

# Psychological and Behavioral Risk Factors for Obesity Onset in Adolescent Girls: A Prospective Study

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Because little is known about risk factors for obesity, the authors tested whether certain psychological and behavioral variables predicted future onset of obesity. The authors used data from a prospective study of 496 adolescent girls who completed a baseline assessment at age 11–15 years and 4 annual follow-ups. Self-reported dietary restraint, radical weight-control behaviors, depressive symptoms, and perceived parental obesity—but not high-fat food consumption, binge eating, or exercise frequency—predicted obesity onset. Results provide support for certain etiologic theories of obesity, including the affect regulation model. The fact that self-reported, weight-control behaviors identified girls at risk for obesity implies that high-risk youths are not engaging in effective weight-control methods and suggests the need to promote more effective strategies.

Adolescent obesity is associated with serious medical problems, including high blood pressure, adverse lipoprotein profiles, diabetes mellitus, atherosclerotic cerebrovascular disease, coronary heart disease, colorectal cancer, and death from all causes, as well as lower educational attainment and higher rates of poverty (Dietz, 1998; Pietrobelli et al., 1998). Adolescent obesity also dramatically increases the risk of adult obesity (Whitaker, Wright, Pepe, Seidel, & Dietz, 1997), which is alarming because obesity is credited with 400,000 deaths annually in the United States (Mokdad, Marks, Stroup, & Gerberding, 2004). Moreover, the prevalence of adolescent obesity has doubled over the last three decades (Troiano, Flegal, Kuczmarski, Campbell, & Johnson, 1995).

The rapid increase in the prevalence of obesity suggests that psychological and behavioral factors, rather than biological factors, are primarily responsible for this trend (Wadden, Brownell, & Foster, 2002). Surprisingly, there have been relatively few prospective studies on the risk factors that predict onset of adolescent obesity. A risk factor is a variable that has been shown to prospectively predict onset of an adverse outcome among individuals who are initially free of the condition (Kraemer et al., 1997). Prospective data are necessary to differentiate the precursors from the consequences of obesity, test etiologic theories, isolate malleable risk factors for prevention programs, and identify high-risk subpopulations in need of targeted intervention. Thus, the goal of this study was to test whether theoretically derived psychological and behavioral risk factors predicted onset of obesity.

The energy balance model of adiposity stipulates that weight gain occurs if caloric intake exceeds energy expenditure (Rosenbaum, Leibel, & Hirsch, 1997). Thus, elevated caloric intake should predict obesity onset. Consumption of high-fat foods is thought to be a particularly powerful predictor of weight gain because of the efficiency with which fat is metabolized and its high caloric density and palatability (Golay & Bobbioni, 1997). Furthermore, because fat intake produces weak satiety signals relative to other macronutrients, it results in greater overall intake (Rolls, 1995). Self-reported caloric intake and high-fat food intake has predicted future increases in body mass in adults (French et al., 1994; Klesges, Isbell, & Klesges, 1992), although null findings have been observed (Klesges, Klem, & Bene, 1989). One study found that total energy intake, as assessed by the doubly labeled water method, predicted increases in body fat during childhood (Salbe, Weyer, Harper, et al., 2002). However, total caloric intake and fat intake did not predict change in body mass during childhood in another study (Maffeis, Talamini, & Tato, 1998). Because we were unable to locate any prospective research that tested whether high-fat food intake predicts obesity onset during adolescence, we investigated this relation in the present study.

Another form of high caloric intake—binge eating—is receiving attention as a potential risk factor for obesity (McGuire, Wing, Klem, Lang, & Hill, 1999; Stice, Cameron, Killen, Hayward, & Taylor, 1999). Binge eating might play a particularly insidious role in obesity promotion because it putatively leads to physiological changes that increase the likelihood that binge eating will persist over time. Research suggests that binge eating results in an enlarged stomach capacity (Geliebter et al., 1992), which could increase the likelihood of future binge eating through two mechanisms. First, because stomach distension activates gastric stretch receptors and mechanoreceptors that transmit satiety signals (Goldfein, Walsh, LaChaussee, Kissileff, & Devin, 1993), an individual with an enlarged stomach will consume more calories before the body initiates satiety signals to terminate eating. Second, an enlarged gastric capacity also appears to result in a slower rate of gastric emptying, which delays duodenal release of cholecystokinin and therefore also delays satiety signals (Gibbs, Young,

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This study was supported by National Institutes of Health Career Award MH01708 and Grant MH/DK61957. We thank the following project research assistants: Sarah Kate Bearman, Emily Burton, Melissa Fisher, Lisa Groesz, Natalie McKee, and Katy Whinton. We also thank our numerous undergraduate volunteers, the Austin Independent School District, and the participants who made this study possible.

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& Smith, 1973). Binge eating has predicted future weight gain (Stice et al., 1999) and onset of obesity (Stice, Presnell, & Spangler, 2002) in adolescent girls, although one study did not replicate this effect (Field et al., 2003). Binge eating also has predicted subsequent weight gain in adults (McGuire et al., 1999). Thus, we tested whether binge eating predicted obesity onset.

A second determinant of obesity that follows from the energy balance model is caloric expenditure. When the level of total caloric expenditure is lower than caloric intake, accumulation of adipose tissue ensues. Thus, sedentary activities and low levels of exercise should predict onset of obesity. Low levels of exercise and sedentary behavior have predicted future weight gain among adults (Ching et al., 1996; French et al., 1994), although null effects have occurred (Klesges et al., 1992). Low levels of exercise and high rates of sedentary behaviors also have predicted increases in body mass during adolescence (Berkey et al., 2000; Dietz & Grotmaker, 1985; Proctor et al., 2003), though these relations have not been consistently observed (Maffeis et al., 1998; Robinson et al., 1993; Salbe, Weyer, Harper, et al., 2002). Indeed, one study found that adolescents who reported exercising for weight-control purposes were at increased risk for future obesity onset (Stice et al., 1999). These findings might suggest that exercising for weight-control purposes is qualitatively different from other types of exercise, but these findings may also suggest that self-reported exercise is of questionable veracity. Because few studies have tested whether physical activity is a risk factor for onset of obesity during adolescence, we investigated this relation in the present study.

Paradoxically, adolescent girls with elevated scores on dieting scales are at increased risk for future onset of obesity (Stice et al., 1999) and weight gain (Field et al., 2003; Stice, 2001)—an effect that has also occurred in prospective studies of adults (French et al., 1994; Klesges et al., 1989, 1992). Radical weight-control behaviors, such as vomiting and laxative abuse, also have predicted increases in body mass over time (Stice et al., 1999). One interpretation of these findings is that dieting and radical weight-control methods may promote weight gain because these behaviors lead to increased metabolic efficiency (Klesges et al., 1992). Alternatively, individuals might overestimate the number of additional calories they can consume if they engage in these weight-control behaviors (i.e., people might overestimate the caloric expenditure afforded by these activities and may use these behaviors to justify overeating). Finally, self-reports of weight-control behaviors may simply identify people at high risk for onset of obesity (i.e., are proxy risk factors). It is possible that individuals with a chronic overeating tendency find themselves attempting to control their intake (e.g., dieting) but ultimately fail in these efforts and show obesity onset (Stice, 2002). It is important to test whether weight-control behaviors are risk factors for obesity onset in an independent sample and to extend these findings by comparing the predictive power of these weight-control behaviors with measures of caloric intake and expenditure.

In addition, researchers have posited that negative affect is a risk factor for obesity (Hoppa & Hallstrom, 1981). The affect regulation model posits that dysphoric individuals eat in an effort to provide comfort or distraction from negative emotions, which increases the risk for weight gain. The serotonin dysregulation that characterizes depression may also increase the risk for obesity, as individuals with disturbances in serotonin regulation may consume

excessive amounts of carbohydrate-rich foods in an effort to regulate their serotonin levels (Wurtman et al., 1985). Dietary or pharmacological serotonin administration leads to normalized eating and decreased depression (Ciarella, Ciarella, Graziani, & Mirante, 1991; Lieberman, Wurtman, & Chew, 1986; Wurtman et al., 1985). Furthermore, depression prospectively has predicted future increases in weight in adults (Hoppa & Hallstrom, 1981; McGuire et al., 1999) and future increases in body mass and onset of obesity in adolescents (Goodman & Whitaker, 2002; Pine, Goldstein, Wolk, & Weissman, 2001). Therefore, we tested whether depressive symptoms constituted a risk factor for obesity onset.

We also investigated parental obesity because we wanted to compare the predictive power of the psychological and behavioral factors with this widely recognized risk factor for obesity. Theoretically, children of obese parents are at greater risk of becoming obese because of both shared genetic factors and within-family environmental factors (Faith, Rha, Neale, & Allison, 1999). Parental obesity has generally been found to prospectively predict onset of offspring obesity in adulthood (Stettler et al., 2000; Whitaker et al., 1997). Parental body mass also has predicted future increases in offspring body mass during childhood (Salbe, Weyer, Lindsay, Ravussin, & Tataranni, 2002), but this effect was not replicated in one study of adolescents (Maffeis et al., 1998).

In sum, the aim of this study was to test whether behavioral and psychological risk factors predict onset of obesity during adolescence and to compare the predictive power of these factors with that of parental obesity. We attempted to improve on past research by using a multiwave prospective design with a long follow-up period, a large sample, direct measurements of height and weight, and multiple data collection methods (structured psychiatric interviews and self-report surveys). We focused on adolescent girls because data were drawn from a longitudinal study of the risk factors for eating pathology, which predominantly affects women. Nonetheless, we felt this was an important population to study because rates of obesity are greater for women than men and adolescence is a high-risk period for obesity onset (Wing, 1993).

## Method

### *Participants*

Participants were 496 adolescent girls from four public (82%) and four private (18%) middle schools in a metropolitan area of the southwestern United States. Adolescents ranged in age from 11 to 15 years ( $M = 13.50$ ,  $SD = 0.67$ ) at baseline (T1). Of the participants, 2% were Asian/Pacific Islander, 7% were African American, 68% were Caucasian, 18% were Hispanic, 1% were Native American, and 4% specified other or mixed racial heritage, which was representative of the ethnic composition of the schools from which we sampled (2% Asian/Pacific Islander, 8% African American, 65% Caucasian, 21% Hispanic, 4% other or mixed racial heritage). Average parental education, a proxy for socioeconomic status, was 29% high school graduate or less, 23% some college, 33% college graduate, and 15% graduate degree, which was representative of the metropolitan area from which we sampled (34% high school graduate or less, 25% some college, 26% college graduate, 15% graduate degree).

### *Procedures*

The study was described to parents and participants as an investigation of adolescent mental and physical health. An active parental consent

procedure was used to recruit participants, wherein an informed consent letter that described the study and a stamped self-addressed return envelope were sent to parents of eligible girls (a second mailing was sent to nonresponders). Adolescents provided assent immediately before data collection. This resulted in an average participation rate of 56%, which was comparable with rates in other school-recruited samples that used active consent procedures and structured interviews (e.g., 61% for Lewinsohn et al., 1994). Participants completed a survey, participated in a structured interview, and had their weight and height measured by female research assistants at T1 and at four follow-ups that occurred on a yearly basis (T2, T3, T4, T5). Female clinical assessors with at least a bachelor's degree in psychology conducted all interviews. Assessors attended 24 hr of training, wherein they were taught structured interview skills, reviewed diagnostic criteria for relevant *Diagnostic and Statistical Manual of Mental Disorders* (4th ed.; *DSM-IV*; American Psychiatric Association, 1994) disorders, observed simulated interviews, and role-played interviews. Assessors had to demonstrate an interrater agreement ( $\kappa > .80$ ) with experts using tape-recorded interviews before collecting data. Interviews were recorded periodically during the study to ensure that assessors continued to show acceptable interrater agreement ( $\kappa > .80$ ) with experts. Assessments took place at the school during or immediately after school hours or at participants' houses. Girls received a gift certificate to a local book and music store or a cash payment for compensation at each assessment.

## Measures

Participants completed all of the following measures at T1. Height and weight data were also collected at each of the four annual follow-up assessments.

**Fat consumption.** Fat intake was assessed with 18 items adapted from the Fat-Related Diet Habits Questionnaire (Kristal, Shattuck, & Patterson, 1999). Participants indicated how frequently they ate common high-fat foods during the past month using a 5-point scale ranging from 1 (*never or almost never*) to 5 (*5 or more times a week*; items were averaged). This adapted scale evidenced internal consistency ( $\alpha = .79$ ) and 1-week test-retest reliability ( $r = .90$ ) in a pilot study, and it also evidenced internal consistency ( $\alpha = .81$ ) and 1-year test-retest reliability ( $r = .57$ ) in the present study.

**Binge eating.** Frequency of binge eating was assessed with an adapted form of the Eating Disorder Examination (EDE; Fairburn & Cooper, 1993), a structured psychiatric interview assessing diagnostic criteria for *DSM-IV* anorexia nervosa and bulimia nervosa. Because binge eating frequency was skewed, a dichotomous variable was formed, wherein participants who reported two or more *DSM-IV* defined binge eating episodes in the past 3 months were coded 1, and all others were coded 0. The EDE has been found to have acceptable internal consistency ( $\alpha = .76-.90$ ), test-retest reliability ( $r = .83-.97$ ), interrater reliability ( $\kappa = .70-.99$ ), and to discriminate between individuals with eating disorders and controls (Rizvi, Peterson, Crow, & Agras, 2000; Williamson, Anderson, Jackman, & Jackson, 1995). To assess the interrater reliability in our study, a second clinical assessor who was blind to the first diagnosis reinterviewed a randomly selected subset of participants (5%) within a 3-day period, which resulted in high interrater agreement ( $\kappa = .88$ ). Another randomly selected subset of participants (5%) completed a second diagnostic interview 1 week later with the same clinical assessor, which resulted in high test-retest reliability ( $\kappa = 1.00$ ).

**Exercise.** The Past Year Leisure Physical Activity Scale was used to assess exercise level (Aaron et al., 1993). Participants indicate whether they engaged in 24 activities more than 10 times over the past year, then they report how many months out of the year, days per week, and minutes per day they engaged in each endorsed activity. Items were weighted according to metabolic expenditure (Wilson, Paffenbarger, Morris, & Havlik, 1986) and summed to create an overall measure of past year caloric expenditure through exercise. Because this variable was skewed, a square

root transformation was used to normalize the distribution. This scale has acceptable 1-year test-retest reliability ( $r = .55-.66$ ), correlates with behavioral fitness measures, and shows concordance with objective measures of exercise frequency (Aaron et al., 1993, 1995). This scale had adequate 1-year test-retest reliability in the present study ( $r = .56$ ).

**Dietary restraint.** The Dutch Restrained Eating Scale (van Strien, Frijters, van Staveren, Defares, & Deurenberg, 1986) was used to assess dietary restraint. Participants indicate the frequency of dieting behaviors using 5-point scales ranging from 1 (*never*) to 5 (*always*; items were averaged). This scale has acceptable internal consistency ( $\alpha = .95$ ) and test-retest reliability ( $r = .82$ ) and correlates negatively with self-reported caloric intake (Laessle, Tuschl, Kotthaus, & Pirke, 1989; Stice, 2001; van Strien et al., 1986). This scale had acceptable 1-year test-retest reliability ( $r = .62$ ) in the present study.

**Compensatory behaviors.** The EDE was also used to assess compensatory behaviors, including vomiting for weight-control purposes, laxative abuse, and diuretic abuse (Fairburn & Cooper, 1993). Because the frequency of compensatory behaviors was skewed, a dichotomous variable was formed, wherein participants who reported two or more episodes of *DSM-IV* compensatory behavior in the past 3 months were coded 1, and all other participants were coded 0. As noted previously, there is considerable evidence in support of the reliability and validity of the EDE (Rizvi et al., 2000; Williamson et al., 1995).

**Depressive symptoms.** An adapted version of the Schedule for Affective Disorders and Schizophrenia for School-Age Children (Puig-Antich & Chambers, 1983), a structured diagnostic interview, was used to assess *DSM-IV* symptoms of major depression. Severity ratings for each symptom were averaged. The Schedule for Affective Disorders and Schizophrenia for School-Age Children has been found to have acceptable test-retest reliability ( $\kappa = .63-1.00$ ), interrater reliability ( $\kappa = .73-1.00$ ), internal consistency ( $\alpha = .68-.84$ ), and to discriminate between depressed and nondepressed individuals (Ambrosini, 2000). To assess the interrater reliability in our study, a second assessor who was blind to the first diagnosis reinterviewed a randomly selected subset of participants (5%) within a 3-day period, which resulted in high interrater agreement ( $\kappa = 1.00$ ). Another randomly selected subset of participants (5%) completed a second diagnostic interview with the same assessor 1 week later, which resulted in high test-retest reliability ( $\kappa = 1.00$ ). This scale had acceptable internal consistency ( $\alpha = .85$ ) and 1-year test-retest reliability ( $r = .64$ ) in the present study.

**Perceived parental obesity.** We assessed perceived parental obesity with a dichotomous item created for this study ("Are either or both of your parents overweight?"). Responses were coded 1 = *yes* and 0 = *no*. Although this was a rudimentary measure of parental obesity, it showed acceptable temporal reliability over the 1-year period from T1 to T2 (test-retest  $r = .68$ ) and reasonable concordance (72% overall agreement),  $\chi^2(1, N = 432) = 47.89, p < .0001$ , with parental self-report of overweight at T5 (as assessed by a body mass index [BMI]  $> 25$ ).

**Body mass.** The BMI ( $\text{kg}/\text{m}^2$ ) was used as a proxy measure of adiposity. Height was measured to the nearest millimeter with a portable direct-reading stadiometer. Students were measured without shoes and with the body positioned such that the heels and buttocks were against the vertical support of the stadiometer and the head aligned so that the auditory canal and lower rim of the orbit were in a horizontal plane. We assessed weight to the nearest 0.1 kg using digital scales, with participants wearing light clothing without shoes or coats. At each assessment, two measures of height and weight were obtained and averaged. The BMI correlates with direct measures of total body fat—such as dual energy X-ray absorptiometry ( $r = .80-.90$ )—and with health measures, including blood pressure, adverse lipoprotein profiles, atherosclerotic lesions, serum insulin levels, and diabetes mellitus in adolescent samples (Dietz & Robinson, 1998; Pietrobelli et al., 1998). Because of the ease of assessment of the BMI, it has been recommended as the measure of choice for epidemiologic research (Dietz & Robinson, 1998). We defined adolescent obesity using the

age and sex adjusted 95th centiles of BMI, based on nationally representative data, as recommended by Barlow and Dietz (1998).

## Results

### Preliminary Analyses

Of the initial 496 participants, we did not collect follow-up data for 2% at T2, 2% at T3, 1% at T4, and 2% at T5, although less than 1% did not provide data at any follow-up assessment. Attrition analyses verified that girls who were missing data at any assessment point did not differ significantly from the remaining girls on demographic factors or any of the variables examined in the report, suggesting that attrition should not introduce bias.

### Descriptive Statistics

The correlations among the predictors are reported in Table 1, along with the means and standard deviations. Tests of independence, which were not based on the assumption of linear relations (e.g., chi-square tests for relations between dichotomous variables), were used to assess whether the dichotomous variables showed significant relations to dichotomous and continuous variables. In general, there were only weak relations between these variables. The relation between compensatory behaviors and dietary restraint was moderate, suggesting that individuals who endorse dietary behaviors also resort to other unhealthy compensatory behaviors for weight control. Use of compensatory behaviors also showed a moderate association with depressive symptoms. The fact that fat intake showed a very weak relation to binge eating suggests that these measures capture relatively orthogonal dimensions of intake. It is also noteworthy that perceived parental obesity showed weak relations with the behavioral and psychological predictors.

Adolescent age did not show significant relations to any of the variables examined in this report. There were no ethnic differences on the variables examined, with one exception: Caucasians were significantly less likely to report binge eating at T1 relative to ethnic minority participants (odds ratio [OR] = 0.26, 95% confidence interval [CI] = 0.10–0.68,  $p = .003$ ).

The mean fat intake score indicated that on average participants reported consuming each of the 18 high-fat foods 1–2 times in the past month. The median amount of exercise per week was 5.5 hr.

The mean dietary restraint score indicated that on average participants reported seldom use of the various dietary behaviors over the past 6 months (only 9% of the girls indicated that they were currently on a weight-loss diet). Within the past 3 months, 4% of participants reported at least two episodes of binge eating, and 14% reported at least two episodes of compensatory behaviors (e.g., vomiting). The mean depressive symptom score indicated that on average participants did not meet criteria for any of the *DSM-IV* symptoms of major depression (2.2% met *DSM-IV* criteria for current major depression at T1). Finally, 33% of the participants reported that one or both of their parents were overweight.

At T1, 42 (8.3%) of the 496 adolescents met age-adjusted criteria for obesity (2 participants did not provide BMI data at any follow-up). Although these initially obese participants were generally similar in age to the rest of the sample ( $M = 13.4$ , range = 12.0–15.3), African American and Hispanic participants were overrepresented, and Caucasian participants were underrepresented in this group (2% Asian-Pacific Islander, 16% African American, 47% Caucasian, 28% Hispanic, and 7% other or mixed racial heritage). Of the 453 nonobese participants at T1, 15 (3.3%) showed onset of obesity over the 4-year study period, as operationalized as exceeding the age-adjusted 95th centile at any of the four annual follow-up assessments. Obesity showed high temporal stability, in that an average of 84% of the participants who met the criteria for obesity at one assessment point met criteria at the following assessment point. As a frame of reference, the mean BMI was 21.1 at T1, 21.6 at T2, 22.0 at T3, 22.5 at T4, and 22.8 at T5. The rates of obesity and increases in BMI were generally consistent with estimates from our previous study on adolescent obesity (Stice et al., 1999) and nationally representative data (Kuczmarski et al., 2002).

### Logistic Regression Models

We estimated logistic regression models to test whether T1 fat intake, binge eating, exercise level, dietary restraint, compensatory behaviors, depressive symptoms, and perceived parental obesity increased the risk for onset of obesity over the 4-year follow-up period among adolescents who were not obese at T1. Participants who met criteria for obesity at T1 were excluded to ensure that we conducted a truly prospective test of our hypotheses.

Table 1  
Means, Standard Deviations, and Bivariate Correlations Among Time 1 Risk Factors

Factor	1	2	3	4	5	6	7	<i>M</i>	<i>SD</i>
1. High fat intake	—	.02	-.01	-.15*	-.13*	-.02	-.08	2.43	0.57
2. Binge eating		—	.02	.16*	.25*	.26*	.02	0.04	0.19
3. Exercise			—	.07	.13*	-.07	-.03	3.49	1.77
4. Dieting				—	.41*	.30*	.10*	2.22	0.92
5. Compensatory behaviors					—	.32*	.08	0.14	0.35
6. Depressive symptoms						—	.14*	1.34	0.37
7. Perceived parental obesity							—	0.33	0.47

Note. Pearson product-moment correlation coefficients were used to reflect relations between continuous variables; point-biserial correlation coefficients were used to reflect relations between continuous and dichotomous variables; tetrachoric correlation coefficients were used to reflect relations between dichotomous variables. Descriptive statistics are from the transformed version of the exercise variable used in the analyses.  
\*  $p < .05$ .

We first estimated univariate models to understand the effects of each potential risk factor without the complication of collinearity. Table 2 presents the ORs, 95% CIs, and *p* values for the univariate relations between the T1 predictors and the risk for obesity onset. As hypothesized, elevated dietary restraint,<sup>1</sup> use of compensatory behaviors, elevated depressive symptoms, and perceived parental obesity significantly increased the risk for obesity onset. Fat intake, binge eating, and exercise level did not predict obesity onset in these models. The ORs for the significant effects correspond to moderately large effect sizes.

We next estimated a multivariate model to investigate the unique effects of the risk factors that showed significant univariate effects, while we controlled for the effects of the other risk factors. Table 3 presents the ORs, 95% CIs, and *p* values for the multivariate relations between the T1 predictors and the risk for obesity onset over the study. The effects for dietary restraint and perceived parental obesity remained significant in the multivariate model. However, compensatory behaviors and depressive symptoms did not show significant unique effects in the multivariate model. The two significant effects showed moderate effect sizes.

### Discussion

In this study, our aim was to test whether psychological and behavioral risk factors predicted onset of obesity in adolescent girls. One of the most striking findings was that participants with elevated dietary restraint scores showed an increased risk for obesity onset, which replicates the results from previous studies with adolescents (Stice et al., 1999) and adults (French et al., 1994; Klesges et al., 1992). The use of maladaptive compensatory behaviors for weight control—such as vomiting or laxative abuse—likewise increased the risk for obesity onset, which replicates the effects from an earlier prospective study (Stice et al., 1999). The fact that these effects have been observed in several independent studies implies that these effects are moderately robust. The ORs suggested that there was more than a threefold increase in risk for obesity onset with every unit increase on the dieting scale and that recurrent use of compensatory behaviors was associated with more than a fivefold increase in the risk for obesity onset. Thus, the magnitude of these ORs implies that both of these effects are clinically meaningful.

One interpretation of these findings is that weight-control behaviors may promote weight gain because they lead to increased

Table 3

*Multivariate Relations Between Time 1 Risk Factors and Future Risk for Obesity Onset During the 4-Year Follow-Up Period*

Time 1 risk factors	OR	95% CI	<i>p</i>
Dietary restraint	3.16	1.58–6.31	.001***
Compensatory behaviors	1.35	0.40–4.60	.632
Depressive symptoms	2.32	0.62–8.65	.210
Perceived parental obesity	3.97	1.24–12.72	.020*

*Note.* Logistic regression models excluded initially obese participants. OR = odds ratio; CI = confidence interval.

\* *p* < .05. \*\*\* *p* < .001.

metabolic efficiency or other alterations in homeostatic processes (Klesges et al., 1992). Blundell (1995) argued that dieting results in an erratic delivery of nutrients with aberrant triggering of physiological responses, which leads to a desynchronization between behavior and physiology. This dysregulation of the normal appetite system is thought to promote weight gain because biological regulatory processes oppose undereating but not overeating. Alternatively, weight-control behaviors may increase the risk for binge eating, which in turn results in weight gain, although this interpretation does not accord with the finding that binge eating did not predict obesity onset in the present study. These findings might also be interpreted as providing additional evidence that self-report, weight-control behaviors are not valid. Studies have failed to find the expected inverse correlations between dietary restraint scales and unobtrusive observational and biological measures of acute and long-term caloric intake (Bathalon et al., 2000; Stice, Fisher, & Lowe, 2004). Finally, scales measuring behaviors aimed at managing weight may simply identify people with overeating problems who are at elevated risk for obesity (Stice et al., 1999). That is, an overeating tendency may increase the likelihood that a person will engage in weight-control strategies and may ultimately result in obesity onset despite the weight-control efforts. This interpretation would suggest that self-reported, weight-control behaviors are simply a proxy measure of a tendency toward overconsumption.

We also found support for the hypothesis that depressive symptoms would predict obesity onset. This finding converges with results from other prospective studies with adolescents and adults (Goodman & Whitaker, 2002; McGuire et al., 1999; Pine et al., 2001), which implies that this effect is relatively robust. The OR suggested that for each additional depressive symptom reported by the adolescent, there was more than a fourfold increase in risk for obesity onset, which suggests that this effect is clinically meaningful in magnitude. Thus, results provide support for the affect regulation etiologic model of obesity. Theoretically, depressed individuals eat to provide comfort or distraction from negative emotions. It is also possible that the serotonin dysregulation that characterizes depression leads individuals to consume excessive

Table 2

*Univariate Relations Between Time 1 Risk Factors and Future Risk for Obesity Onset During the 4-Year Follow-Up Period*

Time 1 risk factor	OR	95% CI	<i>p</i>
High-fat diet	0.50	0.19–1.32	.162
Binge eating	2.34	0.29–19.11	.429
Exercise	1.21	0.96–1.53	.111
Dietary restraint	3.39	1.92–5.99	.001***
Compensatory behaviors	5.17	1.77–15.13	.003**
Depressive symptoms	4.62	1.67–12.74	.004**
Perceived parental obesity	4.58	1.53–13.64	.006**

*Note.* Logistic regression models excluded initially obese participants. OR = odds ratio; CI = confidence interval.

\*\* *p* < .01. \*\*\* *p* < .001.

<sup>1</sup> Post hoc analyses revealed that girls who indicated that they were currently on a weight-loss diet at T1 were significantly more likely to show onset of obesity than girls who indicated that they were not on a weight-loss diet (OR = 6.07, 95% CI = 1.96–18.81, *p* = .002). We thought it would be useful to report this effect because some investigators have used this type of current dieting item.

amounts of carbohydrate-rich foods in an effort to regulate serotonin levels. An interesting finding was the fact that the effect for depressive symptoms was only significant in the univariate model, which implies that this effect may be accounted for by the collinearity it shows with one or more of the risk factors that showed significant univariate and multivariate effects. The correlations between these risk factors suggest that individuals who engage in dieting and weight-control behaviors often experience elevated depression.

Perceived parental obesity also predicted onset of obesity. This finding also appears to be robust in that it converges with past evidence that parental obesity has predicted obesity onset during childhood and adulthood (Salbe, Weyer, Lindsay, et al., 2002; Stettler et al., 2000; Whitaker et al., 1997). The OR indicated that adolescents who reported parental obesity were at more than a fourfold increase for obesity onset than their peers not reporting this risk factor. However, this effect should be interpreted with caution because we did not define obesity in our single-item measure of parental obesity, which made this measure somewhat subjective. In addition, because we did not ask participants to report on their biological parents, it is not possible to draw conclusions regarding the degree to which this predictive effect may reflect genetic versus environmental contributions to obesity. Fortunately, this single-item measure did show temporal reliability and convergent validity with parental report of overweight, and the present findings suggest that this measure also possessed predictive validity.

Contrary to hypotheses, elevated intake of high-fat foods, binge eating, and exercise did not predict obesity onset. Although these findings appear to be inconsistent with the energy balance model of obesity, two of the five prospective studies that tested whether caloric intake and intake of high-fat foods predicted increases in body mass with data from adults or children found null effects (Klesges et al., 1989; Maffei et al., 1998), and one of the two studies that tested whether binge eating predicted obesity onset in adolescent girls found a null effect (Field et al., 2003). Likewise, five of the ten prospective studies that we located did not generate the expected significant inverse correlation between exercise and future weight gain or onset of obesity (Klesges et al., 1992; Maffei et al., 1998; Robinson et al., 1993; Salbe, Weyer, Harper, et al., 2002; Stice et al., 1999). The fact that the one study that used an objective biological measure of caloric intake found a prospective effect, despite a small sample size, suggests that the documented underreporting of caloric intake (Lichtman et al., 1992) might make it difficult to consistently observe prospective effects between caloric intake and obesity onset. Similar reporting biases might make it difficult to elicit reliable reports of exercise behavior, which serves to attenuate this effect. Alternatively, it may be that eating and exercise behaviors fluctuate widely over time, which makes it difficult to observe a prospective effect over a long follow-up period. Consistent with this suggestion, the 1-year test-retest reliability coefficients were lower for caloric intake and exercise (mean  $r = .57$ ) than for the other risk factors examined in this report (mean  $r = .65$ ).

### *Strengths and Limitations*

The confidence that can be placed in our findings is bolstered by the fact that we collected direct measures of height and weight.

Moreover, because predictors were assessed via surveys or structured psychiatric interviews, whereas research assistants measured height and weight, it is unlikely that shared method or reporter bias explain our findings. The fact that risk factors assessed at baseline were used to predict future risk for onset of obesity over the study permits stronger inferences about the direction of effects than is possible with cross-sectional data. We also had a large sample and a long follow-up period.

Despite these strengths, there were several limitations that should be considered when interpreting the findings. First, it is possible that some of the null findings occurred because certain self-report measures possessed limited validity—particularly the self-report measures that assessed caloric intake and exercise behaviors. Second, although we observed effects for adolescent report of parental obesity, these findings should be interpreted with care because we did not define obesity in our question or specifically ask adolescents to report on biological parents. Third, whereas the BMI is the recommended measure of adiposity in epidemiologic research (Dietz & Robinson, 1998), it can reflect elevations in other aspects of body composition rather than adipose tissue, such as muscle mass. Fourth, our findings should be generalized with caution to individuals who are Asian/Pacific Islander, African American, and Native American because our sample only contained small proportions of individuals from these ethnic groups. Also, because the sample consisted exclusively of female adolescents, results may not generalize to male adolescents. Finally, as is the case with all longitudinal research, it is possible that some unmeasured variable explains the observed prospective effects.

### *Clinical Implications and Directions for Future Research*

The present results have several clinical implications. First, the evidence that adolescent girls who report dieting and radical weight-control techniques are at increased risk for obesity onset suggests that obesity prevention efforts should target this high-risk population. These results also imply that it is particularly important to educate youths about more effective weight-control strategies, as randomized trials have clearly found that a reduction in caloric overconsumption and an increase in exercise result in decreased risk for obesity onset (Klem, Wing, Simkin-Silverman, & Kuller, 1997; Stice, Presnell, Groesz, & Shaw, in press). Second, the evidence that adolescent girls with depression are at higher risk for obesity onset implies that prevention and treatment interventions that effectively reduce depressive symptoms might also successfully reduce the risk for onset of obesity. Third, our results also suggest that children of obese parents are at elevated risk for obesity onset, which implies that it might be advantageous to target this high-risk population with obesity prevention programs.

Results suggest that future research should investigate the hypothesis that weight-control behaviors paradoxically increase risk for obesity onset. It will be important for future studies to use objective and valid measures of weight-control behaviors to ensure that the countermending findings are not the product of social-desirability biases. Research should also begin to test multivariate etiologic models that specify how the various risk factors might work together in a mediational or moderational fashion to predict onset of obesity. For example, it would be useful to test whether increases in binge eating or metabolic efficiency mediate the

relations between weight-control behaviors and onset of obesity. Finally, it will be vital to conduct randomized prevention trials that test whether reducing these weight-control behaviors actually results in reduced risk for obesity onset. These types of experiments would effectively rule out the possibility that some unmeasured variable is responsible for the prospective effects observed in longitudinal studies. An improved understanding of the etiologic processes that give rise to obesity and the groups that are at greatest risk for obesity should ultimately facilitate the design of effective prevention programs for this public health epidemic.

## References

- Aaron, D. J., Kriska, A. M., Dearwater, S. R., Anderson, R. L., Olsen, T. L., Cauley, J. A., & LaPorte, R. E. (1993). The epidemiology of leisure physical activity in an adolescent population. *Medicine and Science in Sports and Exercise*, *25*, 847–853.
- Aaron, D. J., Kriska, A. M., Dearwater, S. R., Cauley, J. A., Metz, K. F., & LaPorte, R. E. (1995). Reproducibility and validity of an epidemiologic questionnaire to assess past year physical activity in adolescents. *American Journal of Epidemiology*, *142*, 191–201.
- Ambrosini, P. J. (2000). Historical development and present status of the schedule for affective disorders and schizophrenia for school-age children (K-SADS). *Journal of the American Academy of Child & Adolescent Psychiatry*, *39*, 49–58.
- American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author.
- Barlow, S. E., & Dietz, W. H. (1998). Obesity evaluation and treatment: Expert committee recommendations. The Maternal and Child Health Bureau, Health Resources and Services Administration, and the Department of Health and Human Services. *Pediatrics*, *102*, E29.
- Bathalon, G., Tucker, K., Hays, N., Vinken, A., Greenberg, A., McCrory, M., et al. (2000). Psychological measures of eating behavior and the accuracy of three common dietary assessment methods in healthy postmenopausal women. *American Journal of Clinical Nutrition*, *71*, 739–745.
- Berkey, C. S., Rockett, H. R., Field, A. E., Gillman, M. W., Frazier, A. L., Camargo, C. A., & Colditz, G. A. (2000). Activity, dietary intake, and weight changes in a longitudinal study of preadolescent and adolescent boys and girls. *Pediatrics*, *105*, E56.
- Blundell, J. E. (1995). The psychobiological approach to appetite and weight control. In K. D. Brownell & C. G. Fairburn (Eds.), *Eating disorders and obesity: A comprehensive handbook* (pp. 13–20). New York: Guilford Press.
- Ching, P. L., Willet, W. C., Rimm, E. B., Colditz, G. A., Gortmaker, S. L., & Stampfer, M. J. (1996). Activity level and risk of overweight in male health professionals. *American Journal of Public Health*, *86*, 25–30.
- Ciarella, G., Ciarella, M., Graziani, P., & Mirante, M. (1991). Changes in food consumption of obese patients induced by dietary treatment combined with dexfenfluramine. *International Journal of Obesity*, *15*, 69.
- Dietz, W. H. (1998). Childhood weight affects adult morbidity and mortality. *Journal of Nutrition*, *128*, 411S–414S.
- Dietz, W. H., & Gortmaker, S. L. (1985). Do we fatten our children at the television? *Pediatrics*, *75*, 807–812.
- Dietz, W. H., & Robinson, T. N. (1998). Use of body mass index (BMI) as a measure of overweight in children and adolescents. *Journal of Pediatrics*, *132*, 191–193.
- Fairburn, C., & Cooper, Z. (1993). The eating disorder examination. In C. Fairburn & G. Wilson (Eds.), *Binge eating: Nature, assessment, and treatment* (12th ed., pp. 317–360). New York: Guilford Press.
- Faith, M. S., Rha, S. S., Neale, M. C., & Allison, D. B. (1999). Evidence for genetic influences on human energy intake: Results from a twin study using measured observations. *Behavior Genetics*, *29*, 145–154.
- Field, A. E., Austin, S. B., Taylor, C. B., Malspeis, S., Rosner, B., Rockett, H. R., et al. (2003). Relation between dieting and weight change among preadolescents and adolescents. *Pediatrics*, *112*, 900–906.
- French, S. A., Jeffery, R. W., Forster, J. L., McGovern, P. G., Kelder, S. H., & Baxter, J. E. (1994). Predictors of weight change over two years among a population of working adults: The Healthy Worker Project. *International Journal of Obesity*, *18*, 145–154.
- Geliebter, A., Melton, P. M., McCray, R. S., Gallagher, D. R., Gage, D., & Hashim, S. A. (1992). Gastric capacity, gastric emptying, and test-meal intake in normal and bulimic women. *American Journal of Clinical Nutrition*, *56*, 656–661.
- Gibbs, J., Young, R. C., & Smith, G. P. (1973). Cholecystokinin decreases food intake in rats. *Journal of Comparative Physiological Psychology*, *84*, 488–495.
- Golay, A., & Bobbioni, E. (1997). The role of dietary fat in obesity. *International Journal of Obesity*, *21*, 2–11.
- Goldfein, J. A., Walsh, B. T., LaChaussee, J. L., Kissileff, H. R., & Devin, M. J. (1993). Eating behavior in binge eating disorder. *International Journal of Eating Disorders*, *14*, 427–431.
- Goodman, E., & Whitaker, R. C. (2002). A prospective study of the role of depression in the development and persistence of adolescent obesity. *Pediatrics*, *109*, 497–504.
- Hoppa, H., & Hallstrom, T. (1981). Weight gain in adulthood in relation to socioeconomic factors, mental illness, and personality traits: A prospective study of middle-aged women. *Journal of Psychosomatic Research*, *25*, 83–89.
- Klem, M. L., Wing, R. R., Simkin-Silverman, L., & Kuller, L. H. (1997). The psychological consequences of weight gain prevention in healthy, premenopausal women. *International Journal of Eating Disorders*, *21*, 167–174.
- Klesges, R. C., Isbell, T. R., & Klesges, L. M. (1992). Relationship between restraint, energy intake, physical activity, and body weight: A prospective analysis. *Journal of Abnormal Psychology*, *101*, 668–674.
- Klesges, R. C., Klem, M., & Bene, C. (1989). Effects of dietary restraint, obesity, and gender on holiday eating behavior and weight gain. *Journal of Abnormal Psychology*, *98*, 499–503.
- Kraemer, H. C., Kazdin, A. E., Offord, D. R., Kessler, R. C., Jensen, P. S., & Kupfer, D. J. (1997). Coming to terms with the terms of risk. *Archives of General Psychiatry*, *54*, 337–343.
- Kristal, A. R., Shattuck, A. L., & Patterson, R. E. (1999). Differences in fat-related dietary patterns between Black, Hispanic, and White women: Results from the Women's Health Trial Feasibility Study in Minority Populations. *Public Health Nutrition*, *2*, 253–262.
- Kuczumski, R. J., Ogden, C. L., Guo, S. S., Grummer-Strawn, L. M., Flegal, K. M., Mei, Z., et al. (2002). 2000 CDC growth charts for the United States: Methods and development. *Vital and Health Statistics*, *11*, 1–190.
- Laessle, R. G., Tuschl, R. J., Kotthaus, B. C., & Pirke, K. (1989). A comparison of the validity of three scales for the assessment of dietary restraint. *Journal of Abnormal Psychology*, *98*, 504–507.
- Lewinsohn, P., Roberts, R., Seeley, J., Rohde, P., Gotlib, I., & Hops, H. (1994). Adolescent psychopathology: II. Psychosocial risk factors for depression. *Journal of Abnormal Psychology*, *103*, 302–315.
- Lichtman, S. W., Pisarska, K., Berman, E. R., Pestone, M., Dowling, H., Offenbacher, E., et al. (1992). Discrepancy between self-reported and actual caloric intake and exercise in obese subjects. *New England Journal of Medicine*, *327*, 1893–1898.
- Lieberman, H., Wurtman, J., & Chew, B. (1986). Changes in mood after carbohydrate consumption among obese individuals. *American Journal of Clinical Nutrition*, *45*, 772–778.
- Maffei, C., Talamini, G., & Tato, L. (1998). Influence of diet, physical activity, and parents' obesity on children's adiposity: A four-year longitudinal study. *International Journal of Obesity*, *22*, 758–764.
- McGuire, M., Wing, R. R., Klem, M., Lang, W., & Hill, J. (1999). What

- predicts weight regain in a group of successful weight losers? *Journal of Consulting and Clinical Psychology*, 67, 177–185.
- Mokdad, A. H., Marks, J. S., Stroup, D. F., & Gerberding, J. L. (2004). Actual causes of death in the United States, 2000. *Journal of the American Medical Academy*, 291, 1238–1245.
- Pietrobelli, A., Faith, M., Allison, D., Gallagher, D., Chiumello, G., & Heymsfield, S. (1998). Body mass index as a measure of adiposity among children and adolescents: A validation study. *Journal of Pediatrics*, 132, 204–210.
- Pine, D. S., Goldstein, R. B., Wolk, S., & Weissman, M. M. (2001). The association between childhood depression and adulthood body mass index. *Pediatrics*, 107, 1049–1056.
- Proctor, M., Moore, L., Gao, D., Cupples, L., Bradlee, M., Hood, M., & Ellison, R. (2003). TV viewing and change in body fat from preschool to early adolescence: The Framingham Children's Study. *International Journal of Obesity and Related Metabolic Disorders*, 27, 827–833.
- Puig-Antich, J., & Chambers, W. J. (1983). *Schedule for Affective Disorders and Schizophrenia for School-Age Children (6–18 years)*. Pittsburgh, PA: Western Psychiatric Institute.
- Rizvi, S. L., Peterson, C. B., Crow, S. J., & Agras, W. S. (2000). Test–retest reliability of the eating disorder examination. *International Journal of Eating Disorders*, 28, 311–316.
- Robinson, T. N., Hammer, L. D., Killen, J. D., Kraemer, H. C., Wilson, D. M., Hayward, C., & Taylor, C. B. (1993). Does television viewing increase obesity and reduce physical activity? Cross-sectional and longitudinal analyses among adolescent girls. *Pediatrics*, 91, 273–280.
- Rolls, B. J. (1995). Carbohydrates, fats, and satiety. *American Journal of Clinical Nutrition*, 61, 960–967.
- Rosenbaum, M., Leibel, R., & Hirsch, J. (1997). Obesity. *New England Journal of Medicine*, 337, 396–407.
- Salbe, A. D., Weyer, C., Harper, I., Lindsay, R. S., Ravussin, E., & Tataranni, P. A. (2002). Assessing risk factors for obesity between childhood and adolescence: II. Energy metabolism and physical activity. *Pediatrics*, 110, 307–314.
- Salbe, A. D., Weyer, C., Lindsay, R. S., Ravussin, E., & Tataranni, P. A. (2002). Assessing risk factors for obesity between childhood and adolescence: I. Birth weight, childhood adiposity, parental obesity, insulin, and leptin. *Pediatrics*, 110, 299–306.
- Stettler, N., Tershakovec, A. M., Zemel, B. S., Leonard, M. B., Boston, R. C., Katz, S. H., & Stallings, V. A. (2000). Early risk factors for increased adiposity: A cohort study of African American subjects followed from birth to young adulthood. *American Journal of Clinical Nutrition*, 53, 1543S–1551S.
- Stice, E. (2001). A prospective test of the dual pathway model of bulimic pathology: Mediating effects of dieting and negative affect. *Journal of Abnormal Psychology*, 110, 124–135.
- Stice, E. (2002). Risk and maintenance factors for eating pathology: A meta-analytic review. *Psychological Bulletin*, 128, 825–848.
- Stice, E., Cameron, R., Killen, J. D., Hayward, C., & Taylor, C. B. (1999). Naturalistic weight reduction efforts prospectively predict growth in relative weight and onset of obesity among female adolescents. *Journal of Consulting and Clinical Psychology*, 67, 967–974.
- Stice, E., Fisher, M., & Lowe, M. (2004). Are dietary restraint scales valid measures of acute dietary restriction? Unobtrusive observational data suggest not. *Psychological Assessment*, 16, 51–59.
- Stice, E., Presnell, K., Groesz, L., & Shaw, H. (in press). Effects of a weight maintenance diet on bulimic pathology: An experimental test of the dietary restraint theory. *Health Psychology*.
- Stice, E., Presnell, K., & Spangler, D. (2002). Risk factors for binge eating onset: A prospective investigation. *Health Psychology*, 21, 131–138.
- Troiano, R. P., Flegal, K. M., Kuczmarski, R. J., Campbell, S. M., & Johnson, C. L. (1995). Overweight prevalence and trends for children and adolescents. *Archives of Pediatric Adolescent Medicine*, 149, 1085–1091.
- van Strien, T., Frijters, J., van Staveren, W., Defares, P., & Deurenberg, P. (1986). Predictive validity of the Dutch Restrained Eating Scale. *International Journal of Eating Disorders*, 5, 747–755.
- Wadden, T. A., Brownell, K. D., & Foster, G. D. (2002). Obesity: Responding to the global epidemic. *Journal of Consulting and Clinical Psychology*, 70, 510–525.
- Whitaker, R. C., Wright, J. A., Pepe, M. S., Seidel, K. D., & Dietz, W. H. (1997). Predicting obesity in young adulthood from childhood and parental obesity. *New England Journal of Medicine*, 337, 869–873.
- Williamson, D. A., Anderson, D. A., Jackman, L. P., & Jackson, S. R. (1995). Assessment of eating disordered thoughts, feelings, and behaviors. In D. B. Allison (Ed.), *Handbook of assessment methods for eating behaviors and weight-related problems: Measures, theory, and research* (pp. 347–386). Thousand Oaks, CA: Sage.
- Wilson, P. W., Paffenbarger, R. S., Morris, J. N., & Havlik, R. (1986). Assessment methods for physical activity and physical fitness in population studies: Report of a NHLBI workshop. *American Heart Journal*, 111, 1177–1192.
- Wing, R. R. (1993). Obesity and related eating and exercise behaviors in women. *Annals of Behavioral Medicine*, 15, 124–134.
- Wurtman, J., Wurtman, R., Mark, S., Tsay, R., Gilbert, W., & Growdon, J. (1985). Dextro-fenflamine selectively suppresses carbohydrate snacking by obese subjects. *International Journal of Eating Disorders*, 4, 89–99.

Received January 20, 2004

Revision received May 27, 2004

Accepted June 3, 2004 ■