for at least three months; (5) persistent overconcern with body shape and weight.

As Russell (1979) first noted, the primary difference between anorexia and bulimia is the occurrence of binge eating, together with the often habitual nature of vomiting or purging, in the latter. Both share an abnormal overconcern with body size, namely an intense fear of fatness, but anorexics display less frequent and habitual vomiting/purging, and usually do not experience binge eating. Additionally, bulimics may menstruate regularly, continue normal sexual activity, remain fertile, and be of normal weight or even slightly overweight; anorexics rarely demonstrate any of these regularities. Yet while the symptomatology of bulimia may seem from this description to be less severe than that of anorexia, a poorer prognosis is usually assigned to bulimics (Garner, Moldofsky, Garner, 1980; Russell, 1979; Hsu, 1980). This is possibly due to the habitual nature of the binge/purge cycle, the interrupting of which is very difficult; indeed, bulimics have reported such a task to be as difficult as stopping alcohol abuse,

smoking, or even drug abuse (Russell, 1979).

Thus, much is known presently about bulimia, but much also remains unknown. There are a great many ideas concerning its pathogenesis, yet the complexity of the disorder has so far obstructed any attempts to explain it in a single, consistent theory. There is certainly the chance that we will never fully understand all of the forces at work in the development of bulimia; as Yager (1984) has commented, "Illnesses have a peculiar way of coming and going without our ever really understanding the critical reasons for their existence" (p. 431). Yet amidst the sea of possibilities there are several consistencies among patients with bulimia, and from these researchers have been able to formulate coherent ideas regarding the etiology of the disorder.

Specific precipitating events that are inevitably followed by bulimia cannot be identified in all people. Such initiators are not only often impossible to extract from the mass of possible initiators, but they also probably vary greatly from one person to the next. Whereas mounting academic pressure from parents may

eventually lead to bulimia in one person, the loss of a loved one may be the prime initiator of the illness in another person. In his classic study of bulimia, Russell (1979) reported 22 of 30 patients as having at least minor premorbid personality disturbances. However, there was no pattern to these histories, leading Russell to the conclusion that, "no characteristic type of personality could be said to have preceded the illness" (p. 442).

Nonetheless, a profusion of hypotheses have been generated in the attempt to delineate a pathogenesis for bulimia. Generally, these hypotheses have emphasized one of seven orientations: developmental (Crisp, 1983; Russell, 1983); genetic (Brisman & Siegel, 1984; Garfinkel & Garner, 1983); endocrinological (Weiner, 1977); biological (Dubois, Gross, Ebert, & Castell, 1979); effects of some other psychiatric disorder (Solyom, Thomas, Freeman, & Miles, 1983; Crisp, 1983); familial (Minuchin, Rosman, & Baker, 1978; Rakoff, 1983); and sociocultural (Garner & Garfinkel, 1980; Striegel-Moore, Silberstein, & Rodin, 1986). Recently, however, much attention has been

given to what Agras & Kirkley (1985) call "the most prominent biological theory" (p. 374), specifically the attempt to link bulimia to affective disorder. The investigation of this proposed relationship is the concern of the present study.

Although Agras & Kirkley's statement labels this theory as a biological one, it actually is multidimensional, attracting the attention of a number of areas of research. Beginning with the first formal suggestion of a relationship by Hudson, Laffer, & Pope in 1982, there has been a steady accumulation of diffuse findings. It is as a result of this heterogeneity, however, that proponents of the theory have been able to construct a supportive foundation of empirical evidence.

In a plea for parsimony, led by the slogan of Ockham's razor--"don't posit plurality without necessity"--Pope & Hudson (1988) have recently articulated their theory that bulimia is a variant form of affective disorder. They assert that the present accumulation of research indicates that bulimia nervosa and major affective disorder share a common biological

abnormality, and further, that both can thus be treated successfully with thymoleptic medications. Drawing analogies from the history of medicine, they acknowledge that bulimia is heterogeneous along many axes, but note that the same can be said of many illnesses. Although the physical and psychopathological symptomatology of bulimics can vary widely, the ultimate realization is that diagnostically all bulimics are the same. There is a stereotypy of the "core" bulimic symptoms, such that they are the same throughout time, across age, and across gender. Psychological and sociological factors, they assert, may influence the appearance of bulimia, and are worthy of empirical attention, but this influence must not negate the possibility of a common biological abnormality. AIDS, for example, is an illness that has a common biological abnormality, yet is also influenced by sociocultural factors such as drug addiction.

Of course, not everyone agrees with the arguments of Pope and Hudson, and thus what follows is a review of the basic areas of research from which Pope and Hudson have formulated their theory. From this, the

reader will hopefully gain a sense of the foundation upon which the present study is based.

A common argument used by those supportive of the bulimia-depression link is the often noted presence of depression among bulimics and anorexics. Indeed, this has been noted by many as a feature of the disorders (Russell, 1979; Cooper & Fairburn, 1986; Hudson, Pope, Yurgelun-Todd, Jonas, & Frankenburg, 1987; Striegel-Moore et al., 1986), although it is probably less common among anorexics (Katz et al., 1984; Garfinkel et al., 1980). Sadness, dejection, and despair are frequently seen in patients, and suicide is not unusual (Katz et al., 1984; Russell, 1979). These observations do not appear to be limited to those bulimics who seek treatment (Hudson et al., 1987), thus dampening the possibility that only the more depressed bulimics are the ones being included in clinical studies.

To draw conclusions regarding a bulilmia-depression link from this clinical evidence, however, may be misleading. Some researchers have suggested that, despite the correlation of symptoms, the presence of depression does not necessarily support its possible

role as a causal agent in bulimia. As Walsh, Roose, Glassman, Gladis, & Sadik (1985) have noted, bulimics may have depressed mood, but "it is not known how many develop a full depressive syndrome, and, if they do, what the characteristics of the depression are, and whether it precedes or follows the development of the eating disorder" (p. 124). With regard to these questions, Walsh et al. studied the histories of current and past psychiatric disturbances in 50 bulimic patients, finding that 70% had experienced a major depressive episode at some point in their lives, but that the episodes were of varying types and in fact followed the eating disorder in 74% of the patients. They concluded that, "it is premature at this point simply to reduce bulimia to a form of affective illness" (p. 129).

Similar conclusions have been drawn by other researchers. Cooper and Fairburn (1986) examined the frequency, nature, and severity of depressive symptoms in bulimic compared to depressed patients, and found the purported similarity of mental states to be only superficial. Anxiety and obsessional ruminations/ideas

were more frequent among bulimics, and, while severity of the symptoms was comparable between the two groups, there seemed to be qualitative differences. Often the symptoms were more ephemeral in the bulimics and seemed related to the eating disorder itself.

Indeed, the depressive symptoms of bulimia often seem to be different from those seen in primary depression (Russell, 1979; Katz et al., 1984). In particular, features associated with slowed or decreased behavior, such as slowed cognition, motor retardation, and motivational decreases, are typically not present. Anorexics often even exhibit behaviors directly opposite of these (Kron, Katz, & Gorzynski, 1978). Additionally, some depressive symptoms (e.g., autonomic abnormalities) may simply be the result of the starvation and/or dietetic alterations of the eating disorder (Katz et al., 1984), and others (most notably disturbed sleep) are extremely common in psychiatric disorders in general (Klein & Seibold, 1985).

Clinical evidence alone, then, is suggestive but hardly convincing of Pope and Hudson's theory.

Similarly, a second body of evidence concerned with the neuroendocrine abnormalities found in both bulimia and depression is not without its problems, but is helpful when interpreted as part of the gestalt.

Research into a neuroendocrine abnormality in bulimia has been primarily focused on determining hyperactivity of the hypothalamic-pituitary-adrenal axis. Similar to studies of this sort in depression research, the dexamethasone suppression test (DST) is commonly employed to investigate whether such an abnormality exists. Dexamethasone is a synthetic steroid that normally suppresses the release of cortisol into plasma by blocking the release of corticotropin-releasing factor from the hypothalamus and of ACTH from the anterior pituitary. Thus, if a patient is given dexamethasone and later found to have an elevated plasma concentration of cortisol, failure of normal cortisol suppression is indicated. The common interpretation of this, given the drug's normal course of action, is hyperactivity of the hypothalamic-pituitary-adrenal axis (APA Task Force, 1987).

Given that the DST, on average, manifests abnormal

results in approximately 50% of depressed patients (APA Task Force, 1987; Green & Kane, 1983), some researchers have investigated whether similar results can be seen in bulimics. The pioneering study in this area was conducted by Hudson et al. in 1983. Administering the DST to 47 bulimic patients and controls, they found 47% of the former and 9% of the latter to have abnormal results. Typically, only 4% of non-depressed normal subjects show abnormal results. High degrees of depressive symptomatology and familial affective disorder were also noted among the bulimics, but they unfortunately do not discuss any correlation of these measures with DST nonsuppression.

Similar but more extensive studies have been conducted by Gwirtsman, Roy-Byrne, Yager, & Gerner (1983) and Hughes, Wells, & Cunningham (1986). Rather than investigating only DST response, the former also administered thyrotropin releasing hormone (TRH) tests. Depressed patients normally show reduced or "blunted" thyrotropin responsivity to thyrotropin releasing hormone. This was the first time bulimics were studied with the TRH test, and dramatic results were obtained;

12 of 18 patients responded abnormally to the DST and 8 of 10 showed a blunted response to the TRH test.

Hughes et al. extended the literature by examining DST response before and after treating bulimic patients with desipramine, an antidepressant. Of their 23 subjects, 11 (approximately 50%) were nonsuppressors; this of course is similar to the percent of depressed patients who respond abnormally, and concurs with the results of the above studies. Additionally, however, 86% of the nonsuppressors before treatment became suppressors after treatment, and pre-treatment DST results were not predictive of treatment response among the patients. Both of these patterns, they note, are similar to those found in DST research with depressives.

These studies, then, appear to indicate that bulimia and depression may share a common neuroendocrine abnormality. Unfortunately, however, there are several problems that inhibit such a pronouncement. Both methodological problems and conflicting findings from other researchers provide for concern over the validity of the findings listed above.

Perhaps the largest problem with using the DST in bulimia is the susceptibility of the test to low and/or fluctuating weight (APA Task Force, 1987). Indeed, one of the less emphasized discoveries of the Gwirtsman et al. study was a correlation between the weight of the subjects and the level of cortisol in the plasma. Hudson, Katz et al. (1987) and Perez, Blouin, and Blouin (1988) investigated the influence of this factor and found results that were no doubt disturbing to proponents of the bulimia-depression relationship.

Using a measure of urinary free cortisol level, which is less susceptible to influence from weight fluctuations, the former group was unable to distinguish any significant difference between a sample of bulimics and normal controls. The latter group, using the DST and controlling for the weight of the subjects, made several interesting discoveries. Not only was suboptimal weight found to account for as much of the variance in DST results as depression was, but also a positive family history of depression in the bulimics was apparently unrelated to the test results. Further, severity of the bulimic symptoms was not found

to be a predictor of response to the DST. This is significant since the assumption that bulimia and depression are related implies that severe bulimia would lead to a greater chance of abnormal responding.

Aside from weight fluctuation, other problems can also be noted with regard to the neuroendocrine studies. Prolonged sleeplessness may bias DST results (Klein & Seibold, 1985), a potential concern when studying eating disordered patients (primarily anorexics), since they tend to exhibit sleep disruption (Walsh, Goetz, Roose, Fingeroth, & Glassman, 1985; Levy, Dixon, & Schmidt, 1988). Also, the existence of a psychiatric disturbance in addition to primary depression may bias DST results (APA Task Force, 1987), and the aforementioned studies of Hudson et al. (1983) and Gwirtsman et al. (1983) did not control for this. And lastly, bulimics, unless they are monitored very closely, may vomit and thereby expell the dexamethasone tablet from the body (Gwirtsman et al. 1983; Perez et al., 1988).

Thus, although the neuroendocrine studies are tempting, it would be presumptuous to draw any finite

conclusions from them. Methodological flaws and limitations, as well as conflicting findings, are proving to be quite a hindrance to their interpretation. However, even the most adamant critic cannot escape the possibility of a common neuroendocrine disturbance in bulimia and depression, given the recurrence of evidence within a wide variety of methodologies.

A third body of evidence often cited as supportive of a bulimia-depression link comes from studies of familial affective disorder among bulimics. The Boston group of Hudson, Laffer, and Pope (1982) once again conducted the ground-breaking study in this area, and the results since then have indeed proven enticing.

Through individual and family interviews of ten bulimics, Hudson et al. (1982) discovered that six had at least one first-degree relative with major affective disorder. This was not only seemingly impressive, it was also demonstrably so. The 60% figure was significantly higher than the 3% prevalence of affective disorder among first-degree relatives of schizophrenics, yet was not significantly different

from the 42% prevalence seen in a reference sample of families of bipolar patients.

Since the appearance of this study, further research has claimed similar findings. Although the reported frequencies vary, the ultimate conclusion is usually that bulimics display a high familial incidence of affective disorder. Herzog (1984) and Walsh et al. (1985) have noted this, as have Gwirtsman et al. (1983), who concluded that bulimic anorexics display twice the prevalence of familial primary affective disorder evident among non-bulimic anorexics. Also, Hudson et al. (1983) found 48% of their bulimic subjects to have at least one first-degree relative with major affective disorder.

Of course, these studies are not without their critics and contraindicative research. Hughes, Wells, Cunningham, and Ilstrup (1986), from a sample of 22 bulimics that were not depressed, found only 3 to have a first-degree relative with major depression. This surprising result raised the question of whether the samples employed in such family history studies as those discussed above were perhaps not representative.

Perhaps, as a result of using only clinical samples, only the more depressed bulimics were being investigated, since they would presumably be more likely to seek treatment.

Wilson and Lindholm (1987) studied this particular possibility and found confirmation of it. Controlling for the severity of depression among 50 bulimics (rated as none, mild, moderate, or severe depression), they compared family histories and discovered that a strong correlation (r=.63) existed between the two. Thus, the more severely depressed the subject was, the more likely she was to have a first-degree relative with depression. In addition, Wilson and Lindholm relate their observation of a methodological flaw inherent in some of the previous studies of family history. They note that the control groups of these studies often excluded any subjects with a family history of depression, a selection process which naturally biases the comparison toward showing more familial history among the bulimics.

These criticisms are intriguing, but may not be entirely accurate. Although the latter issue of a

selection bias may be a valid concern, the more vituperate criticism of a correlation between depression severity and family history is probably not. Hudson, Pope, Jonas, Yurgelun-Todd, and Frankenburg (1987) compared 69 bulimics, 24 depressed patients, and 28 nonpsychiatric controls on various indices, and their results did not support Wilson and Lindholm's assertion. Although the highest rates of familial affective disorder were found among the bulimics who themselves had major affective disorder, even the bulimics without major affective disorder displayed a positive familial history three times that of the normal controls. Thus, they both replicated and dismissed Wilson and Lindholm's criticism!

Family history data, then, lends stronger support than the other two bodies of evidence to the hypothesis of a bulimia-depression link. One last cluster of research and clinical findings, concerned with the results of treating bulimia with antidepressants, provides even further encouragement (and discouragement).

Since 1974, more than 30 reports have assessed the

treatment of bulimia with various medications, and the results have been mixed. Pope & Hudson (1986) have summarized the current status of this research as follows. A total of nine placebo-controlled, double-blind studies have been conducted, five with antidepressants and four with other medications believed to have some thymoleptic properties. Although five of the studies did not demonstrate impressive results, the other four showed a "clear superiority" of drug over placebo (p. 343). Of these four, three used tricyclic antidepressants (imipramine, desipramimine, and amitriptyline) and one used a monoamine oxidase (MAO) inhibitor (phenelzine). Various uncontrolled studies have been reported, most with no better than moderate results.

Thus, the findings of significant effectiveness of some antidepressants is encouraging, but their implications and the similarities that can be drawn between antidepressant use for bulimia and for depression are even more intriguing. Pope and Hudson (1986; 1988) discuss five of these similarities and implications: (1) the dosage and plasma levels of

medications required for effectiveness are the same; (2) the time lapse between initiation of treatment and symptomatic response is similar; (3) equal amounts of bulimic and depressed patients (80-90%) appear to respond to the antidepressants; (4) bulimia responds to the entire spectrum of thymoleptics, some of which have little in common chemically, but does not seem to respond to any non-thymoleptics; (5) both overt bulimic behavior and the associated psychopathology appear to improve. This last observation, they claim, discredits the view of bulimia as distinct from depression, since the multifactorial etiology implied by that view would lead one not to expect such a universally therapeutic response.

These assertions, impressive as they are, must nonetheless be accepted only with speculation. One must first realize that they are for the most part broad generalizations that are based on a very limited number of studies. Although the studies are certainly suggestive, they should probably not be generalized in the manner above, and cannot be regarded as absolute evidence that bulimia is a variant form of depression.

This is especially true when one considers the arguments of opposing research.

First, as Herzog (1984) has commented, "antidepressants may be effective in the short term (for bulimia), but the long-term efficacy of these drugs has not been clearly demonstrated" (p. 1595). The same studies Pope and Hudson cited in the above discussion are the ones Herzog refers to as short term. In addition, these studies have been referred to as "seriously limited", due to their failure to exclude from their samples patients with concurrent major depression (Hughes, Wells, Cunningham, and Ilstrup, 1986, p. 182).

Second, as Walsh et al. (1985) have noted, one must not assume bulimia to be a form of depression simply because both disorders respond similarly to antidepressants. They provide the example of cardiac arrhythmia as a disorder that is also suppressed by antidepressants, yet no one suggests cardiac irritability to be a form of affective disorder. Their conclusion is that "it is premature at this point simply to reduce bulimia to a form of affective

illness" (p. 129).

Third, depression seems to be reduced when cognitive-behavioral therapy improves bulimia. Cooper and Fairburn (1986) note their observation that, for the majority of their patients, when control over eating is gained, the coexisting symptoms of depression and anxiety simply disappear. This same observation has been set forth by Wilson and Lindholm (1987), and would not be expected if bulimia stems from depression.

Last, but certainly not least, are more practical concerns regarding antidepressant treatment. Tricyclics such as imipramine, desipramine, and amitriptyline are widely recognized as having significant weight gain as a frequent side effect (Crane et al., 1987; Wilson & Lindholm, 1987), and MAO inhibitors such as phenelzine require rigid dietary restrictions (Hughes, Wells, & Cunningham, 1986). For the treatment of depression such side effects may be of little relevance, but in the treatment of bulimia they could be devastating. Considering Herzog's (1984) comments discussed above, a long-term study of the results of treatment with tricyclics might reveal quite

a large relapse rate, due to these side effects, after patients are discharged into their own care. Additionally, MAO inhibitors may cause hypertensive crisis during binging, a potential problem not to be ignored in the prescription of such drugs to bulimics (Crane et al., 1987). There is some evidence that "second generation" antidepressants, notably nomifensine, which do not produce the side effects listed above, may be effective in treating bulimia, but more research is needed (Crane et al., 1987), and nomifensine is no longer available.

Overall, then, the existing body of research into the question of a bulimia-depression link is suggestive but far from convincing. Certainly it is an avenue worth pursuing, as many are doing, but any conclusions from the literature would at present be premature. Having introduced the problem as such, it is now essential to examine research on a type of antidepressant therapy that, previous to the present study, has been alien to the bulimia-depression work.

In 1966, Schulte described a female teacher who had related to him that, when she was depressed

(clinically), she found her condition easiest to bear if she stayed awake all night. Despite the paradox of the possibility that a night's deprivation of sleep could alleviate depressive symptoms when one of the most common symptoms is sleep disturbance, Schulte's report eventually led to studies that showed that this could indeed occur (Pflug, 1976). Since then, research in the area has burgeoned, although as a clinical tool it seems to be more popular in Europe than the United States.

The first studies, and in fact those occurring today as well, attempted to determine whether the phenomenon was real and, if it was, what its limitations and applications were. Certainly the effect is real, as virtually every study conducted has witnessed it (Schilgen & Tolle, 1980); however, there has been some variation as to the degree of depression reduction and the percentage of subjects who respond (Schilgen & Tolle, 1980; Gerner, Post, Gillin, & Bunney, 1979).

Apparently, the effect of sleep deprivation on depression is a universal one, in the sense that its

influence is not restricted by the age (King, Baxter, Stuber, & Fish, 1987) or gender (Pflug, 1976) of the patient. Not all patients respond to the treatment, however, as noted above. From the summary of sleep deprivation experiments conducted through 1979 and provided by Gerner et al. (1979), improvement seems to occur in 50-65% of the patients. This summary includes studies of varying types of depression, using varying methods of assessing response as well as of rating improvement, and also includes studies that used patients who were taking antidepressant medication during the experiment. The lowest rate of response was approximately 30%, whereas the highest was approximately 75%, but most studies witnessed a 50-65% rate.

Although endogenous depressives appear to fare better than any others in response to sleep deprivation, all types of depression can potentially be affected (Gerner et al., 1979; Pflug, 1976; Svendsen, 1976). This includes depression that is concurrent with another psychiatric disorder (King et al., 1987; Holsboer-Trachsler & Ernst, 1986; Holsboer-Trachsler,

Wiedemann, & Holsboer, 1988). A depressed state of some severity is essential, however; sleep deprivation does not effect normal, depression-free subjects in any significant manner (Gerner et al., 1979; Klein & Seibold, 1985; Roy-Byrne, Uhde, & Post, 1986). It also has been shown to be specific in its effect, significantly alleviating no psychiatric disturbances other than depression (Roy-Byrne et al., 1986; Holsboer-Trachsler & Ernst, 1986).

The extent of the improvement in a responder's depressed symptomatology is variable, but can occasionally be a complete remission of the depression (Svendsen, 1976; Holsboer-Trachsler & Ernst, 1986; Holsboer-Trachsler et al., 1988). The occurence of such a response is admittedly rare, however. Often when it does occur it is as a result of repeated sleep deprivation, most likely "partial" sleep deprivation, which consists of keeping the patient awake for only the second half of the night (Holsboer-Trachsler & Ernst, 1986; Holsboer-Trachsler et al., 1988). Partial sleep deprivation was introduced by Schilgen and Tolle (1980), and interestingly has at least as potent an

effect, if not more potent, than that of total sleep deprivation. The technique was borne out of consistent reports from patients that the reversal of their depression began just after 2:00 a.m. (Schilgen & Tolle, 1980).

Given the above description, the reader must certainly be perplexed as to why sleep deprivation has not attained international status as a panacea for depression. The answer to this lies in the one unfortunate aspect of sleep deprivation, namely the transient nature of the symptomatic reduction. Indeed, the depression usually relapses within 24 hours (King et al., 1987; Wiegand, Berger, Zulley, Laure, & von Zerssen, 1987) although, as noted above, it occasionally can dissipate into total remission. Pflug (1976) has suggested that the combination of thymoleptic medications with sleep deprivation may help to prevent relapse, but this has not been consistently demonstrated.

In addition to the above limitation, clinicians must closely monitor sleep-deprived patients, as even a short nap during the day following treatment can result

in elimination of any potentially beneficial effects, and may even lead to depression worse than that of the pretreatment state (Wiegand et al., 1987; Gerner et al., 1979; Holsboer-Trachsler et al., 1988). Normally, if no naps are taken, worsening of the depression after sleep deprivation is extremely rare (Pflug, 1976).

No one is entirely certain why sleep deprivation works, but the most commonly proposed explanation is that it serves to resynchronize a disrupted circadian rhythm. As noted earlier, sleep disturbance is common among depressed patients, with the basic dysfunction being an inability to stay asleep for an entire night (Hudson, Pope, Jonas, Stakes et al., 1987; Feinberg, Gillian, Carroll, Greder, & Zis, 1982; Katz et al., 1984). Other disturbances of the circadian cycle have also been noted, including disruption of the levels of several neurochemicals, the pattern of salivary secretion, and the patient's self-report of diurnal mood variation and sense of time (Pflug, 1976). As Svendsen (1976) has noted, however, this explanation is daunted somewhat by the observation that many people are able to adapt their circadian rhythm for late-night

shift work, without experiencing any significant problems.

But despite the lack of understanding regarding the mechanism of action in sleep deprivation, the crucial fact is that it clearly exerts a pronounced effect. This has profound implications for understanding the bulimia-depression relationship. Given the proven influence sleep deprivation has on depressive symptomatology, one can hypothesize that if bulimia is in fact a variant form of depression, it should also respond to the treatment by displaying a reduction in associated behavior and psychopathology. If, as Pope and Hudson (1988) assert, there is a common biological abnormality responsible for the appearance of depression and bulimia, then any intervention that affects one disorder should also affect the other. Indeed, this is the same logic Pope and Hudson use in their interpretation of findings from the treatment of bulimia with antidepressants.

The present study was designed to investigate the above implications, and attempted to accomplish this by treating bulimic subjects with partial sleep

deprivation. The subjects were recruited from a college population based upon scores obtained on measures of depression and bulimia, and were then divided according to the degree of depressive and bulimic symptomatology. The resulting four groups were labelled depressed bulimics, nondepressed bulimics, depressed nonbulimics, and nondepressed nonbulimics. Members of each group gathered for two experimental sessions, one involving partial sleep deprivation and the other involving no sleep deprivation, and thus serving as a control session. At specified times both before and after each session, subjects self-reported their current symptomatic state (for bulimia and depression) using questionnaires specifically chosen for their sensitivity to change in the subject.

Based on the accumulation of research discussed throughout this paper, it was hypothesized that, if bulimia is a form of depression, subjects in bulimic samples should demonstrate a significant reduction in their bulimic psychopathology following partial sleep deprivation. Additionally, the depressed subjects should report a reduction in their depressive

psychopathology. Lastly, no significant reduction in psychopathology related to bulimia or depression was expected to be observed in any group after the control session.

Method

Subjects

Subjects were 27 females (mean age=18.54; range 18-21) obtained from introductory psychology courses at the College of William and Mary. An additional subject began the study but was unable to finish; her data is not included in any of the analyses. Only females were used, as controversy currently exists regarding the similarity of bulimia among males and females (see Scott, 1986, for a review of this issue). All subjects volunteered, although their participation fulfilled a course requirement of involvement in research; each was paid a stipend of $15.00.

Subjects were selected solely on the basis of scores received on the Bulimia Test (BULIT; Smith & Thelen, 1984), a measure commonly used to assess bulimic symptomatology, and the short version of the Beck Depression Inventory (BDI; Beck, 1967). On the

latter, item #9 ("I don't have any thoughts of killing myself") was omitted due to the restrictions of an ethics committee. Smith and Thelen (1984) have suggested a score of 88 as the lower limit of scores indicative of subclinical bulimia, with 102 being representative of clinically severe bulimia. Beck (1967) recommends an upper limit of a score of 10 for a normal population on the BDI; this is, however, for the long form and not for the short version used in the present study. DSM-III-R (American Psychiatric Association, 1987) diagnostic criteria were not employed in the selection of subjects.

Thus, individuals with appropriate scores on the BULIT and BDI were chosen in order to ultimately form four groups: 1) "depressed bulimics"; 2) "nondepressed bulimics"; 3) "depressed nonbulimics"; 4) "nondepressed nonbulimics" (controls). Group characteristics are discussed under "Methodology Checks" in the Results section.

Materials

As noted above, the BDI short-version and the BULIT were employed for prescreening purposes. On the

former, item #9 was omitted due to the restrictions of an ethics committee.

During the experimental sessions, the dependent variables of bulimic and depressive psychopathology were assessed using a total of three measures. The Drive for Thinness, Bulimia, and Body Dissatisfaction scales of the Eating Disorders Inventory (Garner et al., 1986) were used for the former, while an adjective checklist for depression and 7-point Likert scale (ranging from "extremely happy and optimistic" to "extremely sad and depressed") were used for the latter (see Appendix A). Subjects were also asked the question "When do you expect to begin your next menstruation?", in order to control for the possibility of premenstrual bulimic exacerbation (Gladis & Walsh, 1987).

The three EDI scales used have been identified as the ones most effective in distinguishing bulimics and bulimic anorexics from primary anorexics and normal controls (Garner et al., 1983). Most of the questions were modified slightly, in order for them to be more sensitive to change occurring over a short period of

time. The adjective checklist and Likert scale were selected after a review of the currently available depression measures revealed none to be appropriate for the present experimental design.

To control for the possibility of subjects responding dishonestly to the questionnaires, the Marlowe-Crowne social desirability scale (Crowne & Marlowe, 1960) was administered during each assessment. Such a measure was deemed especially important due to the sensitive nature of the questions being asked, although Wilson (1987) has asserted that bulimics usually respond honestly on such questionnaires.

The setting of each session was one of three rooms, all of which were similar in the sense that they were quiet and fairly small. In each instance, one room was available for subjects wishing to talk or watch movies, and at least one other was available for those wishing to study or read. A television, videocassette recorder, and three movies, as well as caffeine-free sodas, were provided for each session. No food was provided.

Procedure

Following the identification of potential subjects, each was contacted by the experimenter over the phone. The requirements of and rewards for participation in the experiment were explained, and any who were then willing were scheduled for one night and one day session, the latter serving as a control (no sleep deprivation). This process was continued until 28 subjects were obtained.

Approximately half of the subjects (n=15) experienced the control session first, while the other half experienced the sleep deprivation first. Assignment of order was done on the basis of the subject's scheduling convenience. All of the trials were virtually identical, the only notable differences being the times before and after the trial that subjects were asked to complete the depression and bulimia self-report measures, and, obviously, the time of day the trials were conducted.

The exact starting times of the control sessions varied due to the scheduling demands of the subjects, with weekday sessions generally beginning later than those on the weekends. Each did start, however, at the

time of 12:00, 1:00, or 2:00 p.m., and thus all took place during the same basic period of the day.

At the outset of each session, subjects were assembled and handed a numbered envelope (for confidentiality/identification) that contained a consent form and the questionnaires noted above. After all had read and signed the consent form, the experimenter reiterated the basic "rules" for the session (see below), emphasized the confidentiality of the data, and answered any questions. The true purpose of the study was not divulged at any time. Subjects then completed the questionnaires and returned them in the envelope to the experimenter.

Following this initial period of approximately 20 minutes, subjects were encouraged to occupy themselves in any of the available manners. Napping was of course not allowed, for reasons elaborated earlier in this paper. A total of 30 minutes was allowed outside of the lab, e.g., to go on a walk, and this time was monitored by the experimenter. Aside from this requirement, however, the activities of the subjects were not strictly monitored, as no relation exists

between such activities and response to sleep deprivation (Svendsen, 1976).

After five consecutive hours, the subjects were given a new set of the questionnaires (in the same envelope) to take with them. They were instructed to complete the measures approximately five hours later, without napping during this intervening time. It was emphasized, however, that completing the questionnaires prematurely was preferable to completing them after a nap; thus, those who could not abstain from napping completed their questionnaires before doing so. The time of completion of each measure was always recorded by the subjects.

Sleep deprivation trials were conducted in the same manner as described above, excepting the time frame. As partial rather than total sleep deprivation was the technique employed, subjects were asked to assemble in the lab at 2:00 a.m. Most studies of partial sleep deprivation have used 2:00 a.m. as the starting time (Schilgen & Tolle, 1980; Holsboer-Trachsler, Weidemann, & Holsboer, 1988; King, et al., 1987). All subjects were offered an escort by the

experimenter, though some declined the offer or were accompanied by a friend to the lab.

At 7:00 a.m. (five hours after beginning) the session was terminated, and the self-report measures were distributed. The subjects were asked to complete them at approximately 12:00 p.m., and the same instructions explained above regarding napping were given.

After completing both sessions, subjects returned the final self-report measures by mail and upon receipt of them the $15.00 stipend was distributed. Once all of the data were collected, each subject was debriefed and given the opportunity to ask questions about the experiment.

Results

A Priori Analyses

Methodology checks. For the following discussion, the group names are abbreviated: depressed bulimic (DB); nondepressed bulimic (NDB); nonbulimic depressed (NBD); nonbulimic nondepressed (CTRL).

Two-way ANOVAs were conducted to assess differences between the groups on a number of

independent measures. Age was first analyzed, and no significant differences were found (group *M* and *SD*, respectively: DB=19, 1.2; NDB=18.3, .5; NBD=18.5, .8; CTRL=18.4, .5).

Second, analysis of BULIT scores revealed a significant difference, $F(1, 23)=98.1$, $p<.05$, between the bulimic and nonbulimic groups, but not between any others (see Table 1). In addition, inspection of the means reveals that, while clinical severity cannot be formally claimed, a high degree of bulimic psychopathology did exist among the bulimic groups.

BDI scores were significantly higher, $F(1, 23)=46.78$, $p<.05$, among the depressed versus the nondepressed groups, but there was also a significant difference, $F(1, 23)=4.64$, $p<.05$, between bulimic groups (see Table 1). Thus, bulimics were more depressed than nonbulimics. Additionally, as above, inspection of the means reveals fairly high scores among the depressed groups.

Lastly, the date of the day (control) session was subtracted from the date of the night session to obtain an index of the time (in days) intervening between the

two. Analysis of this variable did reveal a significant interaction between the depressed and bulimic groups, $F(1, 23)=6.37$, $p<.05$ (see Table 1).

Insert Table 1 about here

To evaluate the construct validity of the dependent measures, a series of Pearson correlations was conducted. Pre-treatment scores (from both the day and night sessions), BULIT, and BDI scores were used in the analyses. The adjective checklist was scored by summing the number of positive and negative items endorsed, then subtracting the latter from the former. Thus, the resulting difference was an index of positive, rather than of depressed, mood.

Neither the day session nor night session pre-scores on the 7-point depression scale correlated strongly with the BDI ($r=.18$ and $.07$, respectively). Similarly, no correlation was found between day or night adjective checklist pre-scores and the BDI ($r=-.26$ and $-.13$). A strong negative correlation was obtained, however, between both day and night pre-

scores on the depression scale and adjective checklist (r=-.88 and -.84). Lastly, the day and night session pre-scores on the EDI both correlated strongly with the BULIT (r=.79 and .80).

Given the above results, further analyses of the dependent measures were deemed necessary. One-way ANOVAs were used to test for differences between groups' scores at the outsets of the sessions. Significant differences were found in the EDI scores of bulimics versus non-bulimics for both sessions, $F(1, 23)=32.56$, $p<.01$ (day) and $F(1, 23)=34.29$, $p<.01$ (night), and in the EDI scores of depressed versus non-depressed subjects for the day session, $F(1, 23)=5.4$, $p<.05$. Means and standard deviations are illustrated in Table 2. Thus, among the putative groups, bulimics

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Insert Table 2 about here

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scored consistently higher than non-bulimics, but depressed subjects also showed higher scores than non-depressed subjects before the day session. No other significant differences were found on any measure for

either session.

Main analyses. Two Pearson correlations were first conducted to investigate the possible relationship between the premenstrual period and exacerbated bulimic symptomatology. For both, the number of days until menstruation (as estimated by the subjects at the time of each session) was correlated with the session's EDI pre-scores. No strong relationship was found at either the day session (r=-.12) or the night session (r=-.05). A t-test was also conducted on the EDI pre-scores of the session closest to versus farthest from menstruation, and the difference between the two was not found to be significant.

For the central analyses, two-way ANOVAs with repeated measures were used on each dependent variable, with the change scores for the day and night sessions serving as the repeated measures. No significant differences were obtained on any of the ANOVAs. A chi-square was then conducted for each dependent measure to compare the number of subjects improving after each session to the number not improving. Again, no

significant differences were discovered.

One- and two-way ANCOVAs were employed, using three covariates: (a) the time elapsed between day and night sessions; (b) and (c) the time elapsed between leaving the lab and completing the questionnaires (for each session). No significant differences emerged.

A series of t-tests was then conducted to assess the change on dependent measure scores after the day and night sessions for the subjects as a whole (i.e., not divided into groups). When compared in this manner, a significant decrease in depression scale scores was evident after the night session (M=-.5, SD=1, p<.05), but not after the day.

Given this latter finding, the results of the methodology checks, and the ambiguous nature of the central analyses, the subjects were regrouped according to their scores on the BULIT and the adjective checklist pre-scores. The assumption was that the BDI did not adequately separate the groups with respect to depression.

The BULIT was used for regrouping because it appeared (from the results above) to be an adequate

selection measure, and the checklist was used because it allowed for more variability than the depression scale. Pre-scores of the latter were used, since they likely were most representative of the subjects' normal state; further, because the day and night session pre-scores were weakly correlated (r=.33), a regrouping and analysis was done according to each. Group assignments of every subject are listed below in Table 3.

Insert Table 3 about here

Grouping By Day Session Pre-Scores

Methodology checks. Day and night session pre-scores for the checklist were not strongly correlated (r=.33), and neither were those for the depression scale (r=.27). Those for the EDI and Marlowe-Crowne were, however (r=.93 for the former and .84 for the latter). These correlations, of course, are relevant to both this grouping and the next. Means and standard deviations are given below.

One-way ANOVAs revealed no significant differences between groups with respect to age. There were

significant differences in the time elapsed between day and night sessions for the groups, $F(1, 23)=9.75$, $p<.01$ (M, SD: DB=5.6, 14.1; NDB=-12.3, 13.8; NBD=-10.3, 14.6; CTRL=1.3, 2.4), but this variable did not affect any of the results when used as a covariate. The BDI scores of the depressed groups were significantly higher than those of the nondepressed groups, $F(1, 23)=5.29$, $p<.05$, and BULIT scores were significantly higher among bulimic than among nonbulimic groups, $F(1, 23)=98.7$, $p<.01$. Table 4 contains these means and standard deviations.

Insert Table 4 about here

One-way ANOVAs were also used to examine differentiation among the groups on the dependent measures (see Table 5 for means and standard deviations). At the outset of both the day and night sessions, the bulimic groups scored significantly higher than the nonbulimics on the EDI (day: $F(1, 23)=37.64$, $p<.01$; night: $F(1, 23)=35.98$, $p<.01$). At the outset of the day session, the depressed groups

also scored significantly higher on the EDI than the nondepressed groups, $F(1, 23)=6.80$, $p<.05$, but this difference did not exist for the night session pre-scores.

Depressed groups scored significantly lower than the nondepressed groups on the adjective checklist at the beginning of the day session, $F(1, 23)=29.31$, $p<.01$, but not at the beginning of the night (though this difference approached significance). This same pattern was seen with the depression scale scores, as depressed groups reported significantly more depression at the outset of the day session, $F(1, 23)=9.60$, $p<.01$, but not at the outset of the night.

Insert Table 5 about here

Main analyses. As before, two-way ANOVAs with repeated measures were used to examine changes in dependent measure scores among the groups. Change scores from the day and night sessions were used as the repeated measures. No significant differences were found between or within groups on the EDI. Analysis of

Marlowe-Crowne scores did reveal a significant difference, as the responses of the NDB group reflected less of a social desirability influence than any other groups' after the night session, $F(1, 23)=5.9$, $p<.05$ (mean change and SD after night: DB=0, 3.3; NDB=-5.7, 6.9; NBD=-1, 1.7; CTRL=.3, .95). Controlling for this factor in an ANCOVA, however, did not impact significantly on any other results.

In examining changes in the depressed state of the subjects, highly similar patterns emerged on the adjective checklist and depression scale. These changes are illustrated below in Figures 1 and 2. On

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Insert Figures 1 and 2 about here

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the checklist, the nondepressed groups demonstrated significantly more change after the night session than the depressed groups, $F(1, 23)=5.35$, $p<.05$. While the depression of the latter remained the same or improved slightly, that of the former worsened significantly. There was also a significant interaction between bulimia and depression, such that the NDB group

reported a greater increase in depression than any other group, $F(1, 23)=6.16$, $p<.05$.

Analysis of the depression scale, also illustrated in Figures 1 and 2, yielded comparable results. Nondepressed subjects were more affected by the sleep deprivation than depressed subjects, showing significant increases in scores on the scale, $F(1, 23)=8.14$, $p<.01$. A significant interaction was again present between bulimia and depression, with the NDB group changing significantly more (becoming more depressed) than the DB group (whose scores remained about the same or decreased) after both the day and night sessions, $F(1, 23)=9.31$, $p<.01$.

Grouping By Night Session Pre-Scores

Methodology checks. One-way ANOVAs revealed that the new groups were not significantly different with respect to age, number of days intervening between sessions, or BDI scores. BULIT scores, however, were significantly higher among the bulimic groups than among the nonbulimics, $F(1, 23)=103.44$, $p<.01$ (M, SD: DB=101.9, 15.7; NDB=97, 7.4; NBD=58.7, 12; CTRL=52.9, 7.2).

One-way ANOVAs also were used to evaluate group differentiation by means of dependent measure pre-scores (means and standard deviations listed in Table 6). At both the day and night sessions, bulimic groups scored significantly higher on the EDI than nonbulimics (day: $F(1, 23)=27.8$, $p<.01$; night: $F(1, 23)=33.4$, $p<.01$).

On the adjective checklist, depressed groups scored significantly lower (indicating greater depression) than nondepressed groups at the outset of the night sessions, $F(1, 23)=35.11$, $p<.01$, but this was not true at the outset of the day session. Similarly, scores on the depression scale were significantly different between depressed and nondepressed groups at the night session's beginning, $F(1, 23)=18$, $p<.01$, but not at the day session's.

Insert Table 6 about here

Main analyses. As with previous groupings, two-way ANOVAs with repeated measures were employed to assess change on the dependent measures. The change

scores on the questionnaires from the day and night sessions were again used as the repeated measures. Analysis of both Marlowe-Crowne and EDI scores revealed no significant differences between or within groups on either measure.

The adjective checklist and depression scale revealed significant differences that were highly similar to those discovered with the previous grouping procedure. Regarding the former, sleep deprivation had a significant effect on the nondepressed subjects, making their scores significantly lower (indicating greater depression) after the night than after the day, $F(1, 23)=7.16$, $p<.01$. These means also reveal that, although the scores of the depressed subjects did not significantly change, they did consistently improve (see Table 7).

On the depression scale, sleep deprivation again produced a change in the nondepressed subjects, making their scores significantly higher, $F(1, 23)=4.58$, $p<.05$. The means also reveal no worsening of depression among the depressed subjects (see Table 7 below).

Insert Table 7 about here

Discussion

The analyses of central interest to the present purpose initially indicated that the original grouping of the subjects into depressed and nondepressed categories was not accurate. There were clear divisions between bulimic and nonbulimic groups with respect to BULIT and pre-session EDI scores, but depressed groups could not be differentiated from nondepressed groups on the pre-session adjective checklist or depression scale scores.

The most likely explanation for this lies in the large amount of time elapsed between completion of the BDI and participation in the study. Though the groups were divided well by their BDI scores, the extent of their depression by the time of the study (for some as many as three months later) was apparently very different. This was evident in the high correlation between the adjective checklist and depression scale, together with the weak correlation between these

measures and the BDI. Seasonal shifts and the moderate, rather than severe, nature of the subjects' depression, may both have played a role in this change.

Given this poor division of subjects, the initial findings of no significant changes among the groups were not surprising. Nonetheless, the high variability in both dependent measures of depression clearly indicated that subjects were different, and thus was the impetus for reformulating the groups.

Both the day session and night session pre-score grouping procedures created well differentiated groups (especially the day session procedure, which produced significant differences between the BDI scores of the depressed and nondepressed groups). Once regrouped, some highly consistent and significant changes were observed, with both procedures producing basically the same changes.

The results are not supportive of the hypothesis that bulimia is a form of affective disorder. After no session did any groups display even a tendency toward change in their bulimic symptoms, as self-reported on the EDI, yet there were significant changes in the

depressive symptoms of subjects. Sleep deprivation, but not the control session, clearly produced increased depression among the nondepressed subjects, yet the depressed subjects remained the same or improved slightly.

Although this is not the same effect of depression alleviation that has been reported by other authors, it is nonetheless important, since only those in a depressed state were shielded from the negative repercussions of the sleep deprivation. If nonbulimic subjects had exhibited increased scores on the EDI after the sleep deprivation, one could reasonably argue that bulimia and depression were not differentially affected by the procedure. However, as this was not the case, the correct inference must be that they were differentially affected.

This interpretation is further supported by the commonly noted finding that mild depression does not respond dramatically to sleep deprivation, as severe depression does (Gerner et al., 1979; Schligen & Tolle, 1980). Modest improvements in comparison to controls are typically seen, and indeed this is what was found

in the present study.

These results seem to concur with the assertion of some (Wiegand et al., 1987) that sleep has a depressiogenic effect for depressed persons. This model predicts that missing a night of sleep prevents the worsening of depression among these persons, but not among normals. The present results support this prediction.

Given the significant response of depression to the sleep deprivation, the immobility of the bulimic symptoms is interesting. As noted above, it does not support the hypothesis that bulimia is a form of depression. The strong relationship between the two disorders, however, was very apparent in the significant interactions obtained with the depression measures. Although the controls and the nondepressed bulimics both showed significant increases in depression following sleep deprivation, the increases of the latter were consistently greater than those of the former.

Perhaps a state of nondepression is very unstable for bulimics, needing only a slight disruption to

reverse it into a depressed state. Certainly the dysfunctional cognitive styles of bulimics (Hood et al., 1982) lend themselves to such a characteristic. A binge-purge episode, for example, is frequently triggered by a minute departure of the bulimic from his/her strict dietary regimen. When such a departure occurs, it is overgeneralized as representing a total lack of self-control, and thus it leads to despair. This is very similar to the rapid degeneration of positive mood witnessed presently in the nondepressed bulimics.

This change among the nondepressed bulimics is of further interest because while their depression increased significantly, their bulimic symptoms did not. Widespread controversy currently exists over whether bulimic episodes lead to depression, or vice-versa, and the present findings strongly suggest the former. If increases in depression lead to bulimic exacerbation, then the nondepressed bulimics should have demonstrated that exacerbation after the sleep deprivation. They did not, and thus this finding supports the view that bulimia does not follow

depression (Cooper & Fairburn, 1986; Wilson & Lindholm, 1987).

Related to this discussion is the possible argument that the EDI was not adequately reformulated for measuring short-term changes. This seems unlikely, since it did occasionally register such changes. In the first regrouping, for example, the EDI scores were significantly higher among the depressed versus the nondepressed groups at the time of the day session, but not at the time of the night session. Given this, the most plausible interpretation of the findings is not that the EDI was inadequate, but rather that bulimia simply did not respond to sleep deprivation as depression did.

Aside from the inference that the two disorders are separate in nature, an explanation for this difference might be found in research on the sleep architecture of bulimics. Most of this work has demonstrated that the sleep architecture of bulimics is dissimilar from that of depressed patients and strikingly similar to that of normal controls. In general, depressed patients tend to exhibit sleep

abnormalities, most notably sleep continuity disturbance, decreased REM latency, and increased REM density (increased eye movement with no concurrent increase in the length of the REM period) (Dippel et al., 1987; Katz et al., 1984; Feinberg et al., 1982). Although two studies have found some of these characteristics in the sleep of bulimics (Katz et al., 1984; Neil et al., 1980), the majority have not (Walsh et al., 1985; Levy, Dixon, & Schmidt, 1988; Hudson, Pope, Jonas, Stakes et al., 1987; Byrne, Nino-Murcia, Gaddy, Doghramji, Keenan, 1989; Levy, Dixon, & Schmidt, 1987). Even the Boston group led by Pope and Hudson concede that "...bulimia may be dissimilar to major affective disorder on this biological test" (Hudson, Pope, Jonas, Stakes et al., 1987, p. 826).

Considering this factor, then, perhaps a difference in sleep architecture between bulimics and depressives allows one, but not the other, to respond to sleep deprivation. This is certainly possible; however, it is probably not accurate. Although the sleep architecture of bulimics may not be dissimilar from that of normal controls, an important

consideration is that people suffering from nonendogenous depression may also exhibit no abnormal sleep (Feinberg et al., 1982; Kupfer et al., 1978). Nonendogenous depression does, however, respond reliably to sleep deprivation (as it did presently); subsequently, if bulimia is truly a form of affective disorder, then it too should have been affected by the treatment.

The results of the present study are clear, but at best only suggestive. Further research needs to be conducted using subjects with clinically diagnosed disorders, and under rigorously controlled conditions, to elicit the maximal effect of the sleep deprivation. The BULIT scores of the present subjects were high, with many exceeding the recommended cut-off for clinical severity, but there was no means of knowing how many (if any) would have met DSM-III-R criteria for bulimia. Similarly, given the time lag between subject selection and participation, the BDI scores were virtually useless for identifying the extent of depression in the subjects.

An issue of secondary interest to the present

purpose was the extent to which bulimic symptomatology was influenced by the premenstrual phase of the menstrual cycle. Gladis and Walsh (1987) have reported a modest, but significant, exacerbation of bulimic symptoms during this time, whereas Leon et al. (1986) did not find any such exacerbation. The present results support the latter group of authors and suggest that, if anything, the premenstrual phase decreases bulimic symptomatology. A possible problem, however, is that few subjects (n=3 for the day and 2 for the night) were five days or less from menstruation.

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Appendix A

Date_____ Time_____ Number_____

Please place a check beside each adjective that describes the way you have been feeling **DURING THE LAST SEVERAL HOURS, INCLUDING NOW.** Leave blank any that do not apply to you now.

wanted____+
hopeful_____+
torn-up_____-
rejected_____-
pleased_____+
tense_____-
lonely_____-
secure_____+
confused_____-
mad_____-
friendly_____+
smart_____+
bitter_____-
shy_____-
courageous_____+
happy_____+
unloved_____-
content_____+
unhappy_____-
loved_____+
annoyed_____-
important_____+
liked_____+
trapped_____-
gloomy_____-
peaceful_____+
worthless_____-
bashful_____-
elated_____+
proud_____+
suspicious_____-
good_____+
infuriated_____-
capable_____+
humiliated_____-
worthy_____+
optimistic_____+
sad_____-
enthusiastic_____+
hopeless_____-

When do you expect to begin your next menstruation?_____

Please rate on the scale below, from 1 to 7, where you would presently rate yourself with respect to the two poles.

1	2	3	4	5	6	7
extremely happy and optimistic						extremely sad and depressed

Note. Plus signs indicate positively keyed items, and minus signs indicate negatively keyed items.

Table 1

Differentiation Between Characteristics of BDI-Produced Groups

	Group			
Characteristic	DB	NDB	NBD	CTRL
BULIT scores				
Mean	99.1	99.7	56.7	54.6
SD	14.1	10.8	13.9	5.3
BDI scores				
Mean	11.8	6.6	11.0	3.3
SD	3.1	2.6	2.1	1.7
Day elapsed				
Mean	2.3	-9.0	-11.7	2.4
SD	15.8	15.9	13.3	.5

Note. A negative number of days elapsed indicates that the night session was experienced first.

Table 2

Average EDI Pre-Scores of BDI-Produced Groups

	Group			
Session	DB	NDB	NBD	CTRL
Day				
Mean	43.3	33.4	19.2	9.4
SD	8.3	13.1	13.8	7.6
Night				
Mean	37.6	37.6	16.2	9.0
SD	4.3	11.5	14.7	11.8

Table 3

Various Group Assignments of Subjects

S	BUL	BDI	BDI Grp	Day	Day Grp	Night	Night Grp
1	93	9.5	DB	2	DB	0	NDB
2	100	15	DB	2	DB	13	NDB
3	85	7	DB	12	NDB	8	NDB
4	84	13	DB	8	DB	-6	DB
5	125	16	DB	-2	DB	1	DB
6	107	11	DB	-4	DB	-4	DB
7	100	11	DB	11	NDB	16	NDB
8	109	8	NDB	17	NDB	15	NDB
9	86	5	NDB	12	NDB	0	DB
10	105	7	NDB	-9	DB	-10	DB
11	98	7	NDB	10	NDB	7	NDB
12	116	11	NDB	11	NDB	-5	DB
13	94	3	NDB	10	NDB	7	NDB
14	90	5	NDB	6	DB	1	DB
15	63	11	NBD	-1	NBD	4	CTRL

(table continues)

S	BUL	BDI	BDI Grp	Day	Day Grp	Night	Night Grp
16	43	9	NBD	7	CTRL	2	NBD
17	46	10	NBD	-5	NBD	10	CTRL
18	78	14	NBD	-1	NBD	-6	NBD
19	46	9	NBD	1	NBD	4	CTRL
20	64	13	NBD	12	CTRL	2	NBD
21	59	2	CTRL	5	CTRL	9	CTRL
22	47	6	CTRL	3	NBD	4	CTRL
23	61	3	CTRL	-8	NBD	2	NBD
24	52	5	CTRL	5	CTRL	2	NBD
25	54	3	CTRL	12	CTRL	1	NBD
26	59	1	CTRL	14	CTRL	6	CTRL
27	50	3	CTRL	13	CTRL	9	CTRL

Note. Group assignments were based on scores from one of the depression measures (BDI or Checklist) and the BULIT. S = subject number; BUL = BULIT score; Grp = group assigned by the respective depression measure; Day = day session adjective checklist pre-score; Night = night session adjective checklist pre-score.

Table 4

Differentiation Between Characteristics of Day Groups

	Group			
Characteristic	DB	NDB	NBD	CTRL
BULIT scores				
Mean	100.6	98.3	56.8	54.4
SD	13.6	11.4	12.9	6.95
BDI scores				
Mean	10.9	7.4	8.7	5.3
SD	4.1	2.9	4.0	4.4

Table 5

Average Dependent Measure Pre-Scores of Day Groups

	Group			
Measure	DB	NDB	NBD	CTRL
Day				
EDI Mean	46.4	30.3	16.3	11.9
EDI *SD*	8.0	9.0	13.95	9.8
Chklst Mean	.4	11.9	2.0	8.1
Chklst *SD*	5.9	2.4	3.8	7.1
Scale Mean	4.0	2.4	3.7	2.7
Scale *SD*	1.3	.6	1.2	1.1
Night				
EDI Mean	41.6	33.6	14.3	10.6
EDI *SD*	7.0	8.0	16.0	11.3

Note. Data are only provided for comparisons that yielded one or more significant differences, as indicated in the text. Scale = depression scale; Chklst = adjective checklist.

Table 6

Average Dependent Measure Pre-Scores of Night Groups

	Group			
Measure	DB	NDB	NBD	CTRL
Day				
EDI Mean	40.7	36.0	14.5	13.4
EDI *SD*	13.0	10.7	12.0	12.2
Night				
EDI Mean	38.9	36.3	14.0	10.9
EDI *SD*	9.1	8.0	16.5	10.7
Chklst Mean	-3.3	9.4	.5	6.6
Chklst *SD*	4.1	5.6	3.2	2.7
Scale Mean	4.1	2.5	3.5	3.0
Scale *SD*	.7	.8	.55	.6

Note. Data are only provided for comparisons that yielded one or more significant differences, as indicated in the text. Scale = depression scale; Chklst = adjective checklist.

Table 7

Average Sleep-Deprivation-Produced Change in Dependent Measure Scores of Night Groups

	Group			
Measure	DB	NDB	NBD	CTRL
Chklst				
Mean	2.1	-7.1	2.5	-4.1
SD	4.4	7.1	5.8	7.8
Scale				
Mean	0.0	1.2	0.0	.7
SD	.8	1.0	.9	1.0

Note. Data are only provided for measures exhibiting significant changes, as indicated in the text. Chklst = adjective checklist; Scale = depression scale.

Figure Captions

Figure 1. Mean change in adjective checklist scores for each group, after each session. Negative scores represent increased depression.

Figure 2. Mean change in Likert depression scale scores for each group, after each session. Positive scores represent increased depression.

Figure 1. Average Change in Adjective Checklist After Day and Night Sessions

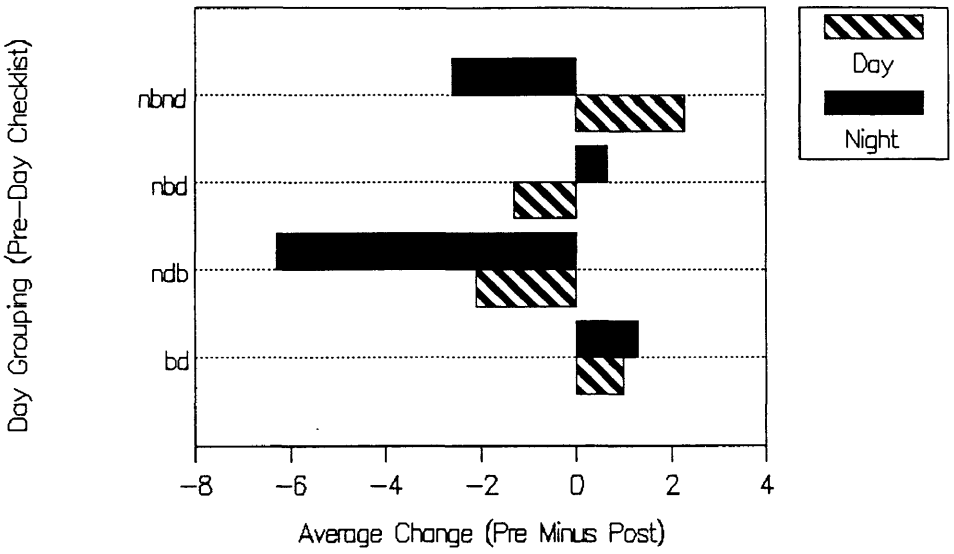
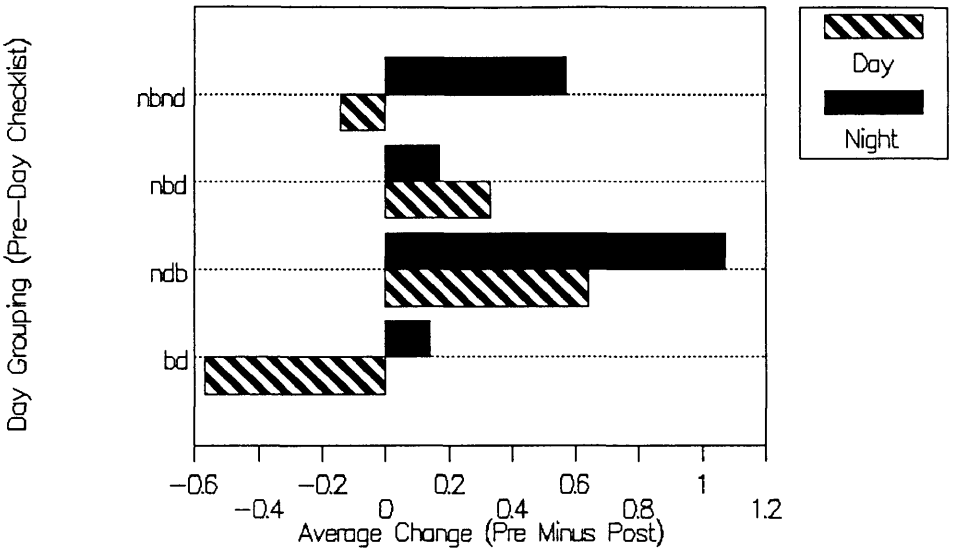


Figure 2. Average Change in Depression Scale After Day and Night Sessions



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