

Population-Based Prevention of Eating Disorders: An Application of the Rose Prevention Model¹

S. Bryn Austin, Sc.D.²

Division of Adolescent Medicine, Children's Hospital, Harvard School of Public Health

Published online January 26, 2001

Background. Several decades of concerted research on eating disorders have generated a broad range of proposed causal influences, but much of this etiologic research does not elucidate practical avenues for preventive interventions. Translating etiologic theory into community health interventions depends on the identification of key leverage points, factors that are amenable to public health intervention and provide an opportunity to maximize impact on the outcome of interest. Population-based preventive strategies, elaborated by epidemiologist Geoffrey Rose, can maximize the impact of public health interventions. In the case of eating disorders, Rose's model is instructive: Dieting stands out as risk behavior that may both fit Rose's model well and be a key leverage point for preventive intervention.

Methods. Grounded in Rose's work, this article lodges a theoretical argument for the population-based prevention of eating disorders. In the introductory section, existing research on the epidemiology of dieting is reviewed, showing that it is extremely common among adolescent girls and women and that the behavior has been implicated as a causal factor for disordered eating. Next, new evidence is offered to build a case for how a population-wide reduction in dieting may be an effective strategy for prevention of eating pathology. Finally Rose's prevention framework is used to introduce a unique and provocative perspective on the prevention of eating disorders.

Results. Dieting is a normative behavior in our culture with psychological and physiological effects in the causal chain leading to eating pathology. This behavior may represent an ideal target for population-based prevention.

Conclusions. Theoretical and empirical evidence suggests that a population-wide reduction in dieting may be a justifiable and effective strategy for prevention of eating pathology. © 2001 American Health Foundation and Academic Press

Key Words: anorexia nervosa; bulimia nervosa; dieting; eating disorders; eating pathology; prevention.

INTRODUCTION

Numerous etiologic models have been proposed for eating disorders, representing a myriad of research orientations, from psychoanalytic to social constructionist to biomedical [1–3]. The proposed causes generally fall in four categories: physiological, psychological, familial, and sociocultural. There is consensus in the field that eating disorders have multicausal origins [4–7]. Biological research suggests that the neurotransmitters serotonin, norepinephrine, and dopamine may be implicated in the etiology and maintenance of eating disorders [8,9]. Personality factors such as low self-esteem, poor impulse control, poor interoceptive awareness, and deficits in interpersonal relationships, along with dysfunctional familial patterns of abuse, parental rigidity, and incompetence in conflict resolution have also been posited as contributory.

Thinness as a cultural standard of female beauty and value and the routinization of dieting as a female rite of passage have been cited as sociocultural determinants of disordered eating [10–13]. The media, especially those targeting women and girls, and their role in engendering and exacerbating body dissatisfaction by promoting unrealistic standards of beauty have been widely critiqued in the eating disorders and feminist

¹ The author is supported by the Leadership Education in Adolescent Health Project Grant 5-T71-MC-00009-09 from the Maternal and Child Health Bureau (Title IV, Social Security Act), Health Resources and Services Administration, Department of Health and Human Services. The Planet Health Study was supported by Grant HD-30780 from the National Institute of Child Health and Development.

² To whom reprint requests should be addressed at Division of Adolescent Medicine, Children's Hospital, 300 Longwood Ave., JB-335, Boston, MA 02115. Fax: (617)232-1851. E-mail: bryn.austin@tch.harvard.edu.



literatures [6,12,13,14–18]. Experiences of weight-related teasing and harassment have also been found to be associated with poor body image, unhealthy dieting practices, and disordered eating [1,5,12,19–21].

Several decades of concerted research on eating disorders have generated a broad range of proposed causal influences, and this breadth of inquiry is indispensable for ultimately understanding eating pathology. Much of this etiologic research, unfortunately, does not elucidate practical avenues for preventive interventions. How to translate etiologic theory into community health interventions is often not transparent and is dependent on the identification of key leverage points [22]. Leverage points, as defined by Stokols [22], are factors that are amenable to and feasible for public health intervention. In addition, they must provide an opportunity to maximize impact on the outcome of interest [22,23]. This compelling public health interest in identifying ways to maximize the effects of interventions is the linchpin of population-based prevention [24,25].

Population-Based Prevention

In *The Strategy of Preventive Medicine* [24], British epidemiologist Geoffrey Rose defines two approaches to prevention. One, the high-risk strategy, aims to identify individuals most at risk for a disease or outcome—usually meaning they exhibit signs of premorbid disease or a high level of a known risk factor—and then target preventive efforts to that subgroup. The other he terms the population-based strategy, which is designed to target the community as a whole, not a subset of high-risk individuals. While similar concepts of primary, secondary, and tertiary prevention predate Rose's work and have been addressed widely in the public health literature [26], Rose's discussion is unique in that he presents a statistically grounded perspective on maximizing potential impact for prevention.

Rose has extended his thinking on population-based prevention from cardiovascular disease (CVD) to mental illness, using examples of suicide and alcohol abuse to argue that prevention efforts in this domain have largely been dominated by a clinical perspective, one that is chiefly interested in case definition and intervention for those already ill or most at risk [27]. Rose's alternative prevention framework does not necessarily apply equally well to all types of mental illness—or somatic illness, for that matter—because it was originally designed to be used in the context of a clearly defined exposure, with both a unimodal population distribution and a graduated level of risk. But in the case of eating disorders, Rose's model can be instructive. Dieting stands out as a risk behavior that may both fit Rose's model well and be a key leverage point for preventive intervention.

As will be argued in the coming pages, dieting is a

normative behavior in our culture with psychological and physiological effects in the causal chain leading to eating pathology and may represent an ideal target for population-based prevention. The following sections first present prevalence data on dieting, showing that it is extremely common among adolescent girls and women in the United States, then review the existing research implicating dieting as a causal factor for disordered eating. Next, new evidence is offered to build a case for why a population-wide reduction in dieting is likely to be an effective strategy for prevention of eating pathology. Finally the central thesis of this article is presented, using Rose's model to introduce a unique—and hopefully compelling—perspective on the prevention of eating disorders.

Epidemiology and Sequelae of Eating Pathology

Among female adolescents and young women in the United States, the prevalence of anorexia nervosa has been estimated at about 0.5%, and the prevalence of bulimia nervosa at about 2% [28]. Eating pathology not necessarily meeting psychiatric criteria for an eating disorder [29] has been found in much higher prevalence in school and community samples [28, 30–38]. The most recent data from the Youth Risk Behavioral Surveillance System (YRBSS), a national survey of over 15,000 high school students conducted by the Centers for Disease Control in 1999, found 19% of girls and 6% of boys reported fasting for periods of 24 h or more in the past month in order to lose or maintain weight [30]. Eleven percent of girls and 4% of boys had taken diet pills, powders, or liquids without a doctor's recommendation in the past month in order to lose or maintain weight. Close to 8% of girls and 2% of boys reported vomiting or taking laxatives in the past month to lose or maintain weight. Prevalence estimates based on self-report measures of self-induced vomiting and other fairly unambiguous behaviors have been found to be similar to those based on structured interviews, although more ambiguous behaviors such as binge eating may be overestimated with self-report measures [39]. Most studies have found adolescent girls to be more likely than boys to induce vomiting or abuse laxatives, diuretics, or diet pills [40].

The medical and psychiatric conditions associated with eating pathology, especially anorexia nervosa, can be severe, including amenorrhea, anemia, osteoporosis, and arrhythmia. In a prospective study of 84 female anorectic patients in Germany, Herzog *et al.* [41] found that 67% of the subjects suffered serious medical comorbid conditions after 12 years of follow-up, including kidney disease and osteoporosis. Eight of the 84 patients had died, with the mean age of death of 29. In a metaanalysis of 13 outcomes studies from seven nations, Harris and Barraclough [42] report that the age-

and sex-adjusted risk of suicide for anorectic patients is 23 times the rate expected in a comparable nonclinical population. Based on a review of the literature, Nielsen *et al.* [43] estimate that the death rate for female anorectics is 6% per decade.

Even eating pathology not meeting clinical criteria for an eating disorder is associated with negative health outcomes. Chronic induced vomiting can lead to erosion of tooth enamel and esophageal damage. Laxative abuse can result in impairment of digestive functioning [29,44,45]. Binge-purge cycling has been found to be associated with secondary amenorrhea, which is itself associated with additional medical problems [46]. Abuse of ipecac syrup, used to cause vomiting, is associated with cardiac toxicity [47].

In sum, the proportion of adolescents and young adults engaging in fasting, induced vomiting, and abuse of laxatives, diuretics, ipecac, and diet pills may be 5 to 10 times the proportion suffering from eating disorders that meet clinical criteria. Given the evidence of serious morbidity associated with these behaviors, identifying ways to prevent young people from adopting them is a significant public health issue.

Epidemiology of Dieting

During the second half of this century, there has been a temporal trend toward increasing prevalence of dieting and weight loss efforts in the United States. In the 1950s and 1960s, national surveys found 7% of men and 14% of women reported trying to lose weight. In the mid 1970s, 16% of adults in the United States reported trying to lose weight. Since the mid 1980s, the estimates of the percentage of adults dieting at any one time have been about 25% of men and 45% women [40,48-50].

Surveys of adolescents similarly show dieting to be very common, especially among girls. The 1999 national YRBSS found that nearly 60% of female and 26% of male high school students were trying to lose weight in the month before the survey [30]. In a large cross-sectional study of Minnesota adolescents in grades 7 through 12, French *et al.* [51] found 62% of girls and 21% of boys had dieted at least once in the previous year; 21% of girls and 4% of boys reported dieting five or more times during the year. The National Adolescent Health Survey, which collected data from a large sample of 8th- and 10th-grade students, found that 61% of girls and 28% of boys had dieted in the past year; 20% of girls and 5% of boys reported dieting four or more times during the year [52].

Overweight is positively associated with self-reported efforts to lose weight [40, 53-58], and recent increases in the prevalence of obesity may partly explain why the prevalence of dieting has risen so enormously since the 1950s. Importantly, dieting and dissatisfaction with weight and shape extend far beyond those who are actually overweight or obese. In a Massachusetts survey of

5th- through 12th-grade girls, 34% of normal-weight girls said that they were overweight, with 37% of those girls reporting that their ideal was less than 90% of their present weight [53]. Among underweight girls in this study, half preferred to weigh less than their current weight [53]. Almost a third (30%) of the girls responding to the Massachusetts Youth Risk Behavioral Survey in 1997 said they did not perceive themselves to be overweight yet were currently trying to lose weight anyway [59]. In another survey of adolescents, Moses *et al.* [58] found 20% of underweight girls reported dieting.

Field and colleagues [53] found weight and shape concerns to be correlated with frequency of dieting ($r = 0.54$). Greater than 11% of the girls were rated as extremely concerned with their weight and shape on a weight and eating concern scale. Among this subset, 30% reported that they were constantly dieting, whereas less than 3% of those rated as not concerned with weight were doing so [53]. Even among preadolescents, weight concern is widespread. In a survey of children in two Midwestern elementary schools, Maloney *et al.* [36] found 79% of girls and 41% of boys in the sixth grade reported wanting to be thinner, while 60% of the girls and 31% of the boys in that grade said they had tried to lose weight. Even the third-graders showed significant weight preoccupation: 40% of the girls and 31% of the boys wanted to be thinner, while 28% of girls and 36% of boys said they had tried to lose weight already.

DIETING AND THE RISK OF EATING PATHOLOGY

Explanatory Models

Based on experimental research with dieters, Polivy and Herman posited one of the first models to explain how dieting could lead to eating dysregulation and bingeing. They proposed that because dieting requires suppression of physiological cues regarding hunger and satiety through cognitive control, people become vulnerable to cognitive disinhibitors that can lead to binge eating [60]. Heatherton and Polivy [61] expanded on this theoretical work, describing a spiral model in which successive failures at dieting can lead people into a self-defeating pattern of attributing unsuccessful weight-loss attempts to personal weaknesses. This pattern, they posit, can lead to increasing psychological distress and body dissatisfaction and render the dieter vulnerable to initiating more extreme weight loss methods, such as induced vomiting and abuse of laxatives. Stice and colleagues [62] similarly propose a model that includes body dissatisfaction and negative affect as important ancillary factors in the causal pathway. Even in this more complicated model, dietary restraint figures as a primary contributor to eating pathology.

Experimental and Epidemiological Evidence

Experimental and epidemiological research supports the hypothesis that dieting has both physiological and psychological effects that can lead to dysregulation of eating, binge/purge cycling, and other symptoms of disordered eating. A food deprivation study by Keys and colleagues in the 1940s offered some of the early evidence that extended calorie restriction can lead to bingeing [63].³ Normal-weight men who were conscientious objectors during World War II were given the option of participating in the Keys study in lieu of other required nonmilitary civil service. A group of volunteers without a history of eating pathology were enrolled and instructed to maintain a low-calorie diet until they reduced their weight by 25%. Once the men were allowed to eat freely again near the end of the study, researchers found the men often binged. But even after they had returned to their normal weights, they continued to show similar patterns of excessive overeating [64]. The Keys subjects were unlike contemporary dieters in that they maintained a reduced-calorie diet for altruistic and other reasons unrelated to feeling overweight. Importantly, though, the men were free-living during the course of the study, and they had easy access to additional food if they chose to disobey instructions from the researchers. Like dieters, they had to exert cognitive control to suppress physiological cues of hunger in order to maintain the strict dietary regimen.

Subsequent experimental studies with dieters have compared their eating to that of nondieters under different conditions. A number of studies, reviewed by Polivy and Herman [60] and Heatherton and Polivy [61], have found that when given the opportunity to eat without any prior experimental manipulation, dieters maintain restraint and eat less than nondieters. When subjects are instructed to eat a high-calorie preload—in other words, induced to break their diet—dieters become unrestrained and consume more at the follow-up meal while nondieters eat less. Under other experimental conditions designed to cause stress, nondieting subjects eat less than under nonstressful conditions. In contrast, dieters overeat under stressful experimental conditions. A study by Telch and Agras [65] found somewhat different results with eating disordered subjects rather than dieters. In an experimental study with subjects who had bulimia nervosa or binge eating disorder or who were overweight with no eating disorder, the researchers randomized subjects to a 1-h or 6-h food deprivation condition. While subjects in the 6-h deprivation ate more than the other group when allowed to eat at a buffet, the total kilocalories consumed by the end

of the day was not significantly different between the two groups.

French and Jeffery [40] offer a thorough review of research documenting negative psychological effects of dieting on obese and nonobese populations. Among the obese in clinical samples, low-calorie diets have been associated with irritability, nervousness, depression, and anxiety [66–69]. In community and college samples, dieters have been found to be more depressed and anxious and to have lower self-esteem than nondieters [70–72]. Dieting may also exacerbate preexisting mood problems [40].

Lending additional credence to the hypothesis that dieting plays a role in the development of eating pathology, in numerous studies of clinical samples the majority of patients report that they began dieting before initiating bingeing or purging [73–77], although some have suggested there may be a subset of people with binge eating disorder who begin dieting following the onset of bingeing [78].

Better evidence can be found in several longitudinal studies with community samples [28,40,79]. In a prospective study with adolescent girls, Patton and colleagues [80] found that 21% of current dieters at baseline developed serious eating disorder symptoms by 1-year follow-up. Accounting for the sampling scheme used by Patton *et al.*, 73% of the incident cases at follow-up had been dieters at baseline, whereas dieters made up 32% of the noncase baseline sample. The increased odds for development of an eating disturbance associated with dieting among girls based on the data published by Patton *et al.* is estimated to be approximately sevenfold (6.96; 95% confidence interval (CI): 2.1, 23.4). Accounting for the sampling scheme, the estimated population attributable risk based on this data is approximately 66%. Population attributable risk percentage represents the proportion of disease in a population that is attributable to an exposure; it is an estimate of the proportion of disease that could be eliminated if the exposure were removed [81].

A second prospective study, this one with college women, similarly found dieting at baseline to be a predictor of later initiation of bingeing, induced vomiting, and abuse of laxatives. Drewnowski and colleagues [82] collected survey data on dieting practices from women in the fall of their first year in college and in the spring semester, 6 months later. Among the women who reported at baseline that they dieted regularly, 13% initiated purging (defined as induced vomiting or abuse of laxatives or diuretics) or fasting within 6 months, while just 4% of nondieters did so. Compared to women who did not report dieting at baseline, the regular dieters had approximately 3.3 (95% CI: 1, 10.7) times the odds of developing eating pathology by follow-up. Based on combined risk information for both regular dieters and

³ The Keys study was designed in part to identify safe and effective ways to refeed starving refugees and prison camp detainees at the end of World War II [63].

casual dieters in the sample, an estimated 52% of incident cases of disordered eating in this population of college women was attributable to dieting.

A third prospective analysis based on data from early adolescents participating in the Planet Health intervention study [83] assessed baseline factors associated with initiation of purging (defined as induced vomiting and abuse of laxatives) and use of diet pills to lose or maintain weight after approximately 2 years of follow-up [84].⁴ Among girls who reported dieting in the past month at baseline, over 8% initiated purging or diet pill use at follow-up; among girls who did not report dieting in the past month at baseline, less than 3% initiated these behaviors. Adjusting for intervention status, baseline dieters experienced 3.0 (95% CI: 0.8, 10.8) times the odds of initiating induced vomiting or abuse of laxatives or diet pills at follow-up compared to nondieters, and 36% of the incident cases at follow-up were attributable to dieting at baseline. Taken together, the results of the three prospective studies described above add to the evidence that dieting is an important behavioral risk factor for disordered eating.

Measurement of Dieting

In their review of the dieting literature, French and Jeffery [40] make several observations about the limitations of epidemiological research on dieting. One important point they make is that comparisons across studies are made difficult by differences in the time period on which subjects are asked to report. Some ask respondents to report current dieting behavior, others to report on behavior over the previous month, others over the past year, and still others do not specify the period, asking subjects simply if they diet. Greater consistency would make comparisons of prevalence estimates across studies more meaningful.

For instance, the estimated population attributable risk in the Planet Health example in the previous section is appreciably smaller than the estimates given for the Patton *et al.* [80] and Drewnowski *et al.* [82] studies. One possible reason for this discrepancy may be that the Planet Health study survey uses a much shorter time frame for the definition of dieting than the other two studies, leading to the potential misclassification of casual dieters—those who diet periodically but had not done so in the 30 days prior to the survey—as nondieters. With the 30-day cutoff used to categorize dieters, we can expect that all of the dieters who diet every month, half of the dieters who diet during 6 months of the year, and only one-twelfth of the dieters who diet during one month of the year will be categorized in the exposed group. As a result, casual dieters

are most likely to be misclassified as nondieters. Misclassification of the exposed as unexposed has the effect of attenuating the estimated population attributable risk of the exposure [85]. That said, a population attributable risk of 36% is nontrivial, regardless of the question of attenuation.

A broader and more complicated issue in the measurement of dieting is that dieting is a colloquial term and not a scientifically validated, specifically defined construct. Because dieting is chiefly a lay concept, survey respondents who report that they are dieting may be engaging in a range of different behaviors that each believes constitutes dieting. Surveys usually only partly define dieting for respondents, and its meaning has not been fully explored by researchers. Dieting is multidimensional, and the behavior may be described in terms of duration, recency, motivation, strategies used, intensity, frequency of starting and ending dieting phases, and meaning attributed to “successes” and “failures” [40,61,86].

Dieting may entail a number of different techniques—only some of which may involve a reduction in total caloric intake—and may differ by population subgroups defined by age, gender, body weight and shape, ethnicity, and other characteristics [40,53,86–88]. For instance, prospective study of adults found self-reported dieting to be associated with reduced consumption of French fries, sodas, and sweets [89]. A study of high school students found self-reported efforts to lose weight to be positively associated with meal skipping, caloric restriction, and physical activity [90]. In a sample of girls in grades 5 through 12, Field *et al.* [53] found that self-report of dieting was associated with lower caloric intake compared to nondieting peers for high school students, but not for younger girls in the sample. They propose that for younger girls, rather than indicating caloric restriction, a self-report of dieting may be capturing heightened concern with weight and behavioral changes such as consumption of products marketed as low-calorie or low-fat, such as diet soda, or refusal of certain foods perceived as forbidden. Field *et al.* [53] did not find frequency of dieting to be correlated with physical activity.

The sequelae of dieting may vary depending on patterns within the different dimensions of dieting. For instance, the psychological and physiological consequences of maintaining a total caloric intake of 1000 kcal per day may be very different from those of simply avoiding desserts. Given the heterogeneous constellation of common dieting behaviors, the current measures of dieting produce very broad exposure categories. A wide range of behaviors may all be classified as dieting, that is, as exposed. As a result, relative risk estimates are likely to be attenuated. For instance, if the exposure is defined so that such widely divergent behaviors as

⁴ For a detailed description of the study design and methodology used in the Planet Health study, please see Gortmaker *et al.* [83].

restricting dietary intake to 1000 kcal per day and simply avoiding desserts are both classified as exposed, then relative risk estimates associated with dieting may be expected to be biased toward the null value. More research will be needed to validate measures of dieting and to determine if there are subtypes of dieting that pose greater risk than others for development of eating pathology [91].

Whether dieting or certain subtypes of dieting should be understood as a distinct causal factor for eating pathology or part of the prodromal syndrome is open to debate, but in the context of prevention, the more important question is whether deterring dieting will lead to a reduction in the incidence of disordered eating. The issue of maximizing the impact of prevention efforts on incidence will be taken up in the next section.

THE CASE FOR POPULATION-BASED PREVENTION

Rose illustrates his argument for population-based prevention with the example of blood pressure, an important risk factor for CVD. Blood pressure is more or less unimodally and normally distributed in populations, a seemingly unextraordinary feature that is, in fact, instrumental to Rose's model. A statistical property of normal curves is that the mean determines the tail of the distribution, in that the top and bottom 5% will fall approximately 2 standard deviations in either direction from the mean. With a risk factor like blood pressure, incidence of disease is highest at one tail of the distribution. Rose describes the high-risk prevention strategy as essentially an attempt to lop off the tail of the distribution. In his example of blood pressure, a high-risk strategy might be to locate people above a certain cutoff point and then treat them with antihypertensive medication to lower their blood pressure to a less risky level. This strategy will reduce the number of cases of disease arising from the small proportion of the population that falls in the high-risk tail of the

exposure distribution. With many risk factors, however, most cases in a population arise from the moderate risk range, because that is where the vast majority of the population falls in the exposure distribution, whereas only a tiny fraction generally falls at the extreme. This is a paramount point in Rose's argument: Even while a small number of people may be at high risk, if a large number of people is at small risk, they will produce the bulk of the cases of a disease in a society. As Rose demonstrates, this is the pattern with hypertension and CVD. He advocates the population-based prevention strategy, which in contrast to the high-risk strategy, aims to shift the exposure distribution so that the whole population, including, most importantly, the vast majority who are in the moderate-risk zone, will shift into a lower risk range. In the context of blood pressure, a population-based approach would entail some type of large-scale change, such as a population-wide change in diet or exercise, that would result in a shift in the mean of the blood pressure distribution.

An Application of the Rose Model to Four Dieting Examples

Following Rose's lead, this article lodges a statistically grounded argument for population-based prevention of eating disorders. To be compatible with the model, an exposure–outcome relationship should satisfy two requirements. First, the exposure ideally should be operationalized in the form of a continuous variable with a normal distribution of values with tails and a mean, or, alternatively, an ordinal variable with multiple levels so that it presents a range of values approximating a normal distribution. Second, the incidence of the undesirable outcome must increase at elevated levels of exposure to the risk factor. If these conditions are met, then we can expect that large proportion of the cases in the total population will arise from the moderate range of exposure. In the following section,

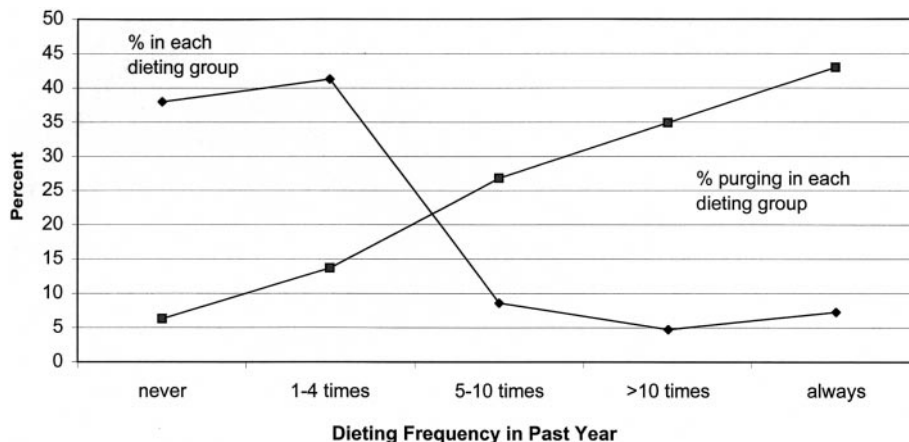


FIG. 1. Dieting frequency and purging in past year, girls in grades 7–12 in Minnesota ($n = 17,135$). Data from French *et al.* [51].

four tentative examples will be offered to justify this argument.

Example 1. The first example uses frequency of dieting, defined as an ordinal variable with five levels. In the large cohort study of Minnesota adolescents mentioned above, students in grades 7 through 12 were surveyed about a range of health behaviors, including frequency in the past year of dieting and purging, defined by the authors as induced vomiting or abuse of laxatives, ipecac, or diuretics [51]. French and colleagues dichotomized purging behavior (yes/no) and categorized frequency of dieting in the past year into five groupings: never, 1 to 4 times, 5 to 10 times, more than 10 times, and always. These five groupings roughly approximate a normal distribution, the first condition of the Rose model. Although they did not report the risk of purging by dieting frequency category, French and colleagues did publish the raw data, which, for girls, show a clear, steady increase in the percentage who purge associated with increasing dieting frequency categories. Figure 1 depicts a plot of the raw data on the dieting behavior of girls. For boys, as shown in Fig. 2, there appears to be a similar but smaller increase. These two figures show that the risk of purging rises as dieting becomes more frequent, therefore satisfying the second condition of the Rose model.

As predicted by the model, a large proportion of cases arises from the moderate range of exposure, which can be demonstrated clearly with the data for girls. While 21% of the purgers (cases) in the sample were found among the most frequent dieters (the group reporting that they always dieted in the past year, representing the girls with the highest dieting exposure), fully three times that many—63%—of the purging cases were found among the girls reporting lower levels of dieting, ranging from the categories 1 to 4 times to 10 or more

times in the past year (see Fig. 3). Although it may seem a paradox, the reason why two-thirds of the cases occur in the moderate exposure group is just as Rose argues: A large number of people exposed to small risk will often produce more cases in a population than will a small number of people at large risk.

Example 2. While the Minnesota data are rich in detail of dieting frequency, the study unfortunately is cross-sectional. A second example instead draws on prospective data. In a longitudinal study with college women, described in the previous section, Drewnowski *et al.* [82] collected survey data on dieting behavior in the fall semester of the students' first year and again 6 months later. Women who did not engage in fasting or purging (defined in this study as vomiting or abuse of laxatives or diuretics) at baseline were classified into three groups according to their dieting severity as nondieters, casual dieters, or regular dieters.

As mentioned above, women who dieted regularly at baseline had 3.3 (95% CI: 1.0, 10.7) times the unadjusted odds of initiating purging or fasting (becoming cases) at follow-up compared to nondieters. Casual dieters showed a nonsignificant trend toward elevated risk compared to nondieters (1.7; 95% CI: 0.5, 5.5). The estimated risk of becoming a case at follow-up for casual dieters falls about midway between that experienced by regular dieters and nondieters. The regular dieters at baseline, who were at greatest risk, produced 53% of the cases at follow-up. But because the casual dieters made up the largest group of noncases at baseline, fully 40% of the cases at follow-up arose from this group, even though they were at lower risk than regular dieters. In this example using prospective data, we can see how, as Rose's model predicts, a sizable proportion of cases in the population arises from those at moderate risk.

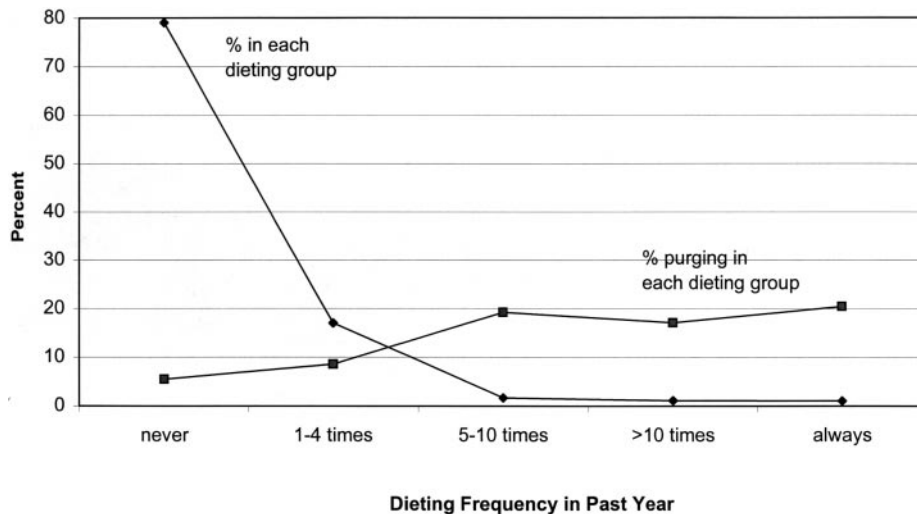


FIG. 2. Dieting frequency and purging in past year, boys in grades 7–12 in Minnesota ($n = 16,258$). Data from French *et al.* [51].

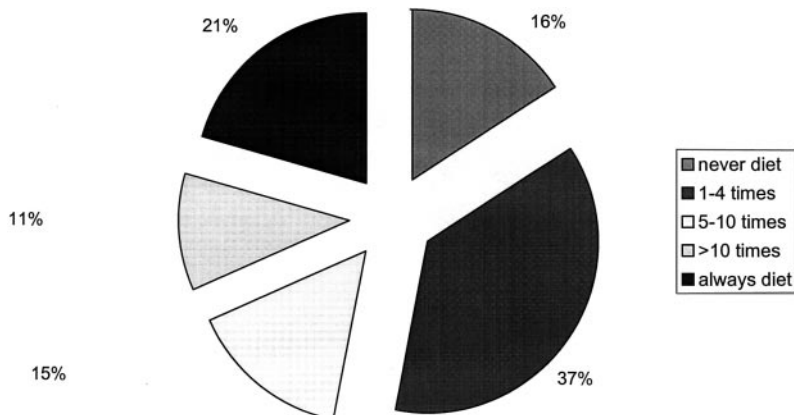


FIG. 3. Percentage of purging caseload occurring in each dieting frequency category in Minnesota, girls in grades 7–12 ($n = 2,596$). Data from French *et al.* [5].

Example 3. A third example draws on prospective data from the Planet Health nutrition and physical activity intervention study [83]. In this study, 1,295 sixth- and seventh-grade boys and girls from 10 middle schools in the Boston area provided survey data on their nutrition, physical activity, and dieting behavior. Data were collected over 2 school years beginning in the fall of 1995. Students were asked if they had dieted in the past 30 days and if they had induced vomiting or taken laxatives or diet pills to lose or maintain weight in the past 30 days. Using three mutually exclusive categories of dieting frequency, students were categorized at baseline as nondieters, moderate dieters, and frequent dieters—hypothesizing that frequency of dieting at baseline will predict initiation of purging (induced vomiting or laxative use) or diet pill use by follow-up in 1997. Students who reported that they were already engaging in these unhealthy behaviors at baseline were excluded for the longitudinal analysis. Figure 4 depicts the percentage of girls in each of the three dieting frequency categories at baseline and the percentage initiating purging or diet pill use at follow-up.

Controlling for intervention status, girls who reported dieting frequently (more than once a week in the past month) at baseline had 4.6 (95% CI: 1.4, 15.5) times the odds of becoming cases at follow-up compared to nondieters. Moderate dieters (once a week or less often in the past month) had 2.5 (95% CI: 0.6, 10.2) times the odds compared to nondieters, although the confidence interval included the null value. Similar to the relationship found in the data from Drewnowski and colleagues [82] described above, the estimated risk of becoming a case at follow-up for moderate dieters falls about midway between that of frequent dieters and nondieters. The frequent dieters at baseline, the group at greatest risk, produced 19% of the incident cases, but 33% of the new cases at follow-up arose from the group of moderate dieters.

Interestingly, among the subset of obese⁵ girls in the Planet Health study, there was a similar dose/response

⁵ Girls scoring at or above the age- and sex-standardized 85th percentile on both BMI and triceps skinfold thickness measures were classified as obese (Must *et al.*, 1991).

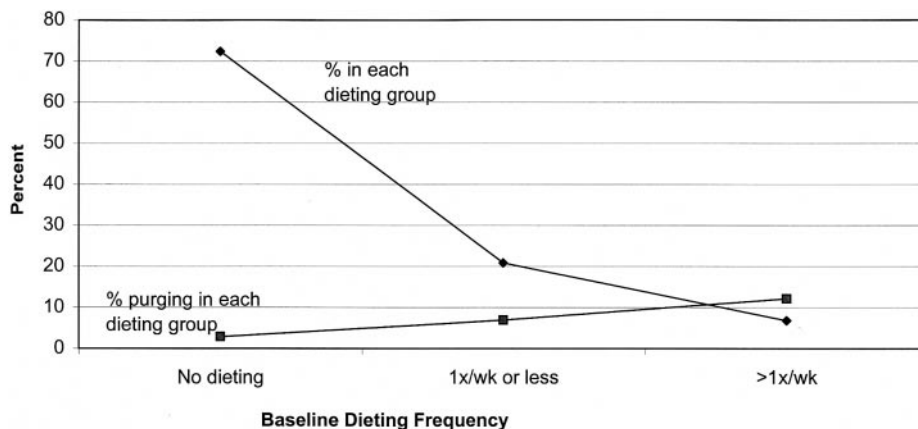


FIG. 4. Baseline dieting frequency and initiation of purging/diet pill use, girls in Planet Health Study ($n = 485$). Girls reporting purging or diet pill use at baseline excluded.

pattern in the association between dieting frequency and the risk of initiating purging, but the odds appeared to be attenuated compared to that of nonobese girls. Consistent with other research [40,53–58], body weight was positively associated with dieting in the Planet Health sample. At baseline, a slightly larger proportion of obese girls reported purging or using diet pills compared to other girls (5.8 versus 3.3%), and at follow-up, a slightly smaller proportion of obese girls had initiated these behaviors compared to nonobese girls (3.0 versus 4.6%), although the differences were nonsignificant. When the sample was stratified by obesity status, it appeared that the dose–response relationship for dieting frequency and initiation of eating pathology was of a higher magnitude for nonobese than for obese girls. Among the nonobese girls, those who dieted frequently at baseline had 8.0 (95% CI: 1.0, 61.4) times the odds of becoming cases at follow-up compared to nondieters, controlling for intervention status; moderate dieters had 3.4 (95% CI: 0.9, 13.6) times the risk compared to nondieters. The risk estimates among the subset of obese girls were somewhat lower and were statistically insignificant. Among the obese girls, those who dieted frequently at baseline had 2.5 (95% CI: 0.2, 38.2) times the odds of becoming cases at follow-up compared to nondieters, controlling for intervention status; moderate dieters had 1.3 (95% CI: 0.1, 22.6) times the risk compared to nondieters.

If obesity does modulate the relationship between dieting frequency and initiation of eating pathology for girls, one explanation may be that other factors besides dieting frequency disproportionately affecting obese girls, such as weight-related harassment and stigma, are driving up the rates of purging in the less frequent dieting groups, thus attenuating the relative odds. Importantly, obese girls are not less likely to purge or take diet pills than nonobese girls; rather, purging is more evenly distributed among the dieting frequency categories for obese girls. Another explanation may be that misclassification of exposed as unexposed may be more of a problem with obese girls than nonobese girls. The Planet Health survey asked respondents to report dieting behavior in the previous month. Perhaps a large proportion of nonobese girls who had not dieted in the past month had in fact never dieted in their lifetimes; whereas, perhaps among obese girls, many of those who had not dieted in the previous month had actually had periods of dieting in the past. As a word of caution, however, it must be noted that the subsample of obese girls was too small to draw any firm conclusions about how the relationship between dieting and purging initiation may differ for obese versus nonobese girls.

Example 4. The fourth example of an application of

the Rose model to eating pathology proposes an alternate way to operationalize dieting for population clusters rather than for individuals. Rather than analyze the risk experienced by individuals associated with their own dieting behavior, instead it is possible to analyze the risk experienced in a population cluster associated with the aggregate behavior of community members. This approach uses an aggregated measure of dieting pooled across data collected from individuals. For example, dieting can be operationalized as a continuous variable representing the proportion of people dieting within a community, such as students within a middle school. The proportion of people dieting in a community represents an ecological indicator in that it is used to characterize a community rather than an individual. Similarly, eating pathology can be operationalized as the proportion of people engaging in a variety of purging behaviors in a community.

Research in social psychology has documented that people tend to adopt behaviors that are the norm among their peers and in their community. For instance, studies of college students and Greek society houses have found this pattern in alcohol consumption [92,93]. One study found that among students who were not heavy drinkers in high school, those who joined fraternities or sororities were more likely than other students to become heavy drinkers in college [92]. Greek society members who lived in fraternity or sorority houses were even more likely than nonresident members to become heavy drinkers. These findings suggest that for society members, living in the cohesive community of fraternity and sorority houses, where high alcohol consumption is the norm, adds additional risk for individuals adopting similar behavior.

Similar patterns have been found with disordered eating behavior. In a prospective study of social influences on binge eating in college sororities, Crandall collected survey data from women on their friendship clusters and eating patterns [94]. She found that the degree of binge eating became more similar among members of friendship clusters over the course of the school year. Interestingly, friendship clusters were very stable over the year. It was the binge eating patterns that changed. She also found that binge eating was associated with popularity, so that the most popular members were those who binged in an amount close to the mean level for the sorority, and the least popular were those who binged comparatively very little or very much. The research on college alcohol consumption and binge eating suggests that the dieting behavior of individuals is likely to be influenced by the norms of their community. The following example considers this issue in middle schools, again using data taken from the Planet Health study [83].

In a plot of the raw baseline data from girls in the Planet Health study, it appears that there may be a

positive association between the proportion of students reporting dieting (without induced vomiting or abuse of laxatives or diet pills) to lose weight in the past month and the proportion engaging in purging or diet pill use. As shown in Fig. 5, a positive association appears plausible. A preliminary univariate linear regression model using the data for girls suggests that with each 5% rise in dieting (without purging or diet pill use) in a middle school, there is a 1% rise in purging and diet pill use in the school ($\beta = 0.20$; $P = 0.09$). Thirty-two percent of the between-school variance in purging and diet pill use in schools was explained by the variance in dieting ($R^2 = 0.32$).

According to this model, we can estimate the prevalence of purging and diet pill use in the past month in a middle school based on how normative dieting is in the school. For example, take hypothetical School A, where 5% of the girls diet. If there were a total of 500 girls in the school, that would translate into 25 girls dieting. Based on the regression model, in this same school, we would expect 1%—or 5 girls—to have induced vomiting or abused laxatives or diet pills in the previous month. Now consider a school of the same size with a high prevalence of dieting among girls, say 40%, which would mean 200 girls report dieting in the past 30 days. In this school, School B, we would expect over 8%—or 41 of the girls—to have recently used purging methods or used diet pills. Compared to School A, School B has an estimated 36 additional cases of induced vomiting or abuse of laxatives or diet pills associated with the high prevalence of dieting in the student body.

As in the previous examples, the next point to consider is what proportion of cases in the total sample arise from high-risk schools (those where many girls diet) and from moderate-risk schools (those where fewer girls diet). In the Planet Health sample, the proportion of girls dieting ranged from 8% in the lowest school

to 45% in the highest school, with most percentages clustered in the middle. In 6 of the 10 schools, about 20 to 30% of the girls dieted; at 3 schools about 35 to 45% dieted; and at 1 school, less than 10% dieted (see Fig. 6). Based on this distribution of dieting prevalence across the 10 schools, it is possible to estimate the proportion of cases in the total population of girls expected to arise from the low-exposure, moderate-exposure, and high-exposure schools. Using the regression model above, we would expect 2% of the total cases to occur in the low-exposure school, 44% in the high-exposure schools, and 54% in the moderate-exposure schools.⁶ (see Fig. 7). More than half of the total caseload, then, is expected to occur in schools with only a moderate prevalence of dieting.

Taking this example one step further, it is also possible to estimate the number of cases that could be prevented by shifting downward the prevalence of dieting among girls in all the schools, say 10%. With a hypothetical sample of 1,000 girls in schools with the expected⁷ range in dieting prevalence, we would predict 57 of the girls to have induced vomiting or abused laxatives or diet pills in the past month. If the prevalence of dieting were reduced in all the schools by 10%, the number of expected cases would drop to 37. More than a third of the cases would be prevented by a 10% decline in dieting in the schools.

⁶ For the three regression equations, the mid-range exposure values for each of the three school groupings were used. Based on the distribution of prevalence of girls dieting in the Planet Health schools, low-exposure schools were defined as having a dieting prevalence of 10% or below, so the middle value used in the equation was 5%. Moderate-exposure schools were defined as having 20 to 30% prevalence of dieting, so the middle value was 25%. High-exposure schools were defined as having 35% to 45% prevalence of dieting, and the middle value used in the regression equation was 40%.

⁷ Expected exposure values are based on the actual distribution of exposures across the 10 Planet Health schools.

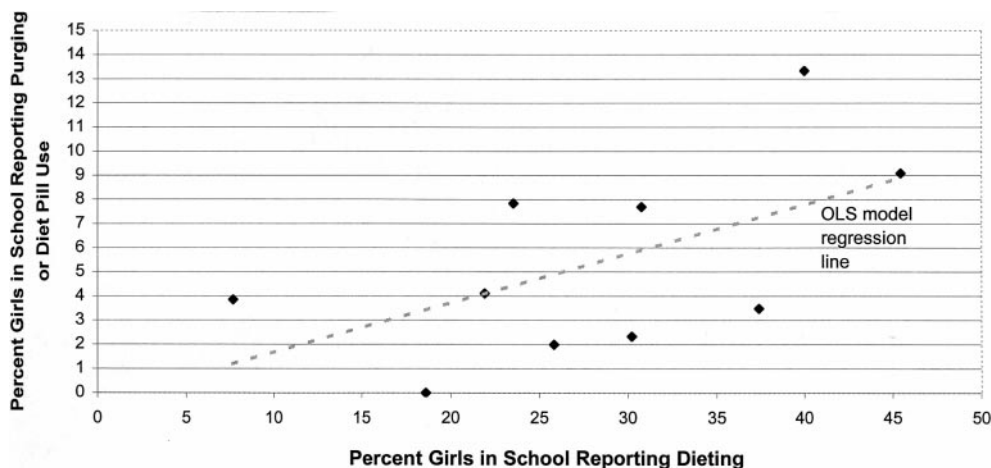


FIG. 5. Percentage reporting purging (vomiting or abuse of laxatives) or diet pill use by percentage reporting dieting, girls only, in 10 Planet Health Middle Schools (1995).

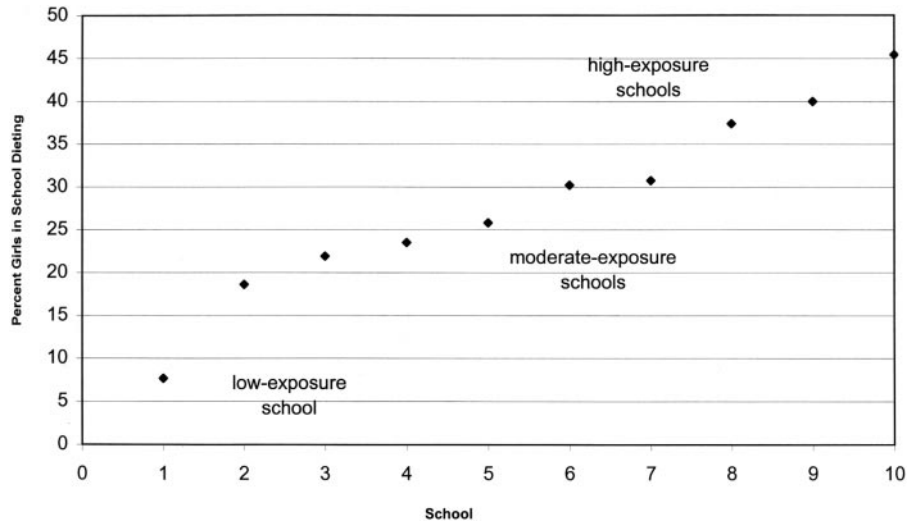


FIG. 6. Distribution of percentage girls dieting across 10 Planet Health Middle Schools.

Given the marginal P value of the model's beta coefficient and the availability of data from only 10 schools, this example should be considered with caution. The results, however, do present some evidence of the following: (a) As dieting becomes more prevalent within a cohesive community (in this case, among girls within a middle school community), disordered eating behavior may also become more prevalent; (b) the majority of cases may occur in schools with average levels of dieting students rather than in the smaller number of schools with very high prevalence of dieting; and, (c) a reduction in dieting in schools may have a sizable impact on the incidence of induced vomiting and abuse of laxatives and diet pills. In the very least, this example extends the operationalization of dieting from the standard individual-level measure to an ecological measure of a population cluster and offers a glimpse at how an alternate approach to measurement integrated with Rose's model can point toward novel insights on potentially effective strategies for the prevention of eating pathology.

Limitations of the Rose Model

When Rose originally proposed his argument for population-based prevention, critics pointed out a potential flaw in his model in dealing with nonlinear exposure effects. If a risk factor has a U -shaped (or otherwise multimodal) exposure–effect curve, then it may have harmful effects at both very high and very low doses. Shifting the population as a whole so that some people move into a lower level of risk may have the unintended consequence of moving others into a higher risk range. In describing this dilemma, Lorion [95] offers the simple illustration of light as a U -shaped exposure effect, where a person may experience heightened stress when living under conditions of either too much light or too little light. Above, in the application of Rose's population-based prevention strategy to disordered eating, it was proposed that high levels of dieting in a population contribute to high rates of disordered eating and that a downward shift in population patterns of dieting would

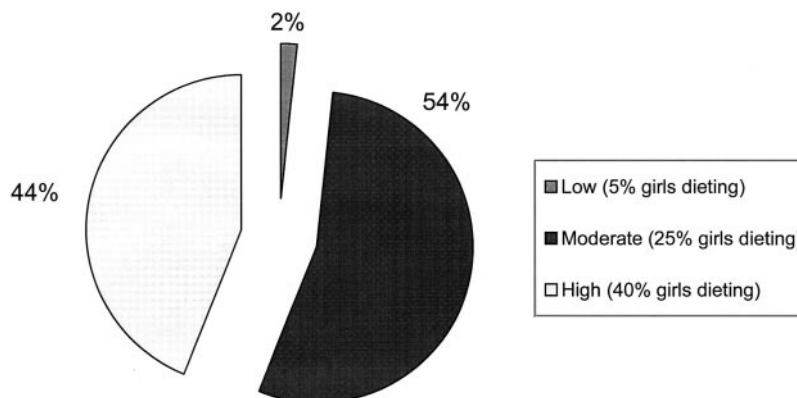


FIG. 7. Expected percentage of purging caseload occurring in each school exposure level category, based on OLS model, girls only. OLS model based on Planet Health baseline data from girls.

reduce the incidence of eating pathology. But the possibility of a *U*-shaped exposure effect forces the question of whether there may also be morbidity associated with low levels of dieting.

There is no evidence to suggest that very low levels of dieting increase the risk of eating disorders, but would a reduction in dieting on the population level lead to an increase in the incidence of obesity? There are at least two reasons why the answer to this question is probably no. One, ecological evidence suggests that widespread dieting in our society has not solved the problem of obesity. Over the past four decades in this country, as the prevalence of dieting has skyrocketed [48–50], so has the prevalence of obesity [96]. Certainly this may be merely an ecological association. Some have speculated that widespread dieting may have had a depressing effect on the rate of increase in overweight in our population [97], although there is no evidence yet to support or refute this hypothesis. What is equally unknown about the historical effect mass dieting has had on our society is whether it may have had the ironic consequence of exacerbating population trends toward increasing overweight. Plausible mechanisms for such an untoward effect of dieting could be either by triggering cycles of overeating among a subset of dieters when their diets “fail” or by distracting people, including health professionals, from attention to other more effective and healthful methods of weight control. These points again are speculative but underscore the need for more research on the historical effects of mass dieting on our population. In the very least, it is not overly speculative to suggest that dieting has been wholly inadequate as a societal strategy to reduce the incidence of obesity.

A second reason why a population reduction in dieting is not likely to increase the incidence of obesity is that several prospective epidemiologic studies in community samples have not found dieting to be predictive of consistent weight loss and maintenance [89,98,99]. Evidence from these longitudinal studies suggests that dieting, in the ways that it is generally practiced in the population, appears to be ineffective. For many people, it may instead do more to amplify weight fluctuation [98,100]. More disturbingly, evidence from a recent observational study with adolescent girls found dieting to be prospectively associated with an increased risk of obesity onset [101].

Much of the current research in the obesity prevention field has moved away from a focus on dieting, with the understanding that behavior change based on restriction and denial is difficult to sustain. Preventive interventions with young people have moved toward an emphasis on increasing fruit and vegetable intake and physical activity, which unlike dieting, have the added benefits of decreasing risk of some of the major cancers and CVD [83,102]. In addition, exercise can improve

mental health, physical stamina, and strength [103]. Again, dieting offers none of these benefits. For these reasons, mass dieting is not considered a priority goal in obesity prevention research.

While the potential dilemma of the *U*-shaped exposure–effect curve may appear on the surface to mean that the goals of eating disorders prevention are at odds with those of obesity prevention, in fact, they can be very much in sync and mutually reinforcing. Ideally, even, health promotion efforts with children and adolescents to deter dieting should go hand in hand with those that encourage fruit and vegetable consumption and moderate physical activity. The rising incidence of obesity in children and adults is undoubtedly a serious public health issue [104]. It is also true that dieting is widely considered by lay people and health professionals alike to be an advisable strategy to combat this problem. It must be emphasized, though, that calling into question the value placed on dieting does not necessarily mean that one is also dismissive of the obesity epidemic. On the contrary, inasmuch as dieting enjoys a degree of legitimacy it does not warrant, it represents a perilous distraction from efforts toward effective public health responses to the epidemic.

CONCLUSION

All four of the examples provided above are tentative and limited in different ways, as has been pointed out. But while they are imperfect, they do suggest a unique and provocative way to think about population-based prevention of disordered eating. It is possible that an unknown third factor not addressed in the examples may explain the association between dieting and disordered eating and be driving the rates of both behaviors. The evidence presented above, however, is both cross-sectional and prospective. In addition, a growing body of experimental research has provided further evidence that dieting may be an important causal factor in the development of eating disorders and a plausible underlying mechanism has been proposed. This article details an epidemiological argument that dieting is an important behavioral risk factor for eating pathology in addition to a statistically grounded argument that the dieting-disordered eating relationship meets the conditions for Rose’s model of population-based prevention. It was demonstrated statistically that, just as Rose’s model predicts, a downward shift in exposure to dieting in the population as a whole—and not just in those at the high-exposure tail—can potentially lead to the greatest reduction in the incidence of disordered eating.

Brownell and Rodin [97] offer perhaps a more tempered assessment of dieting than the one presented in this article, as they argue that while our culture’s largely uncritical and indiscriminate advocacy of dieting should be challenged, a wholesale condemnation of

dieting regardless of a person's circumstances may itself be an overreaction. Their point is well-taken, and although a more stalwart anti-dieting position may yet prove to have merit [105, 106], the aim of this article is not to take on the broad question of whether dieting ever accrues health benefits under certain conditions for some individuals.⁸ Instead, the focus of this article has been to present the evidence that dieting in the various forms undertaken by children and adolescents, particularly girls, substantially elevates their risk of disordered eating and that a reduction in the prevalence of dieting is likely to reduce the incidence of disordered eating.

Brownell and Rodin [97] also propose that rather than debating the value of dieting in all-or-nothing terms, we should consider instead the risk/benefit ratio on an individual basis. But by weighing risk and benefit solely in individual terms, Brownell and Rodin's proposal assumes that the decision whether to diet has no impact on anyone else in the community or on the society as a whole. The findings presented in this article suggest, however, that this assumption is faulty on two counts. One, when a society experiences mass exposure to a small risk, even while the chances of developing the associated disease or disorder are low for any one individual, the incidence of cases for the population as a whole can still be significant.

Two, our society is not a collection of atomized dieters, independently living a personal dieting experience independent of others around them. In the previous section, the last example illustrated that, for at least the Planet Health study sample, the proportion of girls dieting without purging or using diet pills in middle schools appeared to be predictive of the proportion engaging in these unhealthful weight-control methods. One interpretation of this finding is that it appears that the decision to diet made by large numbers of students in middle schools may have consequences for a subset of their peers, perhaps in part by contributing to a normative school climate of mass dieting. If this is true, then an individual's engagement in dieting cannot be considered an isolated act, and the risk/benefit ratio of dieting cannot be considered simply in terms of that individual. The evidence presented in this article suggests that dieting carries consequences for individuals, but it also appears to carry consequences for other members of the community and for the community as a whole.

A basic tenet in the field of public health is that risk and benefit, exposure and disease, cannot be fully

understood through individual-level analysis. *Population* patterns in exposures and the consequences they pose for communities and for society as a whole must also be considered. Enormous segments of our population engage in dieting, and for the majority of women, dieting is in fact "normal" eating [107]. Accumulating research suggests that dieting increases risk of eating pathology, and the more frequently people diet, the greater their risk. But the greatest burden of eating pathology is not arising from the small percentage of girls whose dieting frequency is at the extreme. It is the moderate dieters—the "normal" eaters—making up the bulk of the cases. Perhaps it is time that a population-level perspective on patterns in exposure and incidence be brought to bear on the problem of dieting and eating pathology. Rose's model of population-based prevention may hold a key to the prevention of disordered eating in our society.

ACKNOWLEDGMENTS

The author thanks William DeJong, Anne Becker, Deborah Blacker, and Steven Gortmaker for invaluable insights on earlier drafts of the manuscript.

REFERENCES

1. Striegel-Moore RH. Risk factors for eating disorders. Adolescent nutritional disorders: prevention and treatment. *Annals of the New York Academy of Sciences* (v. 817). New York: New York Academy of Sciences, 1997:98–109.
2. Ponton LE. A review of eating disorders in adolescents. *Ann Am Soc Adolesc Psychiatry* 1995;20:267–85.
3. Crowther JH, Tennenbaum DL, Hobfoll SE, Stephens MAP. The etiology of bulimia nervosa: the individual and familial context. Washington, DC: Hemisphere, 1992.
4. Shisslak CM, Crago M. Toward a new model for the prevention of eating disorders. In: Fallon P, Katzman MA, Wooley SC, editors. *Feminist perspectives on eating disorders*. New York: Guilford Press, 1994:419–33.
5. Striegel-Moore RH. Prevention of bulimia nervosa: questions and challenges. In: Crowther JH, Tennenbaum DL, Hobfoll SE, Stephens MAP. *The etiology of bulimia nervosa: the individual and familial context*. Washington, DC: Hemisphere, 1992.
6. Striegel-Moore RH, Silberstein LR, Rodin J. Toward an understanding of risk factors for bulimia. *Am Psychol* 1986;41(3): 246–63.
7. Garfinkel PE, Garner DM. *Anorexia nervosa: a multidimensional perspective*. New York: Brunner/Mazel, 1982.
8. Brewerton TD. Toward a unified theory of serotonin dysregulation in eating and related disorders. *Psychoneuroendocrinology* 1995; 20(6): 561–90.
9. Jimerson DC. The role of central catecholamine pathways in eating disorders. In: Ferrari E, Brambilla F, Solerte SB, editors. *Primary and secondary eating disorders: a psychoneuroendocrine and metabolic approach*. Oxford: Pergamon, 1993:59–64.
10. Shisslak CM, Crago M. Toward a new model for the prevention of eating disorders. In: Fallon P, Katzman MA, Wooley SC, editors. *Feminist perspectives on eating disorders*. New York: Guilford Press, 1994:419–33.

⁸ For instance, experimental obesity treatment protocols that include calorie restriction under close supervision of clinicians, especially when combined with cognitive and behavioral therapy, have been found to lead to weight loss without increased eating pathology (e.g., Wadden *et al.* [108] and Yanovski and Sebring [109]).

11. Steiner-Adair C. The politics of prevention. In: Fallon P, Katzman MA, Wooley SC, editors. *Feminist perspectives on eating disorders*. New York: Guilford, 1994:381–94.
12. Rodin J, Striegel-Moore RH, Silberstein LR. Vulnerability and resilience in the age of eating disorders: risk and protective factors for bulimia nervosa. In: Rolf J, Masten AS, Cicchetti D, Nuechterlein KH, Weintraub S. *Risk and protective factors in the development of psychopathology*. New York: Cambridge Univ. Press, 1990:361–83.
13. Garner DM, Garfinkel PE. Socio-cultural factors in the development of anorexia nervosa. *Psychologic Med* 1980;10:647–56.
14. Becker AE, Hamburg P. Culture, the media, and eating disorders. *Harvard Rev Psychiatry* 1996;4:163–47.
15. Levine MP, Smolak L. Media as a context for the development of disordered eating. In: Smolak L, Levine MP, Striegel-Moore R, editors. *The developmental psychopathology of eating disorders: implications for research, prevention, and treatment*. Mahwah, NJ: Erlbaum, 1996:235–57.
16. Bordo S. *Unbearable weight: feminism, western culture, and the body*. Berkeley: Univ of California Press, 1993.
17. Vandereycken W. The sociocultural roots of the fight against fatness: implications for eating disorders and obesity. *Eating Disorders: J Treat Prev* 1993;1(1):7–16.
18. Raphael FJ, Lacey JH. Sociocultural aspects of eating disorders. *Ann Med* 1992;24(4):293–6.
19. Paxton SJ. Prevention implications of peer influences on body image dissatisfaction and disturbed eating in adolescent girls. *Eating Disorders: J Treat Prev* 1996;4(4):334–7.
20. Cattarin JA, Thompson JK. A three-year longitudinal study of body image, eating disturbance, and general psychological functioning in adolescent females. *Eating Disorders: J Treat Prev* 1994;2(2):114–25.
21. Shisslak CM, Crago M, Neal ME, Swain B. Primary prevention of eating disorders. *J Consult Clin Psychol* 1987;55(5):660–7.
22. Stokols D. Translating social ecological theory into guidelines for community health promotion. *Am J Health Prom* 1996;10(4):282–98.
23. Rosen JC. Prevention of eating disorders. *National Anorectic Aid Society Newsletter* 1989; April–June: 1–3.
24. Rose G. *The strategy of preventive medicine*. New York: Oxford Univ Press, 1995.
25. Rose G. Sick individuals and sick populations. *Int J Epidemiol* 1985;14(1):32–38.
26. Caplan G. *Principles of preventive psychiatry*. New York: Basic Books, 1964.
27. Rose G. The mental health of populations. In: Williams P, Wilkinson G, Rawnsley K, editors. *The scope of epidemiological psychiatry*. London: Routledge, 1989: 77–85.
28. Hsu LKG. Epidemiology of eating disorders. *Psychiatric Clinics N Am* 1996;19(4):681–700.
29. American Psychiatric Association. *Diagnostic and statistical manual of mental disorders*, fourth edition. Washington, DC: American Psychiatric Association, 1994.
30. Kann L, Kinchen SA, Williams BI, Ross JG, Lowry R, Grunbaum JA, Kolbe LJ. Youth risk behavioral surveillance—United States, 1999. In: *CDC Surveillance Summaries*, June 9, 2000. *MMWR* 2000; 49(No. SS-5):1–96.
31. Fombonne E. Eating disorders: time trends and possible explanatory mechanisms. In: Rutter M, Smith DJ, editors. *Psychosocial disorders in young people: time trends and their causes*. New York: Wiley, 1995.
32. Killen JD, Taylor CB, Hammer LD, Litt I, Wilson DM, Rich T, Hayward C, Simmonds B, Kraemer H, Varady A. An attempt to modify unhealthful eating attitudes and weight regulation practices of young adolescent girls. *Int J Eating Disorders* 1993;13(4):369–84.
33. Childress AC, Brewerton TD, Hodges EL, Jarrell MP. The Kids' Eating Disorders Survey (KEDS): a study of middle school students. *J Am Acad Child Adolesc Psychiatry* 1993;32(4):843–50.
34. Fairburn CG, Beglin SJ. Studies of the epidemiology of bulimia nervosa. *Am J Psychiatry* 1990;4:402–8.
35. Hoek HW. The distribution of eating disorders. In: Brownell KD, Fairburn CG, editors. *Eating disorders and obesity: a comprehensive handbook*. New York: Guilford, 1995: Chap. 36.
36. Maloney MJ, McGuire J, Daniels SR, Specker B. Dieting behavior and eating attitudes in children. *Pediatrics* 1989;84(3): 482–9.
37. Killen JD, Taylor CB, Telch MJ, Robinson TN, Maron DJ, Saylor KE. Depressive symptoms and substance use among adolescent binge eaters and purgers: a defined population study. *Am J Public Health* 1987;77(12):1539–41.
38. Killen JD, Taylor CB, Telch MJ, Saylor KE, Maron DJ, Robinson TN. Self-induced vomiting and laxative and diuretic use among teenagers: precursors of the binge-purge syndrome? *J Am Med Assoc* 1986;255(11):1447–9.
39. Fairburn CG, Beglin SJ. Assessment of eating disorders: interview or self-report questionnaire? *Int J Eating Disorders* 1994;16(4):363–70.
40. French SA, Jeffery RW. Consequences of dieting to lose weight: effects on physical and mental health. *Health Psychol* 1994; 13(3):195–212.
41. Herzog W, Deter H-C, Fiehn W, Petzold E. Medical findings and predictors of long-term physical outcome in anorexia nervosa: a prospective, 12-year follow-up study. *Psychological Med* 1997;27:269–79.
42. Harris EC, Barraclough B. Suicide as an outcome for mental disorders: a meta-analysis. *Br J Psychiatry* 1997;170:205–28.
43. Nielsen S, Moller-Madsen S, Isager T, Jorgensen J, Pagsberg K, Theander S. Standardized mortality in eating disorders: a quantitative summary of previously published and new evidence. *J Psychosom Res* 1998;44(3,4):413–34.
44. Herzog DB, Keller MB, Sacks NR, Yeh CJ, Lavori PW. Psychiatric comorbidity in treatment-seeking anorexics and bulimics. *J Am Acad Child Adolesc Psychiatry* 1992; 31(5): 810–18.
45. Herzog DB, Copeland PM. Eating disorders. *N Engl J Med* 1985;313:295–303.
46. Johnson J, Whitaker AH. Adolescent smoking, weight changes, and binge-purge behavior: associations with secondary amenorrhea. *Am J Public Health* 1992;82(1):47–54.
47. Ho PC, Dweik R, Cohen MC. Rapidly reversible cardiomyopathy association with chronic ipecac ingestion. *Clin Cardiol* 1998; 21(10):780–83.
48. Horm J, Anderson K. Who in America is trying to lose weight? *Ann Intern Med* 1993;119(7):672–6.
49. Serdula MK, Collins ME, Williamson DF, Anda RF, Pamuk ER, Byers TE. Weight control practices of U.S. adolescents and adults. *Ann Intern Med* 1993;119(7):667–71.
50. Williamson DF, Serdula MK, Anda RF, Levy A, Byers T. Weight loss attempts in adults: goals, duration, and rate of weight loss. *Am J Public Health* 1992;82(9):1251–7.
51. French SA, Story M, Downes B, Resnick MD, Blum RW. Frequent dieting among adolescents: psychosocial and health behavior correlates. *Am J Public Health* 1995;85:695–701.
52. American School Health Association, Association for the Advancement of Health Education, Society for Public Health Education, Inc. *The National Adolescent Student Health Survey*; A

- Report on the Health of America's Youth. Oakland, CA: Third Party Publishing, 1989.
53. Field AE, Wolf AM, Herzog DB, Cheung L, Colditz GA. The relationship of caloric intake to frequency of dieting among preadolescent and adolescent girls. *J Am Acad Child Adolescent Psychiatry* 1993;32(6):1246–52.
 54. Krahn D, Kurth C, Demitrack M, Drownowski A. The relationship of dieting severity and bulimic behaviors to alcohol and other drug use in young women. *J Substance Abuse* 1992;4:341–53.
 55. Fisher M, Schneider M, Pegler C, Napolitano B. Eating attitudes, health-risk behaviors, self-esteem, and anxiety among adolescent females in a suburban high school. *J Adolescent Health* 1991;12:377–84.
 56. Frank RE, Serdula MK, Adame D. Weight loss and bulimic eating behavior: changing patterns within a population of young adult women. *South Med J* 1991;84(4):457–60.
 57. Story M, Rosenwinkel K, Himes JH, Resnick M, Harris LJ, Blum RW. Demographic and risk factors associated with chronic dieting in adolescents. *Am J Children* 1991;145:994–8.
 58. Moses N, Banilivy M, Lifshitz F. Fear of obesity among adolescent girls. *Pediatrics* 1989;83(3):393–8.
 59. Massachusetts Department of Education. 1997 Massachusetts Youth Risk Behavioral Survey results. Malden, MA: Massachusetts Department of Education, 1998.
 60. Polivy J, Herman CP. Dieting and bingeing: a causal analysis. *Am Psychologist* 1985;40:193–204.
 61. Heatherton TF, Polivy J. Chronic dieting and eating disorders: a spiral model. In: Crowther JH, Tennenbaum DL, Hobfoll SE, Stephens MAP. *The etiology of bulimia nervosa: the individual and familial context*. Washington, DC: Hemisphere, 1992.
 62. Stice E, Nemeroff C, Shaw HE. Test of the dual pathway model of bulimia nervosa: evidence for dietary restraint and affect regulation mechanisms. *J Social Clin Psychol* 1996;15(3):340–63.
 63. Keys A, Brozek J, Henschel A, Mickelsen O, Taylor HL. *The biology of human starvation (Vols 1 and 2)*. Minneapolis: Univ of Minnesota Press, 1950.
 64. Franklin JS, Schiele BC, Brozek J, Keys A. Observations on human behavior in experimental starvation and rehabilitation. *J Clin Psychol* 1948;4:28–45.
 65. Telch CF, Agras WS. The effects of short-term food deprivation on caloric intake in eating-disordered subjects. *Appetite* 1996;26(3):221–33.
 66. Smoller JW, Wadden TA, Stunkard AJ. Dieting and depression: a critical review. *J Psychosom Res* 1987;31(4):429–40.
 67. Wadden TA, Stunkard AJ, Smoller JW. Dieting and depression: a methodological study. *J Consult Clin Psychol* 1986;54(6):869–71.
 68. Wadden TA, Stunkard AJ. Social and psychological consequences of obesity. *Ann Intern Med* 1985;103(6, Pt 2):1062–7.
 69. Stunkard AJ, Rush J. Dieting and depression reexamined: a critical review of reports of untoward responses during weight reduction for obesity. *Ann Intern Med* 1974;81:526–33.
 70. Rosen JC, Gross J, Vara L. Psychological adjustment of adolescents attempting to lose or gain weight. *J Consult Clin Psychol* 1987;55(5):742–7.
 71. Edwards FE, Nagelberg DB. Personality characteristics of restrained/binge eaters versus unrestrained/nonbinge eaters. *Addict Behav* 1986;11:207–11.
 72. Herman CP, Polivy J. Anxiety, restraint, and eating behavior. *J Abnormal Psychol* 1975;84(6):666–72.
 73. Bulik CM, Sullivan PF, Carter FA, Joyce PR. Initial manifestations of disordered eating behavior: dieting versus bingeing. *Int J Eating Disorders* 1997;22:195–201.
 74. Fairburn CG, Cooper PJ. The clinical features of bulimia nervosa. *Br J Psychiatry* 1984;144:238–46.
 75. Johnson CL, Stuckey MK, Lewis LD, Schwartz DM. Bulimia: a descriptive survey of 316 cases. *Int J Eating Disorders* 1983;2:3–19.
 76. Abraham SF, Beumont PJV. How patients describe bulimia or binge eating. *Psychological Med* 1982;12:625–35.
 77. Pyle RL, Mitchell JE, Eckert ED. Bulimia: a report of 34 cases. *J Clin Psychiatry* 1981;42:60–4.
 78. Marcus MD, Moulton MM, Greeno CG. Binge eating onset in obese patients with binge eating disorder. *Addict Behav* 1995;20(6):747–55.
 79. Stice E, Akutagawa D, Gaggari A, Agras WS. Negative affect moderates the relation between dieting and binge eating. *Int J Eating Disorders* 2000;27(2):218–29.
 80. Patton GC, Johnson-Sabine E, Wood K, Mann AH, Wakeling A. Abnormal eating attitudes in London school girls—a prospective epidemiologic study: outcome at twelve month follow-up. *Psychological Med* 1990;20:383–94.
 81. Hennekens CH, Buring JE. *Epidemiology in medicine*. Boston: Little, Brown, 1987:92.
 82. Drownowski A, Yee DK, Kurth CL, Krahn DD. Eating pathology and DSM-III-R bulimia nervosa: a continuum of behavior. *Am J Psychiatry* 1994;151(8):1217–9.
 83. Gortmaker SL, Peterson K, Wiecha J, Sobol AM, Dixit S, Fox MK, Laird N. Reducing obesity via a school-based interdisciplinary intervention among youth: planet health. *Arch Pediatrics Adolescent Med* 1999;153(4):409–18.
 84. Austin SB. Dieting and disordered eating in early adolescent girls and boys: a prospective study, in preparation.
 85. Wacholder S, Benichou J, Heineman EF, Hartge P, Hoover RN. Attributable risk: advantages of a broad definition of exposure. *Am J Epidemiol* 1994;140(4):303–9.
 86. Jeffery RW, French SA, Schmid T. Attributions for dietary failures: problems reported by participants in the hypertension prevention trial. *Health Psychol* 1990;9(3):315–29.
 87. Field AE, Colditz GA, Peterson KE. Racial/ethnic and gender differences in concern with weight and in bulimic behaviors among adolescents. *Obesity Res* 1997;5(5):447–54.
 88. Robinson TN, Killen JD, Litt IF, Hammer LD, Wilson DM, Haydel KF, Hayward C, Taylor CB. Ethnicity and body dissatisfaction: are Hispanic and Asian girls at increased risk for eating disorders? *J Adolescent Health* 1996;19(6):384–93.
 89. French SA, Jeffery RW, Forster JL, McGovern PG, Kelder SH, Baxter J. Predictors of weight change over two years among a population of working adults: the healthy worker project. *International J Obesity* 1994;18:145–54.
 90. Rosen JC, Gross J. Prevalence of weight reducing and weight gaining in adolescent girls and boys. *Health Psychol* 1987;6(2):131–47.
 91. Tuschl RJ. From dietary restraint to binge eating: some theoretical considerations. *Appetite* 1990;14:105–9.
 92. Wechsler H, Kuh G, Davenport AE. Fraternities, sororities and binge drinking: results from a national study of American colleges. *National Assoc Student Personnel Administrators* 1996;33(4):260–79.
 93. Lo CC, Globetti G. The facilitating and enhancing roles Greek associations play in college drinking. *Int J Addictions* 1995;30(10):1311–22.
 94. Crandall CS. Social contagion of binge eating. *J Personality Social Psychol* 1988;55(4):588–98.

95. Lorion RP. Environmental approaches and prevention: the dangers of imprecision. In: Wandersman A, Hess R, editors. *Beyond the individual: environmental approaches and prevention*. New York: Haworth Press, 1985:193–205.
96. Kuczmarski RJ, Flegal KM, Campbell SM, Johnson CL. Increasing prevalence of overweight among US adults: the National Health and Nutrition Examination Surveys, 1960 to 1991. *J Am Med Assoc* 1994;272(3):205–11.
97. Brownell KD, Rodin J. The dieting maelstrom: is it possible and advisable to lose weight? *Am Psychologist* 1994;49(9):781–91.
98. Heatherton TF, Polivy J, Herman CP. Restraint, weight loss, and variability of body weight. *J Abnormal Psychol* 1991;100(1):78–83.
99. Klesges RC, Klem ML, Epkins CC, Kleges LM. A longitudinal evaluation of dietary restraint and its relationship to changes in body weight. *Addict Behav* 1991;16:363–8.
100. Brownell KD. Effects of weight cycling on metabolism, health and psychological factors. In: Brownell KD, Fairburn CG, editors. *Obesity and eating disorders: a comprehensive handbook*. New York: Guilford Press, 1995:56–60.
101. Stice E, Cameron RP, Killen JD, Hayward C, Taylor CB. Naturalistic weight-reduction efforts prospectively predict growth in relative weight and onset of obesity among female adolescents. *J Consult Clin Psychol* 1999;67(6):967–74.
102. Gutin B, Manos TM. Physical activity in the prevention of childhood obesity. *Ann NY Acad of Sci* 1993;699:115–126.
103. U.S. Department of Health and Human Services. *Physical activity and health: a report of the surgeon general*. Atlanta, Georgia: U.S. Department of Health and Human Services, Centers for Disease Control, National Center for Chronic Disease Prevention and Health Promotion, 1996.
104. U.S. Department of Health and Human Services. *Healthy People 2010*. Washington, DC: 2000.
105. Foreyt JP, Goodrick GK. *Living without dieting*. Houston, TX: Harrison, 1992.
106. Polivy J, Herman CP. Undieting: a program to help people stop dieting. *Int J Eating Disorders* 1992;11(3):261–8.
107. Polivy J, Herman CP. Diagnosis and treatment of normal eating. *J Consult Clin Psychol* 1987;55(5):635–44.
108. Wadden TA, Foster GD, Letizia KA. One-year behavioral treatment of obesity: comparison of moderate and severe caloric restriction and the effects of weight maintenance therapy. *J Consult Clin Psychol* 1994;62(1):165–71.
109. Yanovski SZ, Sebring NG. Recorded food intake of obese women with binge eating disorder before and after weight loss. *Int J Eating Disorders* 1994;15(2):135–50.