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Are Eating Disorders Culture-Bound Syndromes? Implications for Conceptualizing Their Etiology

Pamela K. Keel
Harvard University

Kelly L. Klump
Michigan State University

The authors explore the extent to which eating disorders, specifically anorexia nervosa (AN) and bulimia nervosa (BN), represent culture-bound syndromes and discuss implications for conceptualizing the role genes play in their etiology. The examination is divided into 3 sections: a quantitative meta-analysis of changes in incidence rates since the formal recognition of AN and BN, a qualitative summary of historical evidence of eating disorders before their formal recognition, and an evaluation of the presence of these disorders in non-Western cultures. Findings suggest that BN is a culture-bound syndrome and AN is not. Thus, heritability estimates for BN may show greater variability cross-culturally than heritability estimates for AN, and the genetic bases of these disorders may be associated with differential pathoplasticity.

The influence of culture on the development of eating disorders such as anorexia nervosa (AN) and bulimia nervosa (BN) has been long appreciated. These syndromes are more prevalent in industrialized and often Western cultures and are far more common among females than males, mirroring cross-cultural differences in the importance of thinness for women (Miller & Pumariega, 2001; Pate, Pumariega, Hester, & Garner, 1992). Furthermore, eating disorders seem to have become more common among younger females during the latter half of the twentieth century, during a period when icons of American beauty (Miss America contestants and *Playboy* centerfolds) have become thinner and women's magazines have published significantly more articles on methods for weight loss (Garner, Garfinkel, Schwartz, & Thompson, 1980; Owen & Laurel-Seller, 2000; Rubinstein & Caballero, 2000; Wiseman, Gray, Mosimann, & Ahrens, 1992). Patients with eating disorders often describe intense preoccupation with weight and dieting as the first stages of their illness, and the American Psychiatric Association's (2000) diagnostic criteria for AN and BN require body image disturbance. The cognitive–behavioral theory of eating disorders places weight concerns and dieting at the center of their etiology (Fairburn, Cooper, & Shafran, 2002). These patterns indict current cultural beauty ideals in the etiology and maintenance of eating disorders.

Yet, a recent trend in eating disorders research has been to focus on their genetic bases and deemphasize the role of culture (cf. DeAngelis, 2002). Briefly, data from community-based twin studies have suggested that the heritability of eating disorders is greater than 50% (Bulik, Sullivan, & Kendler, 1998; Bulik, Sullivan, Wade, & Kendler, 2000; Kendler et al., 1991, 1995; Klump, Miller, Keel, McGue, & Iacono, 2001; Kortegaard, Hoerder, Joergensen, Gillberg, & Kyvik, 2001; Wade, Bulik, Neale, & Kendler, 2000), and several recent reports have emerged indicating specific genetic loci for susceptibility to eating disorders (Grice et al., 2002; Koronyo-Hamaoui et al., 2002; Ricca et al., 2002; Westberg et al., 2002). Indeed, one eating disorders researcher has equated the shift from psychosocial to biological theories of etiology in eating disorders to the shift that occurred within schizophrenia research (DeAngelis, 2002). However, unlike schizophrenia, eating disorders have been characterized as culture-bound syndromes.

Prince (1985) defined a culture-bound syndrome as "a collection of signs and symptoms (excluding notions of cause) which is restricted to a limited number of cultures primarily by reason of certain of their psychosocial features" (p. 201). With this definition, he proposed that AN might represent a culture-bound syndrome. However, he cautioned that "the decision hinges on the empirical question of whether or not the syndrome occurs in non-Western cultures or segments of them which are not markedly influenced by Western cultures" (Prince, 1985, p. 201). In contrast, Swartz (1985) argued that evidence of AN in non-Western cultures would not decrease the extent to which it represents a culture-bound syndrome because AN cannot be understood separated from its cultural context:

> The identical symptoms now may mean different things from what they may have meant in the late 19th century when the disorder was first documented in the form we now recognize. This implies that it may be valid to relate symptoms now occurring to social factors which may not have existed in the same form years ago. (p. 727)

Thus, the same disorder could be attributed to different social factors (the aspect of the syndrome that is culture bound) despite having existed in different cultural contexts. Swartz's distinction between disorder and culturally embedded syndrome resembles

Pamela K. Keel, Department of Psychology, Harvard University; Kelly L. Klump, Department of Psychology, Michigan State University.

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Correspondence concerning this article should be addressed to Pamela K. Keel, who is now at the Department of Psychology, University of Iowa, Iowa City, Iowa 52242. E-mail: pamela-keel@uiowa.edu

Kleinman's (1987) distinction between disease and illness. According to Kleinman, *disease* represents the biological entity for which the pathophysiology is known, and *illness* represents the culturally shaped manifestation of the disease.

Because research into the genetic diathesis of eating disorders represents a search for their underlying disease, this search would benefit from an examination of whether or not core features of these syndromes exist outside of modern Western influences. We begin by evaluating whether rates of eating disorders have increased significantly over recent decades because evidence of a secular increase has been used as the cornerstone of arguments that they are caused by the recent emphasis on thinness (Garner et al., 1980; Russell, 1985). However, this evidence has been challenged by several investigators (Fombonne, 1995, 1996; Lucas, 1992; Pawluck & Gorey, 1998; Van't Hof & Nicolson, 1996), sparking considerable debate on this point. We then examine the cross-historical and cross-cultural evidence for eating disorders. The purpose of this article is to explore the extent to which eating disorders, specifically AN and BN, represent culture-bound syndromes and to discuss the implications of these findings for understanding the role genes play in their etiology.

Method

In order to examine the extent to which eating disorders are represented outside of their current sociohistorical context, this review was subdivided into three sections: epidemiology since the recognition of the modern syndromes, historical evidence of the syndromes prior to their formal recognition, and cross-cultural evidence of the syndromes.

Meta-analyses were conducted to investigate whether AN and BN have become more common during the twentieth century by examining secular trends in incidence rates (newly identified cases per 100,000 population per year) according to the methods described in Rosenthal and Rosnow (1991). Specific details of this method are described within the *Epidemiology of AN in the Twentieth Century* section in the *Data coding* subsection. As noted by Hoek (1993; Hoek, Van Hoeken, & Katzman, 2003), epidemiological studies of AN tend to use incidence, and epidemiological studies of BN tend to use prevalence (percentage of individuals who currently [point] or have ever [lifetime] met criteria for a disorder regardless of when the disorder first emerged). Because incidence rates track when cases are first identified, only incidence rates are considered in the meta-analyses. Both the cross-historical and cross-cultural sections represent a qualitative review, as they necessarily relied more on specific case histories for sufficient detail to evaluate the presence of eating disorders.

We restricted our review to published works. Benefits of this approach include the increased scientific rigor of published (particularly peer-reviewed) work compared with unpublished work and permanent availability of published work within the public domain for independent evaluation by others. Because restriction to published articles has the potential to be biased by the "file-drawer phenomenon" (Rosenthal & Rosnow, 1991), we have included information concerning the number of studies with nonsignificant results that would alter meta-analytic findings and interpretations within the *Epidemiology of AN in the Twentieth Century* and *Epidemiology of BN in the Twentieth Century* sections.

Definitions

We sought information for the diagnostic entities of AN and BN as they are conceptualized in the *Diagnostic and Statistical Manual of Mental Disorders* (4th ed.; *DSM–IV*; American Psychiatric Assocation, 1994; 4th ed., text rev.; *DSM–IV–TR*; American Psychiatric Association, 2000). We did not attempt to limit our review to cases meeting full *DSM–IV* criteria for AN and BN. Rather the *DSM–IV* was used to define boundaries that separate these as distinct syndromes. Although a great deal of recent attention has been paid to binge-eating disorder in the literature, less emphasis has been placed on the role of culture in the etiology of this proposed syndrome, and significant findings on its genetic bases remain limited (Burnet, Smith, Cowen, Fairburn, & Harrison, 1999; Branson et al., 2003; Ricca et al., 2002). We therefore restricted our review to AN and BN.

Using the *DSM–IV* conceptualization of AN and BN to organize our search introduces a sociohistorical bias. Thus, a brief discussion of both our rationale and our attempts to recognize these biases is warranted. The first issue concerns the overlap of AN and BN. In the *Diagnostic and Statistical Manual of Mental Disorders* (3rd ed.; *DSM–III*; American Psychiatric Association, 1980), a diagnosis of AN trumped a diagnosis of bulimia. In the *Diagnostic and Statistical Manual of Mental Disorders* (3rd ed., rev.; *DSM–III–R*; American Psychiatric Association, 1987), both diagnoses could be given concurrently. In the *DSM–IV*, a diagnosis of AN again trumps a diagnosis of BN. Based on data concerning age of onset, naturalistic course, treatment response, and mortality, the *DSM–IV* conceptualization has considerable empirical support (Keel & Mitchell, 1997; Keel, 2003). Moreover, women with concurrent symptoms of AN and BN do not differ from women with the restricting subtype of AN, and all women with AN differ significantly from women with BN alone on naturalistic course, treatment response, and mortality (Herzog et al., 1999; Keel et al., 2003). The second issue concerns the combination of binge eating with inappropriate compensatory behavior in defining BN. In the *DSM–III*, inappropriate compensatory behavior in the form of self-induced vomiting was included as a possible, but not necessary, feature of bulimia. Subsequent work comparing individuals with binge eating alone versus binge eating and inappropriate compensatory behavior, most often purging, shows significant differences in gender ratios, etiologic factors, clinical correlates, and course (Halmi, 2003; Keel, 2003). Moreover, the *DSM–IV* definition of BN has demonstrated predictive validity in being distinguished from both AN and binge-eating disorder (Keel, Mitchell, Miller, Davis, & Crow, 2000). Despite evidence supporting the validity of the *DSM–IV* conceptualizations of AN and BN, there remain several definitional shortcomings. The impact of these shortcomings on evidence of these disorders outside of their current sociohistorical context is considered and discussed throughout the review. Particular attention is given to the cognitive features of body image disturbance that define these disorders.

One aspect of this review that became challenging was the need to define the construct of a "non-Western" culture. In reading other reviews, it became clear that what was considered non-Western varied considerably. Indeed, the *Cassell Dictionary of Modern Politics* (East & Joseph, 1994) describes *the West* as "a misleading term in a variety of usages" (p. 319). For the purposes of this article, we selected the definition for *non-Western* used by Karan (in press):

> The area whose cultures developed essentially apart from the Greco–Judaic–Christian tradition of the Western culture. Thus, it includes East Asia (China, Japan, and Korea), Southeast Asia, the Indian subcontinent, the Middle East, and Sub-Saharan Africa. Latin America, Russian Asia, and Oceania are excluded. The indigenous cultures of Latin America and Oceania are not western, but they are also not non-Western . . . the contemporary cultural pattern in Latin America and Oceania, unlike the non-Western cultures of Asia and Africa, is based dominantly on western traditions and values. (p. 1)

Although clear cultural differences exist among countries such as Bulgaria, Portugal, and the United States and may seem greater than cultural differences among Hong Kong, Japan, and the United States, Karan's (in press) definition of *non-Western* is influenced less by recent political and economic changes that essentially coincide with the recognition of eating disorders within Western cultures. Further, this definition acknowledges aspects of culture that are derived from historically unique evolutions of civilization.

Literature Search

Literature reviews were conducted using computer databases (PsycINFO, 1887–June 2002; MEDLINE, 1966–June 2002; RLG's Eureka Anthropological Literature, 1900–June 2002; and ABC-CLIO Historical Abstracts, 1954–June 2002) and hand searches of the reference sections of all articles, books, and book chapters retrieved by computer and hand search. The following search terms were used: *eating disorder*, *anorexia nervosa*, *bulimia*, *epidemiology*, *incidence*, *prevalence*, *history*, *culture*, *cross-cultural*, and the names of every nation and continent in the world with the explode option to detect relevant permutations of terms. For the historical search, the terms *binge*, *purge*, *vomit*, and *starvation* were also included using the explode option. Pamela K. Keel reviewed references for the epidemiology and cross-historical sections. Titles and abstracts retrieved for non-Western nations (n = 1,190) were evaluated by Kelly L. Klump for potential relevance, and articles were obtained to determine whether cases of AN or BN were reported in non-Western nations. Although several articles provided evidence of elevated levels of disordered eating behaviors and attitudes in non-Western cultures as assessed by standardized questionnaires (Al-Subaie, 2000; Al-Subaie et al., 1996; Hooper & Garner, 1986; A. M. Lee & Lee, 1996; S. Lee, 1993; S. Lee & Lee, 2000; S. Lee, Lee, & Leung, 1998; S. Lee, Lee, Leung, & Yu, 1997; S. Lee, Leung, Lee, Yu, & Leung, 1996), we sought specific instances of clinical eating disorders in these regions. Because we were specifically interested in evidence of eating disorders outside of our current sociohistorical context, an attempt was made to include information from articles regardless of language. Thus, some information is from English translations of work originally published in another language and abstracts written in English for articles written in non-English languages, as well as reviews of these non-English works by other authors. Much of the historical information prior to the mid-seventeenth century was collected through secondary sources because English translations were unavailable for many original sources. Although this section of the review is vulnerable to the interpretations of the secondary source, many of the cases were reviewed by multiple authors with differing theses, thus allowing a reasonably comprehensive presentation.

Anorexia Nervosa

According to the *DSM–IV–TR*, AN is defined by the following diagnostic criteria:

> A. Refusal to maintain body weight at or above a minimally normal weight for age and height (e.g., weight loss leading to maintenance of body weight less than 85% of that expected; or failure to make expected weight gain during period of growth, leading to body weight less than 85% of that expected).
>
> B. Intense fear of gaining weight or becoming fat, even though underweight.
>
> C. Disturbance in the way in which one's body weight or shape is experienced, undue influence of body weight or shape on self-evaluation, or denial of the seriousness of the current low body weight.
>
> D. In postmenarcheal females, amenorrhea, i.e., the absence of at least three consecutive menstrual cycles. (A woman is considered to have amenorrhea if her periods occur only following hormone, e.g., estrogen, administration.) (American Psychiatric Association, 2000, p. 589)

Attempts to characterize the presence of AN across time or across cultures have been marked by debates concerning the core features of the disorder. Although there is universal agreement that AN represents a disorder marked by starvation, some (Banks, 1992, 1997; Katzman & Lee, 1997; S. Lee, 1995; Palmer, 1993) have argued that weight phobia (Criterion B) represents an aspect of the culturally bound illness that is not necessarily related to the disease that underlies AN. Others (Beumont, 1988; Habermas, 1989) have argued that weight phobia is a necessary motivating force behind food refusal to distinguish AN from "other social (deprivation), physical or psychiatric causes of undernutrition" (Beumont, 1988, p. 171). Similarly, there have been debates over whether or not amenorrhea (Criterion D) should be retained as a diagnostic criterion or viewed as a consequence of malnutrition (Cachelin & Maher, 1998). Because the definition of AN continues to be contested, we included studies using various definitions of AN and noted how these definitions differed from that presented in the *DSM–IV–TR*.

Epidemiology of AN in the Twentieth Century

Table 1 provides reported incidence rates for AN from studies evaluating secular trends, organized by study cohort and presented in ascending order of year (r values and probability values were calculated by Pamela K. Keel as described below). Attempts to determine changes in incidence rates of AN have been plagued by the low base rate associated with the disorder and diagnostic uncertainty (Fombonne, 1995). Indeed, differences in diagnostic criteria have contributed to incidence rates differing more across studies than across time (see Table 1). Further, the definition of AN has changed over recent decades. Thus, studies relying solely on diagnoses recorded in case registers (e.g., Jones, Fox, Babigian, & Hutton, 1980; Kendell, Hall, Hailey, & Babigian, 1973; Møller-Madsen & Nystrup, 1992; Nielsen, 1990; Shinkwin & Standen, 2001; Williams & King, 1987) may be tracking decreasing stringency of diagnostic criteria for AN as well as secular trends for true incidence. To guard against this particular confound, several investigators (e.g., Eagles, Johnston, Hunter, Lobban, & Millar, 1995; Hall & Hay, 1991; Hoek et al., 1995; Joergensen 1992; Lucas, Beard, O'Fallon, & Kurland, 1988, 1991; Lucas, Crowson, O'Fallon, & Melton, 1999; Pagsberg & Wang, 1994; Szmukler, McCance, McCrone, & Hunter, 1986; Willi & Grossmann, 1983; Willi, Giacometti, & Limacher, 1990) have ensured application of uniform diagnostic criteria across years by directly evaluating medical records, interviewing clinicians and patients, or training clinicians to assess eating disorders.

Data coding. Across the epidemiological studies included in Table 1, 10 articles reported a statistically significant secular increase in AN incidence, 9 reported a failure to find a significant increase, and one reported a significant decrease. There was a clear relationship between study finding and inclusion of statistics required for meta-analyses. Only 2 studies (Lucas et al., 1988; Turnbull, Ward, Treasure, Jick, & Derby, 1996) that failed to find a significant secular increase provided specific data concerning either effect size or probability value. Thus, findings from 7 studies (Hall & Hay, 1991; Hoek et al., 1995; Joergensen, 1992; Nielsen, 1990; Szmukler et al., 1986; Willi et al., 1990; Williams & King, 1987) indicating at least one nonsignificant secular trend would be excluded from a meta-analysis of reported statistics. To avoid this bias, original data concerning annual incidence per 100,000 were recorded from each study from a combination of tables and figures. Tabulations of these raw data are available on request from Pamela K. Keel. For articles reporting annual incidence based on averaged data over a period of several years, this

Table 1
Anorexia Nervosa Incidence

Midpoint	Range	Location	Criteria	Reported incidence	*r*	*p*	Study
1945	1931–1960	Sweden	—	0.24	.948	.002	Theander (1970)
1962	1935–1979	Rochester, Minnesota	*DSM–III*, *DSM–III–R*	7.3[a]	.404	.109	Lucas et al. (1988)[b]
	1935–1984			8.2[a]			Lucas et al. (1991)[c]
	1985–1989			8.3[a]			Lucas et al. (1999)[c]
1968	1960–1969	New York	—	0.37	.501	.058	Kendell et al. (1973)[c]
	1960–1976			0.47			Jones et al. (1980)[c]
1968	1965–1971	London	—	0.66	.886	.004	Kendell et al. (1973)[c]
1970	1956–1975	Zurich, Switzerland	—	0.38–1.12	.822	.0005	Willi & Grossmann (1983)[c]
	1983–1985			1.43			Willi et al. (1990)[b]
1974	1966–1969	Scotland	Russell (1970)	1.6	.804	.000004	Kendell et al. (1973)[c]
	1965–1982			4.06			Szmukler et al. (1986)[b]
1977	1972–1981	England	—	—	.648	.0215	Williams & King (1987)[b]
1978	1965–1991	Northeast Scotland	ICD 8	—	.708	.00001	Eagles et al. (1995)[c]
1980	1970–1989	Bornholm County, Denmark	ICD 10	1.6–6.8	.815	.0925	Pagsberg & Wang (1994)[c]
1981	1977–1985	Ireland	ICD 8, ICD 9	4.18	−.935	.0001	Shinkwin & Standen (2001)[d]
1982	1970–1989	Denmark	ICD 8	0.42–1.17	.179	.087	Møller-Madsen & Nystrup (1992)[c]
	1973–1987			1.04			Nielsen (1990)[b]
	1970–1993			—			Munk-Jørgensen et al. (1995)[c]
1982	1977–1986	Fyn County, Denmark	*DSM–III–R*	11.0[e]	.004	.496	Joergensen (1992)[b]
1982	1977–1986	Wellington, New Zealand	*DSM–III*	5.0	−.230	.261	Hall & Hay (1991)[b]
1987	1985–1989	The Netherlands	*DSM–III–R*	8.1	.674	.212	Hoek et al. (1995)[b]
1989	1978–1992	Yamagata Prefecture, Japan	*DSM–III–R*	—	.805	.202	Nadaoka et al. (1996)
1991	1988–1993	England and Wales	*DSM–IV*	4.2[a]	.128	.405	Turnbull et al. (1996)[b]

Note. *DSM–III* = *Diagnostic and Statistical Manual of Mental Disorders* (3rd ed.); *DSM–III–R* = *Diagnostic and Statistical Manual of Mental Disorders* (3rd ed., rev.); ICD 8 = *International Classification of Diseases* (8th ed.); ICD 10 = *International Classification of Diseases* (10th ed.); ICD 9 = *International Classification of Diseases* (9th ed.); *DSM–IV* = *Diagnostic and Statistical Manual of Mental Disorders* (4th ed.).
[a] Age- and sex-adjusted incidence rate. [b] Reported no significant change. [c] Reported significant increase in incidence. [d] Reported significant decrease in incidence. [e] Incidence for females ages 10–24 years.

annual incidence was assigned to the year representing the midpoint of that period.

Pearson product–moment correlation coefficients were then calculated between incidence rates and years of observation within each cohort. These correlations along with their associated one-tailed probability values appear in Table 1. Cohorts rather than articles were selected as the unit of analysis because one article (Kendell et al., 1973) included three separate cohorts, two of which were followed later in subsequent articles (Jones et al., 1980; Szmukler et al., 1986). Similarly, continued follow-up has been reported in subsequent articles for two other cohorts (e.g., Hoek, 1991, Hoek et al., 1995; Lucas et al., 1988, 1991, 1999). Finally, incidences for overlapping periods have been reported for one cohort in three separate articles (Møller-Madsen & Nystrup, 1992; Munk-Jørgensen, Møller-Madsen, Nielsen, & Nystrup, 1995; Nielsen, 1990). Thus, conducting analyses by cohort rather than article ensured independence of observations.

One challenge in conducting the meta-analysis was variations across studies in how incidence was calculated. Williams and King (1987) demonstrated that the increased incidence for AN over time observed in their study was due to an increase in the proportion of the population comprising adolescent and young adult women. After age and sex were controlled for, no significant trend was found. Thus, age- and sex-adjusted incidence rates provide the best measure of secular trends, and studies were weighted according to their method of calculating incidence. A coefficient of 3 was given to studies that controlled for both sex and age. Studies controlling only for sex were given a coefficient of 2, and studies reporting incidence without controlling for age or sex were given a coefficient of 1. Because the significance of correlation values was influenced by the number of years of observation (which improves reliability of observed time trends) and not by the size of the population from which data were drawn, population size was used to weight values in the meta-analysis on the following scale: 1 = a population of 10,001 to 100,000; 2 = 100,001 to 1,000,000; and 3 = greater than 1,000,000. Thus, the meta-analysis favored findings from larger populations because they are less vulnerable to minor fluctuations in numbers of cases.

Results. Combining effect sizes across cohorts according to the equations presented in Rosenthal and Rosnow (1991) resulted in a medium effect for increased AN incidence across cohorts over time ($r = .35$). Combining probability values across cohorts according to the equations presented by Rosenthal and Rosnow indicated a significant increase in incidence over time ($p = .00005$, one-tailed). According to a formula presented in Rosenthal and Rosnow, approximately 205 nonsignificant studies would be required to alter the result of this meta-analysis.

Theander (1970) was the first author to publish evidence of increased incidence rates for AN; however, he noted many methodological reasons his findings might not reflect an increase in the true incidence of AN. These included "above all, increasing tendency to arrange medical care and hospitalisation for patients with anorexia nervosa" (Theander, 1970, p. 30). Of interest, during the period in which a significant increase in AN incidence was observed in Monroe County, New York (Jones et al., 1980; Kendell et al., 1973), a significant increase was observed for the number of

adolescents receiving psychiatric treatment in this region. Specifically, the incidence of AN rose from 0.35 to 0.64 per 100,000 population per year from 1960–1969 to 1970–1976 (Jones et al., 1980). Similarly, the incidence of children and adolescents using psychiatric services rose from 348 to 662 per 100,000 population per year from 1960 to 1976 (Roghmann, Babigian, Goldberg, & Zastowny, 1982). This suggests that cases of AN represented approximately 0.1% of children and adolescents receiving treatment from 1960 to 1976 with the apparent increase in AN incidence potentially reflecting the increase in adolescents being entered into the psychiatric case register. Supporting this possibility, Willi and Grossmann (1983) found that the percentage of those diagnosed with AN first hospitalized in psychiatry versus pediatrics or internal medicine increased steadily from 0% to 31.6% between 1956 and 1975. These authors attributed this trend to changes in how emaciation was interpreted in adolescent girls: "In the past, the cause of the disease was more frequently assumed to be organic (hypopituitarism), whereas in recent years, the diagnostic view of anorexia nervosa has changed from somatic to psychic causes" (Willi & Grossmann, 1983, p. 565). Secular increases in psychiatric contact for patients with AN have been reported by others (George, Weiss, Gwirtsman, & Blazer, 1987; Lucas et al., 1999). These patterns may explain why Szmukler et al. (1986) observed a secular increase in AN incidence ascertained through the Aberdeen Psychiatric Case Register in Scotland but observed no significant change in AN incidence ascertained through the general hospital register. Restricting our meta-analysis to just those investigations using general medical records indicates a small effect size ($r = .25$) that is statistically significant ($p = .03$) for an increase in AN incidence over the 20th century. Approximately 23 nonsignificant studies would be required to alter the results of this meta-analysis. Thus, a modest increase in AN incidence has coincided with increasing idealization of thinness (Garner et al., 1980; Owen & Laurel-Seller, 2000; Rubinstein & Caballero, 2000; Wiseman et al., 1992).

Introduction of AN to the medical nomenclature. Although popular and professional attention to AN has increased dramatically over recent decades (perhaps out of proportion with the actual increase in AN incidence), AN is not a "new" disorder that first appeared when Western culture emphasized the thin ideal. The term *anorexia nervosa* was first introduced in the medical literature by William Gull in 1874 to describe four cases of adolescent girls with deliberate weight loss, among whom three went on to achieve full weight recovery (Gull, 1874). Independent descriptions of a similar syndrome appeared under the labels *l'anorexie hysterique* (Laségue, 1873) and *anoressia* (Brugnoli, 1875, as cited in Habermas, 1992b; Ruggerio, Prandin, & Mantero, 2001) throughout Europe at that time. Similarly, at the end of the nineteenth century, American physicians were beginning to differentiate anorexia as a syndrome distinct from the larger category of hysteria (Vandereycken & Lowenkopf, 1990), and German physicians were differentiating AN from the larger category of neurasthenic disorders (Vandereycken, Habermas, Van Deth, & Meermann, 1991). A paper presented at a meeting of the South Australian Branch of the British Medical Association in 1882 documented two cases of anorexia in young women in Australia (Vandereycken & Beumont, 1990), and Kissyel reported a case of severe hysterical anorexia in an 11-year-old girl in Russia in 1894 (as cited in DiNicola, 1990b).

Both Gull (1868, 1874, 1888) and Laségue (1873) acknowledged the presence of several cases of anorexia prior to their providing the syndrome with a specific name. Laségue (1873) commented, "I wish to treat of a symptomatic complexus *too often observed* [italics added] to be a mere exceptional occurrence" (p. 265). Similarly, Gull (1888) stated his case was "an illustration of most of these cases" (p. 517) and in an earlier article referred to "young women emaciated to the last degree through hysteric apepsia" (Gull, 1868, p. 175). Thus, both the language used in these articles and the widespread attention and confirmation these works received from other physicians support that the condition was a familiar entity, although previously known by other names or viewed within a larger category of mental disorders.

The features of Gull's (1888) AN included significant weight loss, slow pulse rate, skin changes, and loss of menstruation with "perversions of the 'ego' being the cause and determining the course of the malady" (p. 517). Gull (1874) specifically noted the following: "The want of appetite is, I believe, due to a morbid mental state. I have not observed in these cases any gastric disorder to which the want of appetite could be referred" (p. 25). In a similar vein, Gull (1874) advised physicians to treat the illness with feeding despite the likely protests of the patient. Laségue (1873) viewed l'anorexie hysterique as a form of hysteria in which psychological distress was converted into food refusal motivated by "disgust" or "uneasiness after food, vague sensations of fullness, suffering, and gastralgia *post-prandium*" that "although hypothetical, is dreaded in advance" (p. 265). Laségue's description, based on eight cases, resembled present-day AN in that his patients were primarily women between the ages of 15 and 20 years. Both Gull's (1874) and Laségue's formulations of the syndrome focused on the food refusal itself; emaciation (Criterion A) and amenorrhea (Criterion D) were viewed as consequences of the syndrome. This resembles the current view of bradycardia and constipation as sequelae of AN rather than core features of the syndrome. Another distinction between the formulation of the illness by both Gull (1888) and Laségue versus present-day AN was the absence of mention of weight or shape concern.

Cross-Historical Evidence of AN

Historical cases of anorexia. Prior to the formal recognition of AN as a distinct syndrome, several reports in the medical literature suggest the presence of AN throughout the nineteenth century. Marce (1860) described a form of hypochondria in which "young girls, who at the period of puberty and after a precocious physical development, become subject to inappetency carried to the utmost limits" (p. 264). He noted that patients actively refused to eat in the absence of true gastric incapacity ("the stomach is well able to digest, and suffers only from want of food"; Marce, 1860, p. 265) and that the result is that "all trace of adipose tissue had disappeared, and the patients were reduced to skeletons" (p. 264). Marce emphasized that deliberate food refusal and resistance to treatment could lead to death in some cases; however, he also reported that patients could be cured if forcibly refed. From 1812 to 1917, W. L. L. Parry-Jones (1985) found evidence of 40 cases of AN among 36,000 hospital admissions in England, suggesting that 0.1% of psychiatric inpatients may have suffered from AN (similar to the rate reported from 1960 to 1976 in Monroe County, New York). Two cases were described in greater detail. One involved a young woman who was admitted to the Warneford

Asylum in 1831 who was emaciated from chronic food refusal. She was noted to be "delirious from debility rather than insane" (W. L. L. Parry-Jones, 1985, p. 97), and she died after 12 days from starvation. The second case involved a 26-year-old woman admitted in 1862 who was emaciated from food refusal. She was described as "sensible of all that is said to her" (W. L. L. Parry-Jones, 1985, p. 99), and she was ultimately discharged following a 7-month treatment of refeeding. In an 1840 text on women's diseases, Imbert characterized *anorexie nerveuse* by loss of appetite, refusal to eat, and emaciation (as cited in Vandereycken & Van Deth, 1994). Chipley (1860) described cases of extreme emaciation among females who actively refused to eat (*sitomania*) despite pangs of hunger and attributed the deliberate self-starvation to a desire for attention and notoriety.

Chipley's (1860) conclusion concerning the motivation behind sitomania overlaps with the motivations attributed to so-called fasting girls of the eighteenth and nineteenth centuries. Throughout this period, cases of fasting girls gained great attention in the popular media throughout America and Europe (Bemporad, 1996; Brumberg, 1989; Vandereycken & Van Deth, 1994). Typically, these cases involved adolescent girls who abruptly refused to eat. The cases drew a mixture of medical concern and religious awe. Particularly notorious cases attracted national and international attention with details of the fast reported in newspapers and skepticism about the miraculous fasts expressed by medical professionals in scholarly journals. Thus, in all recorded cases, food refusal leading to emaciation was associated with the secondary gain of fame. And, for some, continued food refusal was a lucrative occupation. Not surprisingly, this secondary gain attracted fraud. Cases involving proven deception include Anne Moore, Margaretha Weiss, Barbara Kremers, and Eva Vliegen (Bemporad, 1996; Brumberg, 1989; Vandereyeken & Van Deth, 1994). Some fasting girls were acclaimed as miracles for their ability to eat nothing yet remain completely healthy (including maintaining a healthy weight). Conversely, other fasting girls became extremely emaciated, in some cases leading to their deaths. Among these were Lina Finch (1886), Kate Smulsey (1885), and Lenora Eaton (1881), who all reportedly died of starvation before the age of 22 as a result of their food refusal (Brumberg, 1989).

Sarah Jacobs (1869), the "Welsh Fasting Girl," represents a particularly tragic case of self-starvation leading to death in a fasting girl (Bemporad, 1996; Brumberg, 1989; Vandereycken & Van Deth, 1994). She began to fast in 1867 at the age of 12. Her parents publicized her behavior with the support of a local reverend who confirmed the authenticity of the claims. She became a tourist attraction for the curious and a source of inspiration for religious pilgrims. Sarah Jacobs's fame attracted concern and skepticism from the medical profession. To resolve ongoing debate over her case, nurses from Guy's Hospital were dispatched to keep watch on the girl. As a condition of the watch, her parents insisted that she not be offered unsolicited food. Over the course of the week, Sarah Jacobs grew feeble and lost the ability to regulate her own body temperature. The nurses and supervising doctors attempted to end the watch and recommended refeeding, but her parents refused to end or modify the conditions of the test, and Sarah never requested food. After 10 days, she died of starvation (Brumberg, 1989; Vandereycken & Van Deth, 1994).

According to Brumberg (1989), Anglo American girls during the Victorian era were well-acquainted with the religious fasting of medieval saints, and St. Catherine of Siena's biography was included in inspirational books for girls. Indeed, the actions of Sarah Jacobs's parents seem to reflect a deep faith in a divine source of their daughter's food refusal. However, the growing field of psychiatry viewed these individuals as suffering from "nervous" conditions. Thus, fasting girls of the eighteenth and nineteenth centuries embraced the continuity between the religious medieval fasting of saints and their own behaviors. Meanwhile, psychiatrists endorsed the continuity between the extreme fasts leading to death among adolescent girls in the United States, England, France, and Germany and the newly identified syndrome of AN.

Between the seventeenth and eighteenth centuries, "miraculous maids" were girls between the ages of 14 and 20 who engaged in self-starvation, modeling themselves explicitly after ascetic medieval saints (Bemporad, 1996). From 1685 to 1710, Bliss and Branch (1960) found seven medical doctoral theses on anorexia, including one authored by Hardenus in 1703 that attributed the disorder to dysfunction of the gastric nerves as well as disturbances in the spirit and feelings (p. 8). Morton (1689) described *nervous atrophy* or a *nervous consumption* characterized by loss of appetite, extreme emaciation, amenorrhea, overactivity, and indifference to the condition (as cited in Morton, 1694, English translation). Morton (1694) detailed two cases in the first chapter of his thesis on consumption. Mr. Duke's 18-year-old daughter experienced amenorrhea and significant weight loss "like a Skeleton only clad with skin" (Morton, 1694, p. 9) that could not be attributed to tuberculosis or chlorosis. The patient engaged in "continual poring upon Books, to expose her self both Day and Night to the injuries of the Air" (Morton, 1694, p. 8). After initial compliance with treatment she "quickly tired with Medicines, she beg'd that the whole Affair might be committed again to Nature, whereupon consuming every day more and more, she was after three Months taken with a Fainting Fit and dyed" (Morton, 1694, p. 9). The cause of death is unclear; however, refeeding syndrome seems to be one plausible explanation (see Walsh, Wheat, & Freund, 2000, for a review of treatment complications in AN). The second case involved a 16-year-old boy whose loss of appetite was also attributed to "studying too hard," although he responded to treatment with a "Milk Diet," "Riding," and "Country Air" (Morton, 1694, p. 10).

For possible cases of AN in the sixteenth century, McSherry (1985) suggested that Mary, Queen of Scots (1542–1587), suffered from AN. However, too little information is included in the short report to examine the supposition. Conversely, a great deal has been written of fasting medieval religious ascetics (Bell, 1985; Bynum, 1987; Carroll, 1998; Rampling, 1985), and cases of self-starvation were recorded by hagiographers from the eighteenth century back to the twelfth century.

Bell (1985) reviewed the vitae of 261 saints who lived in the Italian peninsula from A.D. 1200 on for evidence of what he termed *holy anorexia*. Holy anorexia, like AN, involved food refusal resulting in emaciation overtly motivated by the belief that this reflected divine intervention. Of the 261 saints, approximately 170 had adequate information for determination of eating pathology, and Bell argued that over half of these 170 individuals demonstrated a pattern of holy anorexia, such as that presented by St. Catherine of Siena and St. Veronica.

St. Catherine of Siena entered a pattern of self-starvation at around 16 years of age that continued until her death in 1380 (at age 32 or 33). Her death was brought on by her refusal to consume food or water. Because St. Catherine was a prolific writer, more is

known about her internal experience of self-starvation than about any other historical case. She portrayed herself as feeling unable to eat and claimed that "I prayed continually and I pray to God and will pray that he will grace me in this matter of eating so that I may live like other creatures" (St. Catherine, as cited in Bell, 1985, p. 23). As with patients treated by Gull, (1874), Laségue (1873), and Marce (1860) St. Catherine refused to eat because she viewed herself as afflicted by an inability to eat. St. Veronica (Veronica Giuliani) began a pattern of self-starvation at age 18 that may have represented a relapse from a previous episode at age 15. Fellow nuns reported seeing St. Veronica sneaking into the kitchen and gorging herself on food when she thought no one else was around, and she was placed repeatedly in the infirmary where she was forced to eat and prevented from binge eating. Although St. Veronica wrote several diaries, little information is given concerning the motivation behind her food refusal. The closest revelation on this point is that she felt she was "in a race against all the other novices to show who loved God the most" (Bell, 1985, p. 71). In contrast to St. Catherine, St. Veronica ultimately achieved recovery sometime between her 30s and 50s and lived until the age of 67 (death 1727).

Despite the acceptance of divine intervention as the reason for self-starvation in these cases, Bell (1985) described in detail the repeated attempts of peers and superiors to induce eating in the fasting saints to help them avoid the sin of vainglory, an inability to engage in holy responsibilities, or the sin of suicide. According to Bell, the Reformation functionally removed self-starvation as a means of achieving holiness for women such that St. Clare of Assisi (death 1253) and St. Catherine of Siena (death 1380) were revered for their self-starvation whereas Sister Domenica (death 1553) and Catherine Vannini (death 1606) were viewed with significantly greater suspicion. Bynum (1987) argued for a plurality of meaning for self-starvation among the fasting saints throughout this period such that extreme asceticism was alternately viewed as a mark of God's grace, demonic possession, fraud, and illness within the lifetimes of all fasting saints, not just those following the Reformation. Bynum's (1987) text includes the following quote from St. Catherine's confessor: "If food was ever forced down her throat, intense pain followed, no digestion took place, and all that had been violently forced down was violently forced back again" (p. 168). The use of forced feeding emphasizes the ambivalence that St. Catherine's fasting met. According to Ruggiero et al. (2001), psychiatric interpretations of self-starvation in young women were published in Italy by Pietro di Abano (1270–1316), Alessandro Benedetti (1533), Simone Porta (1551), and Becarri (1745). Thus, self-starvation in pursuit of a religious ideal was not unambiguously sanctioned in medieval times any more than self-starvation in pursuit of a thin ideal is sanctioned in modern times.

Purported cases of AN prior to the twelfth century include St. Wilgefortis, who allegedly engaged in self-starvation resulting in emaciation and lanugo (fine, downy body hair usually restricted to the fetal stage of development) sometime between A.D. 700 to 1000 (Lacey, 1982). Lacey (1982) argued that the popularity of this legend reflected the presence of AN throughout the European countries in which the bearded saint was known by various names (St. Ontkommena, St. Kummernis, St. Livrade, St. Leberata, and St. Uncumber). Hajal (1982) reviewed the case of self-starvation in al-Mut'tazz billah, son of Khalifah, as a case of AN successfully treated by Bukhtishu'ibn Jibrail (870). Cases of self-starvation attributed to demonic possession or cured by exorcism were documented during the 5th and 8th centuries, and early Christianity offers a possible case of AN from the late 4th century in Blessila in which an individual died from self-starvation at the age of 20 (Bemporad, 1996; Lacey, 1982). However, these earliest cases are not without controversy. For example, Bynum (1987) characterized the description of St. Wilgefortis as having AN as a "bizarre communication to a British medical journal" (p. 194). Indeed, the story of St. Wilgefortis recounted by Lacey is open to numerous interpretations, among which AN is one possibility. Moreover, prior to the twelfth century, details become sparse, increasing the difficulty of interpreting these potential cases.

Interpretation of historical cases of anorexia. Although all of the above cases have been included in reviews of the history of AN, controversy exists concerning the validity of retrospectively diagnosing these women with AN. Much of the debate centers on the motivation behind food refusal, and limited information on this point is the greatest challenge to interpretation. Habermas (1989, 1996) has argued that modern-day AN extends to historical cases presented in the latter half of the nineteenth century by Gull (1874, 1888), Laségue (1873), and Marce (1860) but not to fasting found among medieval religious ascetics or Victorian-era females with hysteria or fasting girls. He argued that fasting among religious ascetics and Victorian females with hysteria was not AN because (a) fasting was used to heighten spirituality along with other forms of self-mortification, (b) fasting was associated with convalescence and paralysis, and (c) fasting was interpreted in religious terms both by the fasting women and by the clergymen and doctors who wrote of these cases: "if some of the fasting women had been worried about their body weight, it would have been a concept accessible also to the reporting clergymen and doctors" (Habermas, 1989, p. 261). Conversely, Habermas (1989) interpreted early cases of AN (Gull, 1888) and l'anorexie hysterique (Laségue, 1873) as being motivated by weight phobia despite a lack of documentation of this feature. He concluded that "it is plausible that weight concerns have been present in AN from its beginning on but have been overlooked by most German and British medical men" (Habermas, 1989, p. 269) because early accounts of AN provided no "plausible motivation" (p. 269) for self-starvation and patients with AN tend to hide their goal of losing weight and give other explanations for their refusal to eat such as loss of appetite, inability to eat, and stomachache. According to Habermas (1989), weight loss was the "secret motivation" (p. 269), allowing it to be a necessary, if hidden, feature for defining the presence of the disorder since its introduction in the latter half of the nineteenth century. The absence of this motivation in earlier cases makes them invalid in his view. However, Habermas's (1989) discussion leaves unclear why the presence of religious motivation would negate the presence of weight phobia (or vice versa) or why weight concerns would be a more accessible concept for medieval clergymen and doctors than it was for Gull (1874) or Laségue.

The presence of religious motivation does not preclude the presence of weight concerns as evidenced by modern cases of "spiritual starvation" (Banks, 1992, 1997; Bynum, 1988; Katzman & Lee, 1997; Morgan, Marsden, & Lacey, 2000). Cases presented by Banks (1992, 1997) involve women who fasted to engender spiritual purity and thus bear a striking resemblance to historical cases of fasting performed by medieval religious ascetics. In one case, the syndrome resulted in death despite numerous hospitalizations. In the second case, the syndrome remitted and full recov-

ery was achieved. In terms of outcome, these cases are not unlike those of St. Catherine and St. Veronica, respectively. Morgan et al. (2000) reported on three cases of spiritual starvation. Although the authors indicated that *DSM–IV* AN was diagnosed in two of these cases, religious themes appeared to provide primary motivation for deliberate self-starvation in all three cases (Morgan et al., 2000).

It seems unlikely that Gull (1874) and Laségue (1873) overlooked weight concerns as a motivation for food refusal because contemporary authors noted these features in fasting girls. Winslow (1880) provided the following quote attributed to Erasmus Darwin from the end of the eighteenth century:

> Some young ladies I have observed to fall into this general debility but so as but just to be able to walk about, which I have sometimes ascribed to their voluntary fasting, when they believed themselves too plump; and who have thus lost both health and beauty by too great abstinence, which could never be restored. (p. 281)

Similarly, Charcot emphasized the role of weight phobia in the etiology of self-starvation among his patients (as cited in Habermas, 1989; Vandereycken & Van Deth, 1994). The work of both Winslow and Charcot would have been available to Gull and Laségue, suggesting that both men could be aware of this possible explanation for self-starvation. However, neither seemed to regard this information as a compelling explanation for food refusal in their patients. In a later publication, Habermas (1996) elaborated his argument that weight phobia represents the central feature of AN on the basis that diagnostic criteria have to differentiate syndromes in such a way that a syndrome represents a homogeneous group distinct from other syndromes. Although weight phobia achieves this end, it does not appear to be the only means to this end.

Given both current (as well as historical) plurality in motivations behind self-starvation, Rieger, Touyz, Swain, and Beumont (2001) recommended requiring ego-syntonic emaciation rather than weight phobia to differentiate AN from other conditions that lead to weight loss. The authors noted that cases of AN are uniform in the extent to which self-starvation is deliberate and the responses to resulting emaciation range from indifference to pride. Their proposition offers a solution to differentiating fasting to convey religious devotion that is common to most religions (Bliss & Branch, 1960) and that displayed in holy anorexia. Moreover, ritualistic religious fasting is time-limited and tends to focus on avoiding specific foods entirely or not eating during specific time intervals and thus does not typically result in emaciation. It lacks the persistent and pervasive nature of food refusal that characterizes the form of fasting observed in medieval religious ascetics or modern-day AN. In addition, weight loss that results from other physical and psychological conditions is often distressing to the affected individual (Rieger et al., 2001).

Despite the utility of Rieger et al.'s (2001) recommendation, there are other contemporary cases in which ego-syntonic emaciation would not represent an eating disorder or any other form of psychopathology. For example, deliberate self-starvation may be used as an effective means of protest. Although women with AN can be seen as using self-starvation as a means of protest (Brumberg, 1989), this can be differentiated from nonpathological forms of deliberate self-starvation. Individuals who engage in hunger strikes do so as a means to an end. When the end is reached, the self-imposed starvation ceases. Mogul (1980) wrote, "What distinguishes adaptive asceticism from pathological states is not so much its extent, or even the subjective experience of gratification from it, but the degree to which the asceticism becomes an end in itself" (pp. 159–160). Food refusal has been used as a means to different ends in different periods (moral purity, fame, attention, thinness), but in all periods it also becomes an end in itself. What seems to unite fasting saints and women with AN is the paradox that the starvation is both deliberate and nonvolitional. That is, across historical contexts women deliberately refuse to eat food that they require for sustenance. Yet, they do not appear to be able to stop their pattern of food refusal in response to reward or punishment. This characterization resembles Shafran, Cooper, and Fairburn's (2002) depiction of clinical perfectionism as the core pathology of AN. The internal motivation to avoid eating overrides all internal and external drives to eat, and for many adolescent girls throughout history this means becomes their ultimate end.

Although motivations for food refusal may have differed across periods (in many cases this information is simply lacking), purported motivations may not represent the true causes of self-starvation. Instead, they may represent culturally meaningful attempts to understand an affliction that leaves women feeling unable and unwilling to eat. The extent to which fear of fat is viewed as causing AN may be, in part, an illusion (Wegner, 2002). Thus, if the core feature of AN is taken to be an intentional yet nonvolitional self-starvation, then evidence of AN appears to trace back to early medieval times. Furthermore, across the various historical contexts in which AN has emerged, this disorder demonstrates a particular affinity for affecting females beginning in their adolescence and thus satisfies Habermas's (1996) call for defining the core features of a syndrome such that they retain descriptive specificity.

Cross-Cultural Evidence of AN

Although several reports have suggested that AN was nonexistent in non-Western regions such as sub-Saharan Africa (German, 1972), Northern Sudan (Elsarrag, 1968), and Southeast Asia (Neki, 1973), our review of the literature indicated that AN has been observed in every non-Western region of the world. Table 2 summarizes features of reports of AN in non-Western nations, including the country, diagnostic criteria, number or prevalence of cases, presence of weight concern among these cases, and level of Western influence. These publications included case reports, letters to journal editors, and clinical and epidemiological investigations.

Data coding. Presence of weight concerns was evaluated because of the evidence reviewed above that this symptom varies both across and within historical periods. For studies indicating that all cases met diagnostic criteria that require weight concerns and for which details for specific case histories were not provided, we concluded that all cases (e.g., 100%) must have reported weight concern. Unfortunately, this did not preclude an inconsistent or idiosyncratic application of diagnostic criteria in those studies. By contrast, for cases in which weight concerns were explicitly denied by the patient but were perceived by the author to be the secret motivation, we considered the feature to be absent. Some of these cases may resemble patients with AN who have denied fears of becoming fat only for the fears to emerge as weight restoration begins. However, inferring the presence of weight concerns in the absence of any confirming evidence prevents awareness of cases in which this feature is genuinely absent. Further, it runs the risk of

relying on an ethnocentric conception of the core features of AN (S. Lee, Ho, & Hsu, 1993).

For characterization of Western influence, we took authors' estimation of the degree of Western influence when possible. When authors made no comment on this issue, we used all available information to code the study appropriately. We developed six categorizations in this regard: (a) No—at least one reported case where the author specifically noted that the case lacked exposure to Western influence (e.g., Abou-Saleh, Younis, & Karim, 1998, described a case of AN in an 18-year-old nomadic woman from the Empty Quarter in the United Arab Emirates and specifically noted the absence of Western influence in this case); (b) Unlikely—at least one case likely meets all of the following criteria: comes from a nonurban region, is of low socioeconomic status, resides within a traditional family, has not surpassed secondary education, *and* is non-English speaking; (c) Probable—all cases likely meet at least one of the following criteria: reside in an urban setting, are of high socioeconomic status, are English-speaking, *or* have higher educational attainment (i.e., baccalaureate or higher); (d) Definite—all cases spent considerable time in a Western nation before developing eating pathology (e.g., Ford, 1992, reported a case of BN in an Egyptian individual who developed the disorder after spending ages 5–17 in Canada); (e) Indeterminable—not enough information was provided to determine the degree of Western influence (e.g., data come from patients treated in an urban medical center; however, it is not possible to determine if any patients would fit above criteria for unlikely or no Western influence); and (f) Unknown—the original article was not written in English and information on Western influence was not included in the English abstract or English review article from which data were taken. Both the presence of weight concerns and Western influence were coded by consensus between Pamela K. Keel and Kelly L. Klump.

Results. Although food refusal leading to emaciation was reported for all cases in Table 2, the presence of weight concerns as a motivating factor for food refusal does not appear to be universal. S. Lee et al. (1993) described 70 cases of AN in Hong Kong among whom 59% did not report weight concerns. As with the cases described by Laségue (1873) and Gull (1874), digestive discomfort was the most frequent reason for not eating. These data suggest that Westernization and industrialization bring about certain aspects of AN (weight concerns) but are not necessary for producing a self-starvation syndrome. Alternatively, as has been argued for historical cases, weight concerns may have been present but denied by some patients. S. Lee et al. (1993) predicted that Western influence in China would result in increased presentation of weight-phobic AN. Consistent with this hypothesis S. Lee (2000) and Lai (2000) have both described an increasing prevalence of AN characterized by body dissatisfaction in the East. Further, S. Lee and Lee (2000) found that weight concerns were greatest in Hong Kong and least prominent in Hunan, suggesting an association between Westernization and body image disturbance.

Although some authors have indicated that AN cases were limited to the social elite or higher socioeconomic classes (Buchan & Gregory, 1984; Chadda, Malhotra, Asad, & Bambery, 1987), several reports suggested the presence of AN in the absence of Western influence (Abou-Saleh et al., 1998; Chandra et al., 1995; Fahy, Robinson, Russell, & Sheinman, 1988; Gandhi, Appaya, & Machado, 1991). When Western exposure could be determined, 42% (8/19) of articles reported at least one case with no or unlikely exposure to Western ideals.

Epidemiological data not only support the existence of AN in non-Western countries but suggest that its prevalence may be similar to that in Western nations. The lifetime prevalence of *DSM–III* AN in Korea did not differ from estimates produced by the Epidemiological Catchment Area study for lifetime prevalence of AN in New Haven or St. Louis (C. K. Lee et al., 1987, 1990). A recent study (Nobakht & Dezhkam, 2000) reported the lifetime prevalence of AN to be 0.9% in Iranian schoolgirls, a value that is higher than the lifetime prevalence reported in the *DSM–IV–TR*, and the incidence and prevalence of AN in Hong Kong (Chen et al., 1993; S. Lee, Chiu, & Chen, 1989) and Japan (Azuma & Henmi, 1982; Kuboki, Nomura, Ide, Suematsu, & Araki, 1996) are similar to those reported in Western nations (e.g., Kendell et al., 1973; C. K. Lee et al., 1987; Møller-Madsen & Nystrup, 1992; Theander, 1970, Willi & Grossmann, 1983). According to reports from Malaysia (Buhrich, 1981; Goh, Ong, & Subramaniam, 1993) and Egypt (Okasha, Kamel, Sadek, Lotaif, & Bishry, 1977), AN constituted between 0.05% and 0.19% of psychiatric cases. These figures are roughly comparable to those reported for Norway from 1990–1994 (Götestam, Eriksen, Heggestad, & Nielsen, 1998). Thus, excluding the criterion of weight concerns, AN appears to represent a similar proportion of the general and psychiatric populations in several Western and non-Western nations.

Summary of Findings for AN

It does not appear to be the case that AN is a culture-bound syndrome. Although cultural factors such as the increasing idealization of thinness may influence rates of AN, such factors seem neither sufficient nor necessary. Moreover, evidence of a secular increase in AN incidence is modest after controlling for factors such as changes in population distribution, population size, and ascertainment of cases through psychiatric registers. Cases of deliberate yet nonvolitional self-starvation, sometimes resulting in recovery, sometimes resulting in death, and primarily affecting young adolescent girls, exist in numerous historical periods. Similarly, specific cases of AN that cannot be attributed to the influence of Western ideals have been reported in the Middle East, the Indian subcontinent, and East Asia. Like historical cases that emerged in the absence of the "modern 'cult of thinness' " (Russell, 1997, p. 23), many of these non-Western cases lack weight concerns. Our results suggest that weight concerns may be a culturally bound phenomenon, restricted to sociohistorical contexts that idealize thinness and denigrate fatness. However, this is not entirely accurate either because a plurality of motivations for food refusal has been described both across and within sociohistorical contexts.

Bulimia Nervosa

The most recent edition of the *DSM* (*DSM–IV–TR*) defines BN with the following diagnostic criteria:

> A. Recurrent episodes of binge eating. An episode of binge eating is characterized by both of the following:
>
> (1) eating, in a discrete period of time (e.g., within any 2-hour period), an amount of food that is definitely larger than most people would eat during a similar period of time and under similar circumstances

Table 2
Studies of Anorexia Nervosa (AN) in Non-Western Nations

Article	Place	Diagnostic criteria	% weight concern	Number–prevalence	Western influence?
			Africa		
Norris (1979)	Johannesburg, South Africa	Study specific	100	54 patients with AN; 8 Afrikaans-speaking and 4 other non-English-speaking patients	Indeterminable
Nwaefuna (1981)	Nigeria	Not given	100	1 case described in a Black Nigerian woman	Indeterminable
Famuyiwa (1988)	Nigeria	Not given	0	2 suspected cases reported; symptoms were loss of > 30% body weight, denial of seriousness of low weight, and amenorrhea	Case 1: definite; Case 2: indeterminable
Buchan & Gregory (1984)	Zimbabwe	Feighner	Questionable case[a]	1 case reported in Black Zimbabwean	Definite
Fahy et al. (1988)	Ethiopia	Not given	100	1 case reported in Ethiopian torture victim	Probable
			Middle Eastern–Arab cultures		
Okasha et al. (1977)	Cairo, Egypt	Not given	Not given	2 cases of AN; 0.19% of psychiatric cases	Probable
Abou-Saleh et al. (1998)	United Arab Emirates	*DSM–III–R* for 3 of 5 patients	80	5 cases reported, among which diagnoses of *DSM–III–R* AN were confirmed by interview for 3 cases; 1 case (20%) lacked exposure to Western influences	No
Nobakht & Dezhkam (2000)	Iran	*DSM–IV*	100	0.9% of 3,100 female high school students age 15–18	Probable
Yager & Smith (1993)	Pakistan	Not given	100	1 case reported	No
			Indian subcontinent		
Neki, Mohan & Sood (1977)	India	Study specific	Not given	2 cases who were monozygotic twins	Indeterminable
Chakraborty (1985)	India	Not given	Absent in 8–10 cases	15–17 cases reported	Indeterminable
Chadda et al. (1987)	India	*DSM–III*	100	1 case of AN from the upper social class in India	Probable
Gandhi et al. (1991)	India	Not given	60	5 cases reported	No
Khandelwal et al. (1995)	India	Modified Feighner	20	5 cases reported	Unlikely
Chandra et al. (1995)	India	Modified *DSM–III–R*	33	3 cases reported	No
			Southeast Asia		
Ong et al. (1982)	Malaysia	Not given	71	7 cases reported—1 case with no formal education from lowest social class	Unlikely
Buhrich (1981)	Malaysia	Study specific	Not given	30 cases reported; 19 Chinese, 8 Indian, 1 Malaysian, 2 Eurasian; no change reported in prevalence over 15- to 20-year period; 0.05% of psychiatric referrals	Indeterminable
Krahl et al. (1981)	Malaysia	Unknown	Unknown	2.5% of cases presenting to a community child guidance clinic	Unknown
Krahl (1983)[b]	Malaysia	Unknown	Unknown	2 cases reported	Unknown
Goh et al (1993)	West Malaysia	Study specific	60	15 cases of partial AN in 9,000 female patients from 1970–1988; 0.16% of female patients	Indeterminable
Ung, Lee, & Kua (1997)	Malaysia	Not given		33 patients with AN; 36% partial syndrome patients with "lack of amenorrhea in anorexic range" (p. 334)	Indeterminable
			East Asia		
S. Lee, Chiu, & Chen (1989)	Hong Kong	Modified *DSM–III–R*	67	Reported < 10 cases treated during 5 years (incidence < 0.4 per 100,000 person years); 3 cases described in detail	Indeterminable
Chiu (1989)	Hong Kong	*DSM–III*	100	1 case reported	No
S. Lee (1991)	Hong Kong	Modified Feighner	19	16 cases reported	Unlikely
S. Lee, Leung et al. (1991)	Hong Kong	Study specific	0	2 cases reported	Indeterminable
S. Lee, Ho, & Hsu (1993)[c]	Hong Kong	Study specific	41	70 cases reported	Indeterminable

Table 2 (*continued*)

Article	Place	Diagnostic criteria	% weight concern	Number–prevalence	Western influence?
			East Asia (*continued*)		
Chen et al. (1993)	Hong Kong	*DSM–III*	100	0.03 lifetime prevalence of AN in 7,229 males and females surveyed	Probable
Lai, Pang, & Wong (1995)	Hong Kong	Not given	100	1 case reported	Probable
Kam & Lee (1998)	Hong Kong	Study specific	50	1 case reported	Indeterminable
S. Lee, Lee, & Leung (1998)	Hong Kong	"Broadly diagnosed"	58	26 cases of AN from case series treated from 1990–1996	Probable
Lai (2000)	Hong Kong	Lask (1992)	88	16 cases reported	Probable
S. Lee (2001)	Hong Kong	Modified *DSM–IV*	67	48 cases reported	Indeterminable
Song & Fang (1990)[d]	China	*DSM–III*	Unknown	9 cases reported	Unknown
Tseng et al. (1989)[e]	Taiwan	*DSM–III*	Unknown	7 cases reported	Unknown
Hung & Cheng (1992)[f]	Taipei, Taiwan	*DSM–III–R*	100	2 cases of AN reported among 12,435 females ages 12–25	Indeterminable
Sonoda et al. (1974)[e]	Japan	Unknown	Unknown	1 case reported in a 10-year-old girl	Unknown
Nogami & Yabana (1977)[g]	Japan	Not given	Not given	9 cases reported	Indeterminable
Azuma & Henmi (1982)[h]	Japan	Unknown	Unknown	0.2% urban areas; 0.05% rural areas	Unknown
Mizushima & Ishii (1983)	Japan	Study specific	100	16 girls and 1 boy with AN among 36,403 girls and 37,520 boys in secondary school	Unknown
Suematsu et al. (1985)	Japan	Study specific	75% of 1,011	2,392 patients treated in 1980–1981; detailed data collected from 1,011 cases	Indeterminable
Suematsu et al. (1986)	Japan	Study specific	100	7 cases reported	Indeterminable
Kuboki et al. (1996)	Japan	Modified *DSM–III–R*	100	2.9–3.7/100,000 general population 1985; 3.6–4.5/100,000 general population 1992	Indeterminable
Nadaoka et al. (1996)	Japan	*DSM–III–R*	100	59 cases of AN treated at Yamagata University Hospital between 1978 and 1992	Probable
Nakamura et al. (2000)	Japan	*DSM–IV*	100	0.005% of female population; 0.003% of general population	Indeterminable
Takei et al (2000)[e]	Japan	*DSM–IV*	100	223 students with AN from 335 college health administration centers across Japan	Unknown
C. K. Lee et al. (1987)	Korea	*DSM–III*	100	0.02% of general population	Indeterminable
C. K. Lee et al. (1990)	Korea	*DSM–III*	100	0.03% of general population	Indeterminable

Note. Not given = authors did not provide the information in the article; Unknown = authors may have provided information in the non-English publication, but the information was not provided in the English abstract or the English review article summarizing it; Study specific = diagnostic criteria were specific to the study indicated; Indeterminable = exposure to Western influence could not be determined from article; *DSM–III–R = Diagnostic and Statistical Manual of Mental Disorders* (3rd ed., rev.); *DSM–IV = Diagnostic and Statistical Manual of Mental Disorders* (4th ed.); *DSM–III = Diagnostic and Statistical Manual of Mental Disorders* (3rd ed.).

[a] Report states "score on the Eating Attitudes Test (Garner & Garfinkel, 1979) was 78—clear evidence of her terror of becoming overweight" (Buchan & Gregory, 1984, pp. 328–329). However, initial weight loss does not seem intentional, and case was characterized by many psychotic and dissociative symptoms. [b] As reported in DiNicola (1990b). [c] 16 (23%) participants also took part in the S. Lee (1991) study. [d] As reported in S. Lee, Ho, and Hsu (1993). [e] Data were taken from an English abstract of a non-English publication. Overlap of cases across reports from a single country is unknown. [f] As reported in Tsai (2000). [g] As reported in Davis and Yager (1992). [h] As reported in Dolan (1991).

> (2) a sense of lack of control over eating during the episode (e.g., a feeling that one cannot stop eating or control what or how much one is eating)
>
> B. Recurrent inappropriate compensatory behavior in order to prevent weight gain, such as self-induced vomiting; misuse of laxatives, diuretics, enemas, or other medications; fasting; or excessive exercise.
>
> C. The binge eating and inappropriate compensatory behaviors both occur, on average, at least twice a week for 3 months.
>
> D. Self-evaluation is unduly influenced by body shape and weight.
>
> E. The disturbance does not occur exclusively during episodes of Anorexia Nervosa. (American Psychiatric Association, 2000, p. 594)

Although the definition of BN has been revised since its introduction to the psychiatric nomenclature, the above definition bears a striking resemblance to that given by Russell (1979) in his seminal description of the disorder. Slightly earlier, independent reports of a binge–purge syndrome in normal-weight young women were published in German and Spanish (Ziolko, 1996) and have since been translated into English by the original authors (Doerr-Zegers, 1994; Ziolko, 1994). Notably, among these first descriptions of BN (Doerr-Zegers, 1994; Russell, 1979; Ziolko, 1994), no author described a large awareness of this syndrome before 1960. Based on BN's recent appearance and dramatically increasing incidence, Russell (1997) concluded, "Bulimia nervosa is a new disorder" (p. 23).

Epidemiology of BN in the Twentieth Century

Table 3 provides incidence rates for BN from studies evaluating secular trends, organized by study cohort and presented in ascend-

Table 3
Bulimia Nervosa Incidence

Midpoint	Range	Location	Criteria	Reported incidence	*r*	*p*	Study
1980	1970–1989	Bornholm County, Denmark	ICD 10	0.7–3.0	.905	.0475	Pagsberg & Wang (1994)
1982	1977–1986	Fyn County, Denmark	*DSM–III*	5.5[a]	.618	.0285	Joergensen (1992)
1982	1977–1986	Wellington, New Zealand	*DSM–III*	6.0–44.0	.869	.0005	Hall & Hay (1991)
1985	1980–1990	Rochester, Minnesota	*DSM–III–R*	13.5[b]	.347	.1475	Soundy et al. (1995)
1987	1985–1989	The Netherlands	*DSM–III–R*	11.5	.828	.042	Hoek et al. (1995)
1989	1978–1992	Yamagata Prefecture, Japan	*DSM–III–R*	—	.863	.1685	Nadaoka et al. (1996)
1991	1988–1993	England and Wales	*DSM–IV*	12.2	.968	.001	Turnbull et al. (1996)

Note. ICD 10 = *International Classification of Diseases* (10th ed.); *DSM–III* = *Diagnostic and Statistical Manual of Mental Disorders* (3rd ed.); *DSM–III–R* = *Diagnostic and Statistical Manual of Mental Disorders* (3rd ed., rev.); *DSM–IV* = *Diagnostic and Statistical Manual of Mental Disorders* (4th ed.).
[a] Incidence for females ages 10–24 years. [b] Age- and sex-adjusted incidence rate.

ing order of year. Among these studies, all reported a significant rise in cases over time (Hall & Hay, 1991; Hoek et al., 1995; Joergensen, 1992; Nadaoka et al., 1996; Pagsberg & Wang, 1994; Soundy, Lucas, Suman, & Melton, 1995; Turnbull et al., 1996). Of interest, several of these studies found a significant increase in BN incidence despite finding no significant change in AN incidence (Hall & Hay, 1991; Hoek et al., 1995; Turnbull et al., 1996). Combining results across the seven studies that provide BN incidence data over time (Hall & Hay, 1991; Hoek et al., 1995; Joergensen, 1992; Nadaoka et al., 1996; Pagsberg & Wang, 1994; Soundy et al., 1995; Turnbull et al., 1996) weighted by method of calculating incidence and population size (as described above for the meta-analysis of AN incidence data), we found a large effect size ($r = .89$) for a secular trend of increasing BN incidence that is statistically significant ($p = .0000001$). Approximately 62 nonsignificant studies would be required to alter the results of this meta-analysis. Restricting our meta-analysis to include only those studies that used general medical records also results in a large effect size ($r = .90$) that is statistically significant ($p = .00002$). Approximately 28 nonsignificant results would be required to alter the results of this meta-analysis. Thus, studies tracking a secular trend in BN incidence from 1970 to 1993 suggest a significant increase.

Fombonne (1996) argued that the patterns seen for BN frequency follow that expected for a newly defined disorder but do not necessarily represent the emergence of a previously nonexistent syndrome. Specifically, one would expect to see a significant increase in incidence immediately following official recognition of a disorder because incidence rates often reflect when individuals first seek treatment for a disorder. Thus, incidence rates are inflated initially by the combination of new onset cases and cases with onset prior to disorder recognition. This description matches changes in incidence rates reported by Soundy et al. (1995). Incidence of BN increased steeply from 7.4 to 49.7 per 100,000 females from 1980 to 1983 and then remained relatively stable around 30 per 100,000 females from 1984 to 1990. According to Fombonne (1996), the decline from 49.7 to 30 cases per 100,000 females per year reflected a decline in cases with onset before the disorder had been defined rather than a true decrease in disorder incidence. Conversely, Soundy et al. noted that the particularly high rate in 1983 was inflated by a BN treatment study that year for which participants were being actively recruited from the community. Unfortunately, there are no incidence data for BN prior to 1970. Thus, one cannot use incidence rates to determine how the frequency of this disorder changed in relation to the current cultural idealization of thinness as was possible for AN.

Despite the ever-present cultural ideal of thinness, BN prevalence estimates were reduced from the *DSM–III* to the *DSM–III–R*. Early studies of BN point prevalence suggested that the disorder affected 8% (Pyle, Halvorson, Neuman, & Mitchell, 1986; Zuckerman, Colby, Ware, & Lazerson, 1986) to 19% (Halmi, Falk, & Schwartz, 1981) of university women whereas later studies suggested a lower point prevalence of BN among college women with estimates ranging from approximately 1% (Schotte & Stunkard, 1987) to 3% (Drewnowski, Yee, & Krahn, 1988). Although these results suggest a decline in the point prevalence of BN running counter to the continued idealization of thinness for this time (Wiseman et al., 1992), several methodological factors, unrelated to changing prevalence, may account for the observed differences.

First, earlier studies used the *DSM–III* criteria (Halmi et al., 1981; Pyle et al., 1986; Zuckerman et al., 1986) whereas later studies used *DSM–III–R* criteria (Drewnowski et al., 1988; Schotte & Stunkard, 1987). Between the *DSM–III* and *DSM–III–R*, the diagnostic criteria for BN grew more stringent. For example, recurrent inappropriate compensatory behavior was required in the *DSM–III–R* but was not strictly required in the *DSM–III*. In addition to being generally less stringent, the *DSM–III* was also less specific. For example, although the *DSM–III* criteria required "recurrent" binge-eating episodes, it did not specify a minimum frequency required for the episodes to be considered recurrent. Zuckerman et al. (1986) demonstrated that the simple stipulation of bingeing more than once per week versus just once per week reduced the point prevalence estimate of bulimia from 8% to 4% for college women in 1982. Similarly, Pyle et al. (1986) reported that the point prevalence of *DSM–III* bulimia in college women was approximately 8% in both 1980 and 1983; however, when weekly binge eating and purging were required, the point prevalence dropped to 1% in 1980 and 3% in 1983. Of interest, the stricter definition resulted in a threefold increase in the point prevalence of bulimia among college women from 1980 to 1983—mirroring results of incidence studies for this period. Thus, had *DSM–III–R* criteria been used in the earliest studies, prevalence estimates might have ranged from 1% to 4%.

Second, different methods of assessment across studies over time may have contributed to the perceived decrease in prevalence rates. Early prevalence studies (Halmi et al., 1981; Pyle et al., 1986; Zuckerman et al., 1986) more often used survey assessments, and later studies more often used interview assessments

(Bushnell, Wells, Hornblow, Oakley-Browne, & Joyce, 1990; Schotte & Stunkard, 1987). Self-report surveys tend to produce higher estimates of BN prevalence than structured clinical interviews (Keel, Crow, Davis, & Mitchell, 2002). Bulimic symptoms such as binge eating are particularly vulnerable to high false-positive rates on self-report surveys compared with interviews because lay definitions tend to be more inclusive than clinical definitions (Whitehouse, Cooper, Vize, Hill, & Vogel, 1992). In addition, women appear to be more willing to admit to bulimic symptoms on self-report surveys compared with interviews because the increased anonymity of questionnaires increases candor (Keel et al., 2002). Both patterns could contribute to an artificial decline in BN prevalence over time.

Third, differences in the samples evaluated have led to varying prevalence rates (Fairburn & Beglin, 1990). Few studies have used random sampling, and inconsistent sampling strategies across studies could influence the extent to which individuals with eating disorders are over- or underrepresented in study groups. Moreover, epidemiological samples have varied in response rates, socioeconomic status, geographic location, ethnic representation, and age. Thus attempts to determine time-related changes in prevalence could be confounded by any of these differences. For example, women ages 18 to 24 (approximately college-aged) have rates of BN that are 2 to 10 times greater than those reported for women ages 25 to 64 (Bushnell et al., 1990).

An alternative to examining prevalence rates over different time periods is to measure lifetime prevalence rates across birth cohorts at one time. If a disorder has a stable incidence and onset occurs throughout life, then lifetime prevalence rates increase as birth cohorts age. If a disorder has a stable incidence but onset is limited to early ages (as it is for eating disorders), then lifetime prevalence rates would remain stable across birth cohorts that had passed through the period of risk. However, as noted above, lifetime BN prevalence rates are actually higher among younger birth cohorts compared with older birth cohorts (Bushnell et al., 1990; Kendler et al., 1991). These findings suggest either that the risk of BN has increased over recent years or that recall of past episodes of BN diminishes with age. Of interest, lifetime prevalence of AN has not differed significantly across birth cohorts (Robins et al., 1984; Wade, Heath, Abraham, & Treloar, 1996). Similarly, in an examination of eating disorders among relatives of AN probands, rates of BN were higher among sisters compared with mothers (4.1% vs. 1.0%), consistent with lifetime prevalence rates for a disorder with increasing incidence rates (Strober, Lampert, Morrell, Burroughs, & Jacobs, 1990). Conversely, rates of AN were 3 times lower in sisters than in mothers (2.0% vs. 6.2%), consistent with lifetime prevalence rates for a disorder with stable incidence rates (Strober et al., 1990). Unfortunately, data on lifetime prevalence rates do not inform us regarding when the disorder developed relative to the cultural ideal of thinness.

There is evidence of changing rates of BN during the latter half of the 20th century; however, the causes of these changes are unclear. Incidence data suggest a significant increase in BN incidence from 1970 to 1990 but do not allow evaluation of BN incidence prior to 1970. Prevalence studies in college and primary care samples suggest a decreasing prevalence from the 1980s to the 1990s; however, inconsistent methods across prevalence studies obfuscate the meaning of this observation. Finally, lifetime prevalence data across birth cohorts support an increasing incidence. Taken together, these data suggest that BN emerged as a new syndrome during the latter half of the twentieth century. However, it is possible that perceived trends resulted from the formal recognition of a disorder that had existed as a hidden form of psychiatric morbidity in earlier periods.

Cross-Historical Evidence of BN

Historical cases of bulimia. Several authors have reviewed historical cases of BN prior to its formal recognition (Habermas, 1989; B. Parry-Jones, 1991; B. Parry-Jones & Parry-Jones, 1991; Pope, Hudson, & Mialet, 1985; Stein & Laakso, 1988; Ziolko, 1996). The case histories of Ellen West (Binswanger, 1994, as cited in Stein & Laakso, 1988; Binswanger, 1958, as cited in Pope & Hudson, 1988) and Laura (Lindner, 1954, as cited in Stein & Laakso, 1988) have been interpreted as examples of BN in the earlier half of the twentieth century. However, several experts in the field of eating disorders have reviewed Ellen West as representing AN, and it is unclear that binge eating and purging ever occurred at normal weight in her case (DiNicola, 1990a; Habermas, 1992a). Cases described by Wulff (1932, as cited in Habermas, 1989 and Stunkard, 1990), Bergmann, (1932, as cited in Habermas, 1992a), and Feuchtinger (1942, as cited in Habermas, 1992a) in the German psychiatric literature have been characterized by binge eating and purging. According to Habermas (1989), Wulff's cases included four women and one man, all of whom experienced periods of compulsive eating at normal weight. In three cases, age of onset was during puberty. In two cases, vomiting was present in addition to dieting. Finally, in three cases, body image disturbance was recorded. However, a different picture emerges from Stunkard's (1990) translation of Wulff's work. First, Wulff described marked obesity in two of four cases (Cases A and C). Further, in three of the four cases (Cases A, B, and C), binge eating and fasting or purging occurred in distinct phases. In Case A, for example, the periods of binge eating, hypersomnia, and lack of personal hygiene would persist for months without attempts to counteract the episodes of overeating. These periods are contrasted with periods in which Patient A would eat very little and experience improved mood and increased energy (Stunkard, 1990). Stunkard agreed with Habermas's (1989) conclusion that these cases represent early instances of "bulimia," but his use of the term *bulimia* rather than *bulimia nervosa* appears to be a deliberate distinction between the syndrome defined in the *DSM–III* versus that defined in the *DSM–III–R*. Given that the *DSM–III* category encompassed what are currently recognized as BN and binge-eating disorder, it is unclear whether Wulff's cases better represent binge-eating disorder. Echoing this diagnostic uncertainty, Habermas (1992a) commented, "Stunkard's (1990) hypothesis of a historical continuity of bulimia probably is not true if it is restricted to bulimia nervosa" (p. 357). Only Patient D was characterized by a pattern in which she experienced a binge–purge cycle within one period; these periods alternated with short periods of abstinence and fasting (Wulff, 1932, as translated by Stunkard, 1990).

Pope et al. (1985) reviewed four individuals presented by Pierre Janet in 1903 who appeared to satisfy *DSM–III* criteria for bulimia on the basis of recurrent binge-eating episodes. However, of these four cases, the combination of binge eating with purging is presented in only one case. This case involved a 17-year-old man, "Ron," who experienced episodes of "voracious appetite," "never felt satiety," (Pope et al., 1985, p. 741) and engaged in self-induced

vomiting after the periods of heavy food consumption. The remaining three cases provide inadequate information to make a differential diagnosis of present-day BN versus AN—binge–purge subtype or binge-eating disorder. Habermas (1991) presented Ludwig Binswanger's description of Irma, published in 1909, as "the first known report on a case of bulimia nervosa at normal body weight" (p. 361). The 22-year-old patient engaged in recurrent binge eating and fasting and experienced fear of becoming overweight. Thus, according to Habermas's (1989, 1991, 1992a) reviews, cases of BN existed during the first half of the 20th century.

Following their review of Janet's cases, Pope et al. (1985) concluded that the major psychiatric texts of the nineteenth century, including those of Esquirol (1838), Briquet (1859), and Laségue (1871; all as cited in Pope et al., 1985), included no apparent cases of *DSM–III* bulimia. Conversely, Habermas's (1989) review of Briquet revealed a case of apparent BN in a woman who "ate well" but vomited everything she had eaten and maintained a normal weight (Habermas, 1989, p. 267). Van Deth and Vandereycken (1995) reviewed cases of hysterical vomiting and noted that some cases occurred in individuals of normal weight who also engaged in binge eating and fasting behaviors. Notably, most of these cases occurred in female adolescents. However, the authors equated these cases more with a modern-day conversion disorder, psychogenic vomiting, or AN rather than BN (Van Deth & Vandereycken, 1995). Rosenvinge and Vandereycken (1994) reviewed a case of "hysteria" described by Selmer in 1892 in which a 12-year-old girl refused to eat but maintained normal weight. This apparent contradiction was explained one night when the girl's mother observed her "eating butter, herrings, potatoes and all the food she was able to find in the house" (Selmer as cited in Rosenvinge & Vandereycken, 1994, p. 280). Thus, this girl appeared to fast during the day and binge eat at night (Rosenvinge & Vandereycken, 1994), although it remains unclear how the parents failed to notice the disappearance of so much food earlier in the course of their daughter's illness. One 1870 report described a 14-year-old girl who would fast for 18 days and then enter a period during which she ate voraciously (B. Parry-Jones & Parry-Jones, 1991). This case resembles those presented by Wulff (Stunkard, 1990) in that extended periods of binge eating alternate with extended periods of fasting rather than there being a cycle of binge eating coupled with inappropriate compensatory behavior. Remaining cases of purported bulimia in the nineteenth century include one 30-year-old man in 1897 who consumed large quantities of food day and night and 2 men whose voracious consumption included living animals and human flesh, but none engaged in purging or other forms of inappropriate compensatory behavior (B. Parry-Jones & Parry-Jones, 1991).

For the eighteenth century, B. Parry-Jones (1992) detailed the case of Samuel Johnson from 1784 as meeting *DSM–III–R* criteria for BN. Johnson engaged in binge-eating episodes as they are defined in the *DSM–IV*, and these caused him to be significantly overweight. To control his weight, he engaged in fasting and used senna as a purging agent. As in cases reported by Wulff (1932, as cited in Stankard, 1990), Johnson's use of fasting and purging seemed quite time limited compared with his long-standing pattern of compulsive overeating. In 1764, a 16-year-old boy developed a voracious appetite during the course of illness with typhus fever; however, no inappropriate compensatory behavior was noted (B. Parry-Jones & Parry-Jones, 1993; Silverman, 1987).

For the seventeenth century, a 50-year-old man (1678) experienced uncontrollable eating followed by vomiting 20 days each year, and, following the 20-day binge–purge cycle, the man fasted for 20 days and then resumed normal eating throughout the remainder of the year (B. Parry-Jones & Parry-Jones, 1991; Ziolko, 1996). Robert Whytt (1714–1766) provided a description of *fames canina* originally observed by Richard Lower (1631–1691) in the seventeenth century (Silverman, 1987). According to Whytt (1764), Lower observed "an uncommon hunger" among patients with hypochondria and hysteria that produced "a great craving for food" (as cited in Silverman, 1987, p. 145). "In other cases, however, the morbid matter affecting the nerves of the stomach in hypochondriac and hysteric patients, sometimes occasions a want of appetite and a *nausea*" (Silverman, 1987, p. 145). Although fames canina is supposed to be a disorder characterized by large food intake followed by vomiting (Stein & Laakso, 1988), Whytt's review of Lower's observations does not make a clear connection between the presence of "uncommon hunger" and "want of appetite and a nausea" in the same patients (Silverman, 1987). B. Parry-Jones and Parry-Jones (1991) reviewed 12 potential cases of BN from the seventeenth to nineteenth centuries. Five of these cases were described above. Among the remaining 7 cases, none were associated with inappropriate compensatory behavior, and parasitic worms were found in 4 cases. As cited in Ziolko (1996), Forestus (1602) described a nun afflicted with canine appetite (fames canina or kynorexia) who was miraculously cured after several unsuccessful medicinal treatments by physicians. Like fames canina, kynorexia was defined by insatiable appetite, eating that is out of control, and then compulsive vomiting as a result of excessive food intake (Stein & Laakso, 1988; Ziolko, 1996).

From the twelfth to the seventeenth centuries, many of the fasting saints were reported to engage in binge eating (e.g., St. Veronica) and self-induced vomiting (e.g., St. Catherine; Bell, 1985; Rampling, 1985). However, these cases appear to fall within the diagnosis of AN—binge–purge subtype (*DSM–IV–TR*). It is unclear whether there were cases of binge eating and purging among normal-weight women seeking spiritual purification through fasting. If these cases were common in convents, there is little reason to think that they would go unnoticed given the religious significance of women's eating during medieval times (Bynum, 1987).

In the eighth century A.D., Avicenna prescribed self-induced vomiting to undo the ill effects of overeating; however, he warned,

> To procure vomiting to an undue degree is injurious for the stomach, it is also prejudicial to the thorax and the teeth . . . and may lead to consumption. The custom of some people who eat to excess and then procure vomiting, is one of the things that end in a chronic disorder. (Gruner, 1930, as cited in Nasser, 1993, p. 130)

This suggests awareness of the morbidity associated with chronic self-induced vomiting and that a disorder characterized by overeating and self-induced vomiting was known in Arabic medicine. However, no further information was provided concerning the individuals with this disorder or whether the disorder was maintained at normal weight.

For cases prior to the second century A.D., Crichton (1996) speculated as to whether the Roman emperors Claudius (A.D. 41–54) and Vitellius (circa A.D. 69) suffered from BN. Notably, Ziolko (1996) rejected these cases on the basis that excessive food intake appeared to be based in intentional gluttony with self-

induced vomiting used as a means to allow continued consumption. However, excerpts from Suetonius's description of Vitellius suggest that the emperor's excessive food intake may not have been under his control: "He was a man of not only such extreme and impulsive, but also disgusting, gluttony that he could not even curb it during a sacrifice or on a journey" (translated in Crichton, 1996, p. 204). Crichton noted that vomiting distinguished Claudius and Vitellius from their historical peers; however, Seneca is known to have commented, "vomunt ut edant, edunt ut vomant [they vomit that they may eat, they eat that they may vomit]" (as cited in Crichton, 1996, p. 206). This suggests that binge eating and purging may have represented a common behavioral pattern among the elite in the Roman Empire, and, in the case of Vitellius, a not entirely volitional pattern of eating.

Interpretation of historical cases of bulimia. In our review of historical cases of AN, we found numerous examples of young women engaging in self-starvation. The overt behaviors, consequences, course, and affected population resembled that for modern-day AN. Thus, the debate centered mostly on the motivation behind food refusal. In contrast, evidence of a binge–purge syndrome outside of its present historical context is quite sparse. Historical accounts of bulimia do not seem to preponderate in adolescent or young adult females. In fact, prior to the nineteenth century, cases involved mostly adult men. Further, most historical cases of bulimia represented the syndrome defined in the *DSM–III* that did not require inappropriate compensatory behavior. Thus, in our attempt to find historical accounts of BN, we seemed to find numerous examples of binge-eating disorder.

According to the restraint hypothesis (Polivy & Herman, 1985), one would expect that the fasting evident throughout the twelfth to the nineteenth centuries would result in binge eating for a number of young women. Consistent with this hypothesis, there were isolated cases in which individuals seemed to engage in binge eating after a period of restricted food intake. However, the use of purging (or other forms of inappropriate compensatory behaviors) to counteract or undo such episodes was lacking in most reported cases of binge eating. This difference may be explained by the motivation behind food restriction. In modern times, food restriction often is intended to achieve weight loss. When a binge episode occurs, purging is motivated by the belief that it will prevent weight gain. Conversely, if fasting is interpreted in a religious framework, then purging cannot prevent the sin of gluttony once the binge episode has occurred (Bynum, 1987). Bynum (1987) related the story of Friderade, who suffered from voracious appetite and "grew enormous" (p. 89). After confessing, she was cured and did not eat for the following 3 years (Bynum, 1987). This story provides a template for most of the historical cases of bulimic syndromes—a period of recurrent binge eating is followed by a period of abstinence. However, this behavioral pattern resembles that of a person with binge-eating disorder in which extended periods of binge eating alternate with periods of dieting.

Our review of AN suggested that specific motivations related to weight concerns did not seem to be necessary to produce the syndrome. We raised the question of whether weight concerns truly cause AN or represent an attempt to understand the disorder by both the individual suffering from AN (Wegner, 2002) and her culture. Conversely, our review suggests that a binge–purge syndrome predominantly affecting normal-weight women may not emerge in the absence of weight concerns.

Cross-Cultural Evidence for BN

Characterizing the actual prevalence of BN outside of a Western context has proven challenging. For example, across several reviews of eating disorders cross-culturally (Davis & Yager, 1992; Dolan, 1991; Miller & Pumariega, 2001; Pate, Pumariega, Hester, & Garner, 1992; Tsai, 2000), references to AN far outnumbered references to BN. This difference is also reflected in the range of countries reporting BN among their citizens; in our review, we found reports of AN in five of five non-Western regions of the world (see Table 2), whereas BN was reported in only three of five non-Western regions (see Table 4). Similar to the construction of Table 2, Table 4 indicates the country in which cases were found, diagnostic criteria used, number or prevalence of cases, and degree of Western influence. We eliminated the column concerning presence of weight concerns because our review of cases indicated that BN was accompanied by weight concerns in *all* cases. As in the analysis shown in Table 2, weight concern and Western influence were established by consensus.

We found no studies reporting the presence of BN in an individual with no exposure to Western ideals. All of the BN cases that could be categorized involved individuals who were probably or definitely exposed to Western ideals and values through urbanization, English-medium schools, previous residence in Western nations, and/or higher socioeconomic status or educational attainment. If BN only emerges in non-Western countries as a result of Western influences, then this may explain why there does not appear to be a non-weight-concerned form of BN.

With the exceptions of Japan and a recent study in Iran, prevalence estimates of full BN in non-Western nations were below the range reported for Western nations in the *DSM–IV–TR*. Degree of Western influence may account for a good deal of variance in BN prevalence estimates. For example, following World War II, Japan has emulated some of the ideals of the United States. Indeed, the *Cassell Dictionary of Modern Politics* (East & Joseph, 1994) indicates that Japan is sometimes included in the definition of the West. Thus, it is not surprising that BN prevalence is the most similar between the United States and this non-Western nation. Epidemiological data for BN in non-Western nations suggest that BN has a lower prevalence than AN in these countries, a pattern that is the opposite of that observed in Western nations. The point prevalence of AN has been found to be four to five times greater than full-syndromal BN in Japan (Nakamura et al., 2000), and it has been noted that AN is more prevalent than BN in Hong Kong (Chen et al., 1993; S. Lee, Hsu, & Wing, 1992). Thus, even when BN is found in non-Western nations, it is not found in the absence of Western influence, and it seems to be less common than AN.

Summary of Findings for BN

It appears to be the case that BN is a culture-bound syndrome. Although epidemiological data for BN are limited, they support a large and significant increase in BN incidence during the latter half of the twentieth century. Our attempts to find evidence of BN in earlier historical periods were largely unsuccessful. Most historical cases of recurrent binge eating seem to represent binge-eating disorder (because of a lack of inappropriate compensatory behavior) or AN—binge–purge subtype (because of the presence of low weight). Finally, although BN does exist in non-Western nations, we were unable to find evidence of the disorder arising in the absence of Western influence.

Table 4
Studies of Bulimia Nervosa (BN) in Non-Western Nations

Article	Place	Diagnostic criteria	Number–prevalence	Cases without Western influence?
		Middle Eastern–Arab Cultures		
Ford (1992)	Egypt	*DSM–III–R*	1 case reported; developed BN upon returning to Egypt after living in Canada for 12 years	No
Nasser (1994)	Cairo, Egypt	Russell (1979)	0.9% full BN; 3.4% partial syndrome BN in 351 high school students	Unlikely
Nobakht & Dezhkam (2000)	Tehran, Iran	*DSM–IV*	3.2% of 3,100 female high school students ages 15–18	Unlikely
Choudry & Mumford (1992)	Pakistan	*DSM–III–R*	0.4% full BN in 271 Urdu medium school girls ages 12–16	Unlikely
Mumford et al. (1992)	Pakistan	*DSM–III–R*	0.002% full BN, and 0.01% subthreshold BN in 369 English-medium school girls ages 14–16	Unlikely
		Southeast Asia		
Ong et al. (1982)	Malaysia	Not given	1 case of nonpurging BN reported in a woman during recovery from AN	Unlikely
Schmidt (1993)	Malaysia	*DSM–III–R*	1 case in a male; vomiting–weight concerns began in Malaysia, binge eating in England	Unlikely
Goh et al. (1993)	West Malaysia	Study specific	1 case reported	Indeterminable
Ung et al. (1997)	Malaysia	Not given	16 cases of "bingeing and/or purging behavior in subjects of normal or above normal weight" (p. 332)	Indeterminable
		East Asia		
Nogami et al. (1984)	Japan	Unknown	17 individuals with both binge eating and purging of 846 university students ages 18–30	Unlikely
Kiriike et al. (1988)	Japan	*DSM–III* and Russell (1979)	2.9% of 456 college women report bingeing and purging at least twice per week	Unlikely
Takeda et al. (1993)[a]	Japan	*DSM–III–R*	1.9% high school girls	Unlikely
Kuboki et al. (1996)	Japan	Not given	1.3–2.5 per 100,000 general population in 1992	Indeterminable
Nadaoka et al. (1996)	Japan	*DSM–III–R*	38 cases treated at Yamagata University Hospital between 1978 and 1992	Unlikely
Nakamura et al. (2000)	Japan	*DSM–IV*	1.02 per 100,000 females	Indeterminable
Takei et al. (2000)[b]	Japan	*DSM–IV*	321 students with BN from 335 college health administration centers across Japan	Unknown
Chun et al. (1992)	China	*DSM–III–R*	1.1% of 509 freshman medical college students (401 female)	Unlikely
S. Lee, Hsu, & Wing (1992)	Hong Kong	*DSM–III–R*	4 cases treated at Department of Psychiatry, University of Hong Kong, between 1980 and 1988	Unlikely
S. Lee (1993)	Hong Kong	*DSM–III–R*	0.3% partial BN syndromes in 1,020 bilingual university students	Unlikely
S. Lee, Lee, & Leung (1998)	Hong Kong	*DSM–III–R*	17 consecutive series of patients treated from 1990–1996	Unlikely
Lam & Lee (2000)	Hong Kong	*DSM–IV*	Report on 30 cases of BN between 1984 and 1998; provided detailed case histories of 3 patients among whom only 1 seemed to have *DSM–IV* BN	Indeterminable; unlikely for 3 detailed cases
Tseng et al (1989)[b]	Taiwan	*DSM–III*	5 patients reported	Unknown
Hung & Cheng (1992)[a]	Taipei, Taiwan	*DSM–III–R*	5.6% in 12,435 females ages 12–25	Indeterminable

Note. Not given = authors did not provide the information in the article; Unknown = authors may have provided indicated information in the non-English publication, but the information was not provided in the English abstract or the English review article summarizing it; Study specific = diagnostic criteria were specific to the study indicated; Indeterminable = exposure to Western influence could not be determined from article; *DSM–III–R = Diagnostic and Statistical Manual of Mental Disorders* (3rd ed., rev.); *DSM–IV = Diagnostic and Statistical Manual of Mental Disorders* (4th ed.); *DSM–III = Diagnostic and Statistical Manual of Mental Disorders* (3rd ed.).
[a] As reported in Tsai (2000). [b] Data were taken from an English abstract of a non-English publication.

Discussion

Divergent patterns emerged in our examination of epidemiological, cross-historical, and cross-cultural evidence of AN and BN as culture-bound syndromes. Unlike previous conclusions that eating disorders either are culture bound or are not culture bound (Habermas, 1989; S. Lee et al., 1993), our findings suggest that BN is culture bound and that AN is not.

Reasons for the differences in findings between AN and BN could be attributed to our definition of the phenotypes. We sought evidence of AN and BN according to their conceptualization within the *DSM–IV*. However, the *DSM–IV* does not represent "nature carved at its joints." If we had allowed a more generous definition of BN to include all syndromes characterized by recurrent binge eating, the conclusions of our review would be significantly altered as the symptom of binge eating appeared in more historical and culture contexts than the syndrome of BN. We chose to follow the *DSM–IV* conceptualization of these syndromes because it has the greatest empirical support. Of interest, the results of our review seem to add support for distinguishing between the

two syndromes. Further support comes from a recent study of the influence of Western media exposure on rates of disordered eating in Fiji (Becker, Burwell, Gilman, Herzog, & Hamburg, 2002). In 1995 (within a month of the introduction of television), 7.9% of Fijian girls reported binge eating. In 1998 (3 years after the introduction of television), the percentage had decreased nonsignificantly to 4.6%. Conversely, the percentage of girls who reported self-induced vomiting increased significantly from 0% to 11.3% over the same period (Becker et al., 2002). Prior to study initiation, one case of AN had been reported in Fiji (Becker et al., 2002). These data suggest that AN and binge eating in the absence of inappropriate compensatory behavior may not be particularly dependent on exposure to Western ideals. However, a syndrome characterized by the combination of binge eating and purging at normal weight may be more culture bound.

Although the evidence leads us to a strong conclusion that AN has existed outside of its current sociohistorical context, the conclusion for BN is necessarily weaker as there were fewer articles concerning BN than AN. This may represent the relatively longer recognition of AN as a syndrome (and thus greater time to accumulate publications on AN); however, the majority of the articles reviewed have been published since the introduction of BN to the psychiatric nomenclature. Further, this publication pattern is the inverse of that observed for the treatment literature in AN versus BN (Peterson & Mitchell, 1999). Thus, it remains unclear whether the recent recognition of BN is the cause or result of limited data concerning its existence. The limited data for BN may not reflect the absence of the disorder because BN may have existed but eluded detection. This seems plausible because there is no overt sign of BN such as the emaciation that characterizes AN. Indeed, Whitehouse et al. (1992) found that 50% of cases of BN determined in the community were unknown to their general practitioners, despite referrals made for complications of bulimic pathology in half of these "hidden" cases. No such hidden cases of AN were found, suggesting that the low weight of AN likely makes AN easier to detect outside of the confines of a recognized syndrome. However, there are reasons why concerns about detection of BN may be overstated. Further, these reasons contribute to the understanding of why BN may represent a more culturally bound syndrome than AN.

Although self-starvation can occur in any context, binge eating requires large stores of readily edible food. Thus, cases may be validly limited to places and periods with abundant food, such as the palaces of the Roman Empire, places of communal living with shared kitchens, or affluent households in Victorian England. In earlier historical contexts, food consumed a greater proportion of household budgets, and distribution was closely monitored with greater portions going to men who were engaging in manual labor (Bailey & Earle, 1999). Indeed, gender differences in access to food may explain why cases of binge eating prior to the nineteenth century more commonly involved men (B. Parry-Jones & Parry-Jones, 1991). Thus, whereas individuals can deliberately starve themselves regardless of access to food—in some cases refusing even the meager portions allotted to them—individuals cannot binge eat without free access to large quantities of food. If large amounts of food were obtainable, it is difficult to believe that recurrent binge episodes would go unnoticed because food was not obtained in anonymity as it is today. Certainly, the food intake of Samuel Johnson was not only noticed but recorded by several individuals during and following his lifetime (B. Parry-Jones, 1992) as were the binge-eating episodes of St. Veronica (Bell, 1985). Indeed, the presence of binge eating as a symptom has been recorded both cross-historically and cross-culturally with the primary constraint being one of access to food. Similar to difficulties in attempting to binge eat without detection, purging would be a difficult behavior to hide prior to the wide availability of modern plumbing. The ability to flush away evidence of self-induced vomiting, or laxative or diuretic abuse, from the privacy of one's indoor bathroom would greatly facilitate secretive purging. Chamber pots and outhouses would not. Further, accounts of fasting and excessive exercise are plentiful among historical cases of fasting girls, even those of normal weight, suggesting that nonpurging forms of inappropriate compensatory behavior would not remain hidden for long. Although no obvious sign characterizes BN, it seems unlikely that BN would go unnoticed in earlier historical periods. Thus, the relative dearth of evidence of BN outside of its sociohistorical cultural context may reflect its lack of existence. Of interest, an ability to obtain large amounts of food inconspicuously may explain why BN, but not AN, is more common in urban than rural areas (Hoek et al., 1995; Robins et al., 1984).

Binge eating may be constrained by access to large quantities of food, and purging may be relatively limited to a context in which prevention of weight gain is culturally meaningful. Thus, unlike AN, which can occur in numerous contexts, the specific combination of binge eating and purging may be limited by two factors specific to modern Western cultures—access to food and use of purging to prevent weight gain. This would suggest greater cross-historical and cross-cultural representation of the restricting subtype of AN compared with the binge–purge subtype. Of interest, a clinical difference between S. Lee et al.'s (1993) patients with fat phobic and non-fat phobic AN was the increased presence of binge–purge symptoms in the patients with fat phobia.

Conceptualizing the Genetic Bases of Eating Disorders

Sociocultural and cognitive–behavioral models of eating disorders emphasize the role of weight concerns in their etiology, whereas behavioral genetic research emphasizes the role of genes. As we stated in the introduction, the purpose of our research was to understand the extent to which eating disorders represent culture-bound syndromes and the implications of our findings for conceptualizing their genetic bases. Our review suggests that BN may be a culture-bound syndrome, influenced by weight concerns, anonymous access to large quantities of food, and a motivation to prevent the effects of binge eating on weight through the use of inappropriate compensatory behavior. Conversely, our review suggests that weight concerns can influence the incidence of AN but that whatever cultural influences contribute to the etiology of AN, they are not particularly limited in their distribution across history or cultures.

Although recent behavioral genetic research has provided substantial evidence of genetic influences on the development of both AN (Klump et al., 2001; Kortegaard et al., 2001; Wade et al., 2000) and BN (Bulik et al., 1998, 2000; Kendler et al., 1991, 1995), heritability estimates are constrained to the cultures from which study samples are drawn. In cultures in which the thin ideal is ubiquitous, these kinds of environmental factors are held relatively constant across individuals and thus cannot account for individual differences in eating disorder development. In cultures in which relevant environmental factors vary across individuals,

cultural pressures to be thin have the potential to play a much larger role in individual differences in the development of eating pathology. As such, heritability estimates of BN are more likely to vary across cultures. Within a culture in which there is large variability in exposure to the thin ideal and access to food, genetic estimates may be quite low for BN. Conversely, heritability estimates of AN are likely to be more stable. Current data do not allow us to examine this hypothesis directly because twin studies of AN and BN have been conducted within industrialized Western nations. Future research should seek to examine this question more closely using, whenever possible, twins from non-Western countries or combining samples from Western and non-Western countries.

Because heritability estimates are statistical entities that gauge the extent to which individual differences within a population can be explained by genetic influences, they do not directly index the action of genes. Although these statistical estimates can vary depending on environmental contexts (with the potential for BN to have low heritability in some contexts), this variation does not mean that genes do not influence BN or that they influence BN less than AN. The genetic diathesis for BN may exhibit more pathoplasticity cross-culturally than the genetic diathesis for AN. When the diathesis for a disorder is general or has high pathoplasticity, the disorder may not be evident cross-culturally or cross-historically. That is, although the genetic diathesis may exist throughout history and cross-culturally, when tapped, it presents as a different form of pathology conforming to its current sociohistorical context. In contrast, when the genetic diathesis for a disorder is specific or has low pathoplasticity, evidence of the disorder should be available cross-culturally and cross-historically. The disorder may be less common in different sociohistorical periods, but whenever the diathesis is tapped, it should produce the same narrow syndrome.

In past historical periods, the genetic predisposition for what is now BN may have produced different forms of psychopathology, such as hysteria in the Victorian era. The demographic features of those afflicted with these two illnesses are quite similar, and the abrupt emergence of these syndromes is also quite similar. Pope and Hudson (1988) have long viewed eating disorders as lying along an etiologic spectrum with numerous other syndromes. The extent to which BN represents a culturally shaped manifestation of a general genetic diathesis may explain why this disorder, but not AN, responds to the same medications and psychosocial treatments that are used to treat major depressive disorder (MDD) and obsessive–compulsive disorder (OCD; Peterson & Mitchell, 1999). Specifically, both antidepressant medications and cognitive–behavioral therapy have demonstrated efficacy in treating BN but not AN (Peterson & Mitchell, 1999). Possibly the genetic diathesis to BN can be expressed as hysteria, BN, MDD, OCD, or any combination of these depending on a variety of environmental factors. Twin studies have the ability to examine this question indirectly by investigating whether disorders share genetic transmission. If there is a high degree of shared transmission among disorders, then the disorders may represent different expressions of a genetic diathesis with high pathoplasticity. An early twin investigation of BN found shared genetic transmission between this disorder and major depression (Walters et al., 1992). However, when examined with a larger, more powerful multivariate model including six psychiatric disorders (BN, phobias, generalized anxiety disorder, panic disorder, major depression, and alcoholism), BN loaded on a single genetic factor with phobias and panic disorder (Kendler et al., 1995). Findings from this study suggest that BN may indeed share genetic transmission with other forms of "neuroses" and that this shared transmission may be indicative of greater pathoplasticity of the disorder. To date, no study has examined AN using multivariate, genetic analyses. Although one study found some common genetic risk factors between AN and major depression (Wade et al., 2000), findings were limited by the inclusion of only one comorbid disorder. Thus, future twin research can evaluate the pathoplasticity associated with the genetic diatheses for AN and BN.

Conclusion

Previous work has posed modern Western idealization of thinness as a common etiologic factor for AN and BN. However, our review reveals that the epidemiology, history, and cultural distribution of these disorders are distinct. Researchers interested in revealing the pathophysiology of these disorders would do well to attend to these patterns, as they suggest distinct hypotheses concerning variability of heritability estimates and pathoplasticity. If our hypotheses are supported in future behavior genetic research, then two lines of evidence would suggest that some or all of the genes contributing to the development of BN differ from those contributing to the development of AN. This would provide the greatest evidence, thus far, for etiologic validity in distinguishing between these two syndromes. The extent to which AN and BN are linked in the medical, academic, and popular press may represent a historical coincidence more than a true relationship between these disorders.

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New Editor Appointed for *Contemporary Psychology: APA Review of Books,* 2005–2010

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