Lehrbuch. (in press). Bulimia Nervosa. In Steiner, H. (Ed.), Handbook of Mental Health Interventions in Children and Adolescents: An Integrated Developmental Approach.

1. Definition of the disorder

Bulimia nervosa is an eating disorder characterized by periods of significant binge-eating followed by different methods of purging in order to compensate for the overeating. Those who suffer from bulimia utilize their binge and purge cycles to prevent weight gain and to regulate other emotional difficulties. Methods of purging include laxative or diuretic use, over-exercising, and most commonly self-induced vomiting. Unlike anorexia nervosa, bulimia generally does not result in significant weight loss, and in many ways is a far more secretive disease. The shame and isolation experienced by those suffering from this disorder further hinders hopes of recovery.

2. Prevalence and Epidemiology

Newer epidemiological studies on eating disorders are population-based, specifically targeting juveniles and using state-of-the-art, two-phase screening designs. Results from these studies reveal that the prevalence of bulimia nervosa in adolescents has increased in the last few decades (Fichter, 1998).

As with other eating disorders, bulimia has a much higher prevalence in women than in men. However, the disorder is increasingly affecting male athletes and performers. Bulimia is more commonly found in Western industrialized nations, specifically in populations that are of middle- and upper class socio-economic status. In the past decade, populations of young African, Latino, and Asian women have also been adversely affected as bulimia has reached more diverse populations.

In France, a two-stage screening for bulimia (survey followed up by interviews) was conducted by Flament et al. (1995). They reported the results from 3,527 unselected secondary school students. Compared to boys, girls reported high rates of overconcern with body weight and shape, dieting, bulimic binges, self-induced vomiting, use of laxatives, and use of diet pills. On the basis of the data, Flament et al. estimated that bulimia has a prevalence rate of approximately 1.1% in girls and 0.2% in boys. It is less frequently found in this age group that in adult women, and the findings from France support clinical observations that most patients with bulimia become ill in the latter half of adolescence.

In the United States, bulimia is thought to affect 3-5% of the entire population, with prevalence in college-aged women reported as high as 19%. Once again, because of the secretive nature of the disorder and the reluctance of young women to seek treatment, exact numbers are hard to quantify.

Epidemiological studies report that even in subjects not qualifying for an eating disorder, patterns of *disordered eating* are highly prevalent. Between 40% and 60% of high school girls in the Unites States diet to lose weight, although the meaning of “dieting” varies

greatly. According to Killen et al., (1986), about 13% induce vomiting or use diet pills, laxatives or diuretics. About 30% to 40% of junior high girls also admit to concerns about weight. Eating disorders not otherwise specified are probably more common than classic eating disorders in juveniles.

Because of the small numbers of males with eating disorders, systematic studies on this population are difficult to conduct. Most studies on bulimia exclude male patients. Males more commonly suffer from bulimia than anorexia, but are still outnumbered by females 5:1 (Flament et al., 1995).

3. Clinical description

The Diagnostic and Statistic Manual-IV (American Psychiatric Association) introduced changes in the diagnostic categories for eating disorders. Both anorexia and bulimia were move to a separate section called "Eating Disorders." For both disorders, body image disturbance may now be expressed in different ways as either 1) distortion of the experience itself or 2) the denial of the seriousness of weight loss.

Like the sub-typing of anorexia indicated the presence of binge-eating/purging versus restricting behaviors, corresponding exclusion criterion were added to the diagnosis of bulimia. Descriptions of binges are now operationalized, and additionally, binge-eating disorder research criteria are included in DSM-IV. Some research demonstrates that the current diagnostic criteria may be too restrictive (Garfinkel et al., 1996).

The DSM-IV diagnostic criteria for bulimia nervosa are:

- recurrent episodes of binge eating (eating an amount (>1000 Calories) that is larger than most people would eat during a similar period of time, and a sense of lack of control over eating during the episode;
- recurrent, inappropriate compensatory behavior in order to prevent weight gain (e.g. self-induced vomiting, misuse of laxatives or diuretics, fasting or excessive exercise);
- these behaviors must occur, on average, at least twice a week for 3 months;
- body shape and weight unduly influence self-evaluation and self-esteem.

The criteria also specify that the disorder must not occur only during episodes of anorexia nervosa. Bulimia nervosa can be either:

- purging type, in which the patient regularly engages in self-induced vomiting or misuses laxatives, diuretics or enemas; or
- non-purging type, in which there is no history of regular self-induced vomiting or misuses of laxatives, diuretics or enemas.

The diagnostic criteria for bulimia nervosa in the ICD-10 are quite similar to those in the

DSM-IV. The only significant difference can be found in the relationship between bulimia nervosa and anorexia nervosa. DSM-IV gives precedence to anorexia nervosa over bulimia nervosa. ICD-10 specifically excludes a diagnosis for anorexia nervosa if binge-eating is present.

4. Etiology and Pathogenesis (with special emphasis placed on the integration of biological and psychosocial factors)

Hypothesized risk factors for the etiology of bulimia nervosa include:

- familial risk factors: first-degree relatives of patients with bulimia nervosa appear to be at increased risk of bulimia nervosa, mood disorders and substance abuse. Additionally, there continues to be evidence for the familial clustering of eating disorders and eating attitudes, which suggest heritable causation for bulimia, especially in families that are more chaotic, conflicted and critical.
- life events: these precipitate onset in 70% cases, and include parental neglect, abuse, indifference, loss, separation
- childhood sexual abuse—in some studies a high incidence of sexual abuse is reported by women with diagnosed eating disorders; rates of abuse are seemingly higher in bulimia than anorexia
- dysregulation of the serotonergic system; most data point to this being secondary to weight changes rather than a primary problem. The study of the neurobiology of eating disorders has demonstrated impressive hormonal and neurohormonal systems differences in adult and late-adolescent patients who are acutely ill (Laue et al., 1991). A study in the United Kingdom suggested that diminished serotonin activity may trigger some of the cognitive and mood disturbances associated with bulimia (Fairburn et al., 1999). The findings also indicate that chronic depletion of plasma tryptophan may be one of the mechanisms whereby persistent dieting can lead to the development of eating disorders in vulnerable individuals.
- pre-determined genetic and physiological factors: being of female gender, having a pear-shaped body, and having a body mass index high in fat (Radke-Sharpe et al., 1990).
- Personality differences: bulimic patients tend to be more affectively labile, undercontrolled and active

A general correlation is that those with low self-esteem and a tendency to depressive symptoms are at an increased risk of bulimia. Other psychological risk factors associated with bulimia nervosa include:

- negative self evaluation
- shyness
- lack of friends
- missing school
- perfectionism
- mood lability

Risk factors for bulimia can also be structured in terms of the major developmental phases of childhood and adolescence. From Steiner et al's (1998) *Anorexia Nervosa and Bulimia Nervosa in Children and Adolescents: A Review of the past 10 Years:*

Preschool: Demographics of early feeding problems suggest discontinuity, because boys are at greater risk of eating disorders in early childhood, whereas in adolescence, girls become so. Marchi and Cohen (1990), using a lagged design, studied two different, overlapping (ages 0 through 10 and 9 through 18) cohorts, following them prospectively for 2.5 years. They studied six eating behaviors at three time points by maternal interview and found that maladaptive early eating patterns increased the likelihood of later problems. Meals during childhood characterized by fighting, indicate problems in the self-regulation of eating and eating-related family struggles. They also are a predictor of the future onset of bulimia.

In a retrospective study of eating disorder patients and controls, Steiner et al., (1991) found that mothers of eating disorder patients report reliance on scheduled feeding and prematurely introduced solids more frequently than controls. Although these practices were independent of the patient's primary diagnosis (anorexia or bulimia), they were also used with siblings who were not ill, suggesting that other factors are likely involved in pathogenesis.

School-Age: In elementary school, children want to be thinner than they are. Thirty seven percent try some form of weight loss, and 6.9% score in the pathological range on an adapted version of the Eating Attitude Test. There were few significant differences between boys and girls. Body image distortions in this group are associated with dieting and weight concerns (Childress et al., 1993). Food refusal, ritualistic behavior during meals, phobic behavior, and elevated Internalizing scales scores on the Child Behavior Checklist are described in school age children that eventually suffer from an eating disorder.

Prepuberty and Adolescence: In a short-term prospective study, Attie and Brooks-Gunn (1989), following 193 girls from 7th through 10th grade for 2 years, tested the hypothesis that the development of eating problems represents an accommodation to puberty. Multiple regressions confirmed that eating problems emerged in response to pubertal change. Girls who felt most negatively about their bodies at puberty were at a higher risk for development of eating difficulties, after initial eating problem scores were taken into account.

Several cross-sectional studies identify associations between disturbed eating and problems with body satisfaction in prepuberty. Altmann et al. (1998) identified a significant association between anxious attachment and eating concerns. Other studies implicate:

1) deficient self-regulation
2) affective lability and pubertal status and
3) concerns about current body shape

Contextual risk factors during this developmental phase include

1) teasing by peers
2) discomfort in discussing problems with parents
3) maternal preoccupation with diets and
4) acculturation to Western values in immigrants

Signs and symptoms
The typical patient with bulimia nervosa is within the normal weight range for age and height. Behavioral signs of bulimia nervosa include:

- surreptitious behavior (e.g. hiding food, spending long periods in the bathroom with vague excuses);
- outwardly restrictive meal patterns or over-concern with dieting and nutrition but with little change in weight or appearance;
- dissatisfaction with body size and shape.

Many of the physical signs and abnormalities that are associated with bulimia nervosa are due to recurrent vomiting, and so are more likely to be seen in patients with purging-type bulimia nervosa:

- tooth decay caused by loss of dental enamel as a result of recurrent vomiting - this loss of enamel affects the inner aspects of the front teeth in particular;
- dehydration;
- fatigue;
- swollen salivary glands;
- scars on the dorsum of hand (Russell's sign) caused by manual stimulation of the gag reflex to induce vomiting;
- electrolyte disturbances;
- esophageal or gastric tears;
- side effects of emetics, diuretics or purgatives.

Patients with bulimia nervosa often have features of depression (30-70% lifetime rates are reported), especially low self-esteem. They may also have features of anxiety (again, 30-70% lifetime rates are reported). Patients also have high rates of anxiety disorder and panic disorder, and post-traumatic stress disorder is common. Deliberate self harm and alcohol and substance misuse are also common. Approximately half the clinical sample report stealing.

5. Differential diagnosis

The differential diagnosis of bulimia nervosa includes:

- binge-eating disorder, which is characterized by bingeing without purging;

- anorexia nervosa, which may also involve bingeing and purging, but not as consistently as in bulimia nervosa; the patient must be at least 15% below expected body weight;
- gastrointestinal disorders.

Comorbid psychological conditions
Bulimia nervosa may be associated with:

- depression;
- anxiety disorder;
- borderline personality disorder;
- anorexia nervosa;
- substance misuse.

Medical complications
Medical complications of bulimia nervosa include:

- gastric distress or bleeding;
- esophagitis or esophageal tears;
- dental problems;
- cardiac complications (e.g. arrhythmia, tachycardia);
- muscle cramping due to electrolyte imbalance;
- renal failure.

Social problems
Social problems that commonly arise as a result of bulimia nervosa include:

- impairment in the patient's social network, social isolation and poor social skills;
- reduction in the patient's financial status as a result of money spent on food and purgatives;
- impairment in family relationships as a result of concealment and lying.

6. Clinical instruments and methods for diagnosis

Assessment instruments for bulimia are often generalized for eating disorders as a whole. Because these problems present themselves with a multitude of disturbances in multiple domains, it is not prudent to isolate specific tests for each of the different disorders. Specific structured interviews such as the Eating Disorder Examination (Cooper and Fairburn, 1987) are available as are screening instruments in parent and child versions (Slade et al., 1990). A study out of the University of Munich found that in a comparison of the SIAB-EX with the EDE, they both showed similar results even though there are a number of differences between the two scales (Fichter et al., 2001). Other clinical self-reports include:

1) the Eating Disorder Inventory, which has normative data down to 14 years (Shore and Porter, 1990)

2) the Eating Attitude Test, which has a version applicable to school-age children (Maloney et al., 1988)
3) the Kids Eating Disorder Survey, which is applicable to middle school children (Childress et al., 1993) and
4) online eating disorder screening websites which have recently been promoted on college campuses. Empirical data is not available as of yet on the success or use of this method, but it is valued for the confidentiality and easy accessibility to younger students
5) Structured Interview for Anorexic and Bulimic Disorders: the 87-item SIAB-EX was originally developed for detailed assessment of eating disorders cross-sectionally and longitudinally. In 1998, German doctors modified earlier versions of it in order to include new research findings and to update the expert rating interview to the diagnostic criteria of DSM-IV and ICD-10 (Fichter et al., 1998). The semistandardized interview was developed for reliable and valid assessment of the specific as well as the general psychopathology of eating disorders. It is divided into the following sections: (I) Body Image and Slimness Ideal; (II) General Psychopathology; (III) Sexuality and Social Integration; (IV) Bulimic Symptoms; (V) Measures to Counteract Weight Gain, Fasting, and Substance Abuse; and (VI) Atypical Binges.

Laboratory studies may be helpful to identify the degree of electrolyte imbalance caused by frequent purging and to determine the individual's nutritional status and state of overall health. Biochemical abnormalities may include:

- hypokalemia, hypochloremia and hyponatremia, caused by frequent purging;
- metabolic alkalosis, caused by loss of stomach acid brought about by vomiting;
- metabolic acidosis, which may result from frequent self-induced diarrhea.

Laboratory profiles can be followed serially to monitor fluid balance and particularly to detect hypokalemia.

7. Analysis of Treatment Studies; Treatment Options for Bulimia

Treatment studies of bulimia are more advanced than those for anorexia. Still, the focus of the majority of these studies is on young adult populations. For patients suffering from bulimia nervosa, the most common modes of treatment involve therapy, medication or a combination of both. There is evidence that treatment can be successful, but even with effort in such cases studies it is still unclear whether significant risks and vulnerabilities for recurrence ever completely resolve (Herzog et al., 1993).

The treatment for bulimia nervosa involves a `stepped care' approach, starting with primary care and followed in turn, as necessary, by hospital outpatient treatment, in-patient treatment in a general psychiatry unit, and specialist unit-based therapies. However, the most promising studies for adolescents with bulimia suggest a role for family therapy. A study of 49 adolescent eating disorder patients and their families found that mother’s critical comment explained 28% to 34% of the variance in outcome in

patients and that this rating was the best outcome predictor (van Furth et al., 1996). Two recent studies of family environments of bulimic patients identified physical punishment as contributing to overall outcome difficulties and as indicative of worse family functioning. Additionally, a pilot study found that treatment of females (aged 14 through 17 years) with brief family therapy resulted in significant decreases in bulimic behavior at 1 year (Dodge, 1995). Interpersonal therapy has not been systematically studied in youth.

Recent studies on the treatment of bulimia have demonstrated that for adults, cognitive interventions and antidepressants, especially selective serotonin reuptake inhibitors, are potentially effective. These findings do not necessarily apply to adolescent populations. Ultimately, data indicates that family therapy and intensive treatment modalities should play an important role in any attempt at recovery.

The primary goals of treatment for bulimia nervosa are:

- to help a patient achieve abstinence from bingeing and purging;
- to help a patient learn, regain and maintain healthy eating patterns;
- to address other psychological issues related to the primary diagnosis of bulimia.

Treatment options
For bulimia nervosa, the most commonly used treatments are psychological interventions and patient-led interventions. Non-pharmacological methods include: 1) cognitive behavioral therapy, 2) behavioral techniques, 3) relaxation training, 4) stimulus control, 5) family therapy, and 6) group therapy. The aforementioned treatments are described in further detail below.

Cognitive-behavioral therapy
There are four distinct phases in cognitive-behavioral therapy for bulimia nervosa:

- Initially, a clinician makes an assessment of the patient's psychological, emotional and behavioral functioning. This is done by means of a clinical interview and through self-monitoring by the patient of nutritional intake, as well as bingeing and purging behaviors.
- The patient is then educated about healthy, regular eating patterns and is encouraged to resume or engage in such nutritious eating. This is an attempt to normalize a bulimics more sporadic and out of control dietary intake.
- Cognitive-behavioral therapy seeks to cognitively restructure the patient’s distortions about food, thinness, achievement and assertiveness.
- Finally, by continually discussing signs of relapse and focusing on preventative strategies an emphasis is placed on the prevention of relapse. With signs of progress, therapy is slowly tapered off.

Behavioral techniques
In the treatment of bulimia nervosa, the technique of exposure plus response prevention is often used. This method is based on a model of anxiety and phobic avoidance that is

involved in the binge-purge cycle. Either gradually or through a binge, the bulimic patient is exposed to foods that he or she fears. They are then prevented from purging. With repeated exposure to the foods, without compensatory purging behaviors, the hope is that the patient's anxiety decreases over time. Concomitantly, the patient also becomes less fearful of normal eating.

Relaxation training
A prime example of relaxation training is progressive muscle relaxation. Bulimic patients benefit from learning about alternative means to deal with negative emotions such as anxiety. Techniques such as this have demonstrated success with bulimic patients. By playing a pre-planned tape of smooth, calming music, with a voice-over of directions, patients follow a routine of tightening and relaxing muscles in their entire body. Relaxation training becomes increasingly useful as patients become more comfortable with the techniques and are able to maximize the benefits of the exercise.

Stimulus control
Antecedent and consequential behaviors associated with bingeing and purging are examined and restructured to prevent binges and purges.

Family therapy
Patients whose family dynamics contribute to or exacerbate bulimic symptoms may benefit from family therapy. However, it is less effective than in anorexia, although it is often helpful for adolescents.

Group therapy
Patients who demonstrate particularly poor social skills and who appear particularly susceptible to group or societal pressures toward thinness may benefit from group therapy targeted at bulimia recovery.

Pharmacological treatment
The mainstay of treatment is non-pharmacological. However, antidepressants have been used in the treatment of bulimia nervosa with encouraging results, and about 30% of patients benefit from pharmacotherapy alone. Fluoxetine (a selective serotonin reuptake inhibitor) and imipramine (a tricyclic antidepressant) are commonly used antidepressants in the treatment of bulimia nervosa.

Standard dosages
Standard antidepressant dosages are not used in bulimia - higher doses are used than for depression, with careful side effect monitoring. Smaller doses will be needed in children and adolescents.

- fluoxetine: 60mg/day;
- imipramine: 150-300mg/day

A recent study investigated whether treatment with fluoxetine was useful for individuals with bulimia nervosa who did not respond to psychotherapy or who had relapsed

afterward. Researchers concluded that fluoxetine may be a useful intervention for patients with bulimia nervosa who have not responded adequately to psychological treatment (Walsh et al., 2000).

Contraindications

Fluoxetine
Fluoxetine is contraindicated in patients taking concurrent monoamine oxidase inhibitors and within 14 days of use of a monoamine oxidase inhibitor.
Care is needed in patients with:

- renal or hepatic impairment;
- a bipolar disorder;
- a seizure disorder;
- diabetes mellitus;
- suicidal ideation.

Imipramine
Imipramine is contraindicated in patients taking concurrent monoamine oxidase inhibitors and within 14 days of use of a monoamine oxidase inhibitor. It should also not be given to patients with:

- a recent myocardial infarction;
- an arrhythmia, particularly heart block;
- a seizure disorder.

Care is needed in patients with:

- hepatic impairment;
- thyroid disease;
- pheochromocytoma;
- closed-angle glaucoma;
- a history of urinary retention.

Main side effects

Fluoxetine
The main side effects include:

- gastrointestinal reactions (nausea, vomiting, indigestion, abdominal pain, diarrhea), which are dose-related;
- seizures, anxiety, fatigue, dizziness, tremor;
- antimuscarinic effects (e.g. dry mouth, constipation, urinary retention).

A withdrawal syndrome is seen in up to 60% of patients in whom a specific serotonin reuptake inhibitor (such as fluoxetine) is stopped suddenly. This syndrome can cause

dizziness, anxiety, agitation, confusion, tremor, paresthesiae, nausea and sweating.

Imipramine
The main side effects include:

- dizziness, drowsiness;
- tachycardia, arrhythmias, orthostatic hypotension;
- anticholinergic effects (e.g. dry mouth, blurred vision, constipation, urinary retention);
- acute renal failure;
- blood dyscrasias.

Main drug interactions

Fluoxetine
Fluoxetine should not be given within 14 days of the use of a monoamine oxidase inhibitor. Other potentially significant interactions include:

- dexfenfluramine;
- non-sedative antihistamines (may carry increased risk of arrhythmias);
- anticoagulants (anticoagulant effect may be enhanced);
- other antidepressants.

Imipramine
Imipramine should not be given within 14 days of the use of a monoamine oxidase inhibitor.

8. Course and Prognosis

The course of bulimia nervosa varies, but the disorder usually persists for at least several years. The majority of victims experience a chronic fluctuation of binge/purge behavior. In one study, at 3-year follow-up approximately one third of patients remained in the index episode; of those who recovered from the index episode, two thirds relapsed within a year and a half, and of those who recovered from this second episode, half relapsed (Keller et al., 1992). Few studies are available on the longer-term outcome of bulimia. A 6-year follow-up study out of Munich, Germany found that based on a composite global outcome score, 59.9% achieved a good outcome, 29.4% an intermediate outcome, 9.6% a poor outcome, and 2 (1.1%) persons were deceased (Fichter et al., 1997). The course and outcome for bulimic women was generally more favorable than for anorexics. A recent study from Oxford, England found that, among young women, bulimia nervosa and binge eating disorder had a different course and outcome. Whereas the prognosis of those with bulimia nervosa was relatively poor, the great majority of those with binge eating disorder recovered in a five-year tracking period (Fairburn, et al., 2000).

Late adolescence and early adulthood are the usual times of onset of bulimia. Binge eating generally begins during an episode of restricting and prolonged dieting. During

this stage, if identified, patients typically seek treatment and appear to be motivated in their desire to overcome such a problem. However, once the act of purging starts and is reinforced, patients become extremely resistant to changing their habits. Although bulimics experience variations in their weight, they rarely approach the low weight ranges associated with anorexia. Because of other medical complications—such as hypokalemia, esophageal tears, gastric disturbances, dehydration, and orthostatic blood pressure changes—bulimics may require intermittent hospitalization (Kreipe et al., 1995).

As opposed to those with anorexia, bulimic patients have a history of impulsive behaviors, including alcohol use and shoplifting. Their preoccupation with food often impairs their functioning in social, school, and work related activities.

Up to 70% benefit from the combination of medication and cognitive-behavioral interventions. The long-term outcome of bulimia nervosa is still under study. Without treatment, the disorder follows a chronic fluctuating course, but 70% are symptom-free at 5-10 years. Mortality is approximately 1%.

With regards to follow-up and management of the disorder, regular review to ensure that healthy eating patterns are being maintained and to assess the patient's coping skills may be beneficial. Some patients find self-help support groups helpful. Regular moderate exercise may help patients to deal with the weight fluctuations that can be expected while their bodies adjust to their new healthy eating patterns. Researchers in the UK suggest that overall, readiness to change is more strongly related to improvement and the development of a therapeutic alliance than the specific type of treatment offered to bulimic patients (Treasure et al., 1999).

9. Promising areas for future investigation

The best documented studies on eating disorders are epidemiological studies on their prevalence and incidence, as well as long-term outcome studies on anorexia nervosa, and short-term treatment response in bulimia nervosa. According to Steiner et al. 1998, “Data approaching eating disorders from a developmental perspective are available in only a few studies. Research is needed addressing normative data on the development of eating behavior and specific risk and resilience factors for pathology in specific developmental periods. Especially lacking are studies regarding the continuities and discontinuities of eating disturbances across the life span.”

Eating disorders are often conceptualized as developmental disorders. However, few prospective studies examine normative and pathological phenomena in populations at risk (Steiner et al., 1995). Only a few studies have used longitudinal designs. In the past 15 years however, a larger portion of the literature has directly addresses problems in youth, resulting in a special volume. Another problem with past studies is that they often mix samples of adults and juveniles. They also rarely control for the age of onset of illness or duration of illness. These factors confound treatment results, and studies addressing these issues from a developmental perspective need to be conducted.

Hypotheses on the etiology of bulimia nervosa have spanned the gamut of possibilities. From familial to organic to psychosocial factors, researchers have come to realize that a combination of several factors often plays into the development of bulimia.

Promising areas of future investigation include studies on genetic predisposition to bulimia. Genetic studies on candidate genes have mainly focused on the serotonergic system and on genes involved in body weight regulation. Studies in Germany by Remschmidt et al (2000) seek to identify new pharmacological treatment strategies for bulimia. Currently, combined efforts within the European Union will answer the question of whether or not the A-allele is involved in the predisposition to anorexia nervosa. A transmission disequilibrium test is being performed in about 300 trios consisting of a patient with anorexia nervosa and both parents. As candidate gene approaches did not unequivocally identify susceptibility genes (alleles) for anorexia nervosa or bulimia nervosa, systematic model-free genome-wide screenings should also be performed in order to identify currently unknown genes involved in eating disorders. This kind of approach on genetic research has already been initiated for anorexia nervosa.

Other avenues of future investigation hope to find a distinction between whether bulimia is an issue of psyche or soma. Researchers from Maudsley Hospital in London emphasize the “complex interaction between biological and psychological factors” leading to a lifestyle of bingeing and purging (Ward, 2000). Future studies are under way, that identify the interplay between organic and psychosocial causation. As many scientists believe, “focusing exclusively on either aspect” would be a disservice to bulimic patients everywhere.

WORKS CITED

Altmann, TM; Killen, JD; Bryson et al: Attachment style and weight concerns in preadolescent and adolescent girls. International Journal on Eating Disorders 23, 39-44, 1998.

Attie, I; Brooks-Gunn, J: Development of eating problems in adolescent girls: a longitudinal study. Developmental Psychology 25, 70-79, 1989.

Childress, A; Brewerton, T; Hodges, E; Jarrell, M: The Kids Eating Disorder Survery (KEDS): a study of middle school students. Journal of the American Academy of Child and Adolescent Psychiatry 32, 843-850, 1993.

Cooper, Z; Fairburn, CG: The Eating Disorder Examination: a semi-structured interview for the assessment of the specific psychopathology of eating disorders. International Journal on Eating Disorders 6, 1-8, 1987.

Diagnostic and Statistical Manual of Mental Disorders, 4th ed Text Revision (DSM-IV-TR). *Washington DC: American Psychiatric Association*; 589-594, 2000.

Dodge, E: Family therapy for bulimia nervosa in adolescents: an exploratory study. Journal of Family Therapy 17, 59-77, 1995.

Fairburn, CG; Cooper, Z; Doll, HA; Norman, P; O'Connor, M: The natural course of bulimia nervosa and binge eating disorder in young women. Archives of General Psychiatry, 2000

Fairburn, CG; Smith, KA; Cowen, PJ: Symptomatic relapse in bulimia nervosa following acute tryptophan depletion. Archives of General Psychiatry 56, 171-176, 1999.

Fichter, M; Quadflieg, N: The structured interview for anorexic and bulimic disorders for DSM-IV and ICD-10 (SIAB-EX): reliability and validity. European Psychiatry 16, 38-48, 2001.

Fichter, MM; Herpertz, S; Quadflieg, N; Herpertz-Dahlmann, B: Structured Interview for Anorexic and Bulimic disorders for DSM-IV and ICD-10: updated (third) revision. International Journal on Eating Disorders 24, 227-249, 1998.

Fichter, MM; Quadflieg, N: Six-year course of bulimia nervosa. International Journal on Eating Disorders 22, 361-384, 1997.

Flament, M;; Ledoux, S; Jeammet, P; Choquet, M; Simon, Y: A population study of bulimia nervosa and subclinical eating disorders in adolescence. In: Steinhausen, HC (ed): Eating Disorders in Adolescence: Anorexia and Bulimia Nervosa. 21-36. De Gruyter, New York 1995.

Garfinkel, PE; Lin, E; Goering, P et al: Should amenorrhoea be necessary for the diagnosis of anorexia nervosa? Evidence from a Canadian community sample. British Journal of Psychiatry 168, 500-506, 1996.

Herzog, DB; Sacks, NR; Keller, MB; Lavori, PW; von Ranson, KB; Gray, HM: Patterns and predictors of recovery in anorexia nervosa and bulimia nervosa. Journal of the American Academy on Child and Adolescent Psychiatry 31, 810-818, 1993.

Keller, MB; Herzog, DB; Lavori, PW; Bradburn, IS; Mahoney, EM: The natural history of bulimia nervosa. Int J Eat Disorders 12, 1992.

Killen, JD; Taylor, CB; Telch, MJ; Saylor, KE; Maron, DJ; Robinson, TN: Self-induced vomiting and laxative and diuretic use among teenagers: precursors of the binge-purge syndrome? Journal of the American Medical Association 255, 1447-1449, 1986.

Kreipe, RE; Golden, NH; Katzman, DK et al: Eating disorders in adolescents: a position paper of the Society for Adolescent Medicine. Journal on Adolescent Health 16, 476-479, 1995.

Laue, L; Gold, PW; Richmond, A; Chrousos, GP: The hypothalamic-pituitary-adrenal axis in anorexia nervosa and bulimia nervosa: pathophysiologic implications. Advanced Pediatric 38, 287-316, 1991

Maloney, MJ; McGuire, JB; Daniels, SR: Reliability testing of a children's version of the Eating Attitude Test. Journal of the American Academy of Child & Adolescent Psychiatry 27, 541-543, 1988.

Marchi, M; Cohen, P: Early childhood eating behaviors and adolescent eating disorders. Journal of the American Academy on Child and Adolescent Psychiatry 29, 112-117, 1990.

Radke-Sharpe, N; Whitney-Saltiel, D; Rodin, J: Fat distribution as a risk factor for weight and eating concerns. International Journal on Eating Disorders 9, 27-36, 1990.

Remschmidt, H; Hinney, A; Hebebrand, J.: Candidate gene polymorphisms in eating disorders. European Journal of Pharmacology 27, 147-159, 2000.

Shore, RA; Porter, JE: Normative and reliability data for 11 to 18 year olds on the Eating Disorder Inventory. International Journal on Eating Disorders 9, 201-207, 1990.

Steiner, H; Lock, J: Anorexia nervosa and bulimia nervosa in children and adolescents: a review of the past 10 years. Journal of the American Academy of Child and Adolescent Psychiatry 37:4, 352-359, 1998.

Steiner, H; Sanders, M; Ryst, E: Precursors and risk factors of juvenile eating disorders. In: Steinhausen, HC (ed): Eating Disorders in Adolescence: Anorexia and Bulimia Nervosa. 21-36. De Gruyter, New York 1995.

Steiner, H; Smith, C; Rosenkrantz, R; Litt, IF: The early care and feeding of anoretics. Child Psychiatry and Human Development 21, 163-167, 1991.

The ICD-10 Classification of Mental and Behavioural Disorders: clinical descriptions and diagnostic guidelines (ICD-10). *Geneva: World Health Organization;* 178-179, 1992.

Treasure, JL; Katzman, M; Schmidt, U; Troop, N; Todd, G; de Silva, P: Engagement and outcome in the treatment of bulimia nervosa: first phase of a sequential design comparing motivation enhancement therapy and cognitive behavioural therapy. Behav Res Therapy 37, 405-418, 1999.

Van Furth, EF; van Strien, DC; Martina, LM; van Son, MJ; Hendrickx, JJ; van Engeland, H: Expressed emotion and the prediction of outcome in adolescent eating disorders. International Journal of Eating Disorders 20, 19-31, 1996.

Walsh, BT; Agras, WS; Devlin, MJ; Fairburn, CG; Wilson, GT; Kahn, C; Chally, MK: Fluoxetine for bulimia nervosa following poor response to psychotherapy. American Journal of Psychiatry, 2000.

Ward, A; Tiller, J; Treasure, J; Russell G. Eating disorders: psyche or soma? International Journal on Eating Disorders 27, 279-287, 2000.

IV. Author's questionnaire

Place/ Date: Stanford, CA; December, 2001
Title of volume: **please fill in**
Title of contribution: Bulimia Nervosa
Author(s) name(s), first name(s): Ashwini Sagar
Academic Title: MD Candidate
Address: Stanford University, PO Box 13495; Stanford, CA 94309
Telephone: 480-768-1533
E mail: ashsagar@stanford.edu

Ashwini Sagar
Birthdate: November 12, 1980.
University of graduation: Stanford University ('02); B.A. in Psychology (with Honors)
Currently: MD Candidate.
Professional goals: I hope to work in the women's health field, and am specifically passionate about issues of mind and body wellness.