A twin study of the relation between age at dieting onset and to adult BMI and dieting behaviors

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Abstract

**Background:** The association between dieting behaviors and weight gain has been well documented in numerous studies. However, there is an ongoing debate whether individuals who are predisposed to a greater BMI are less likely to successfully diet, or if dieting behaviors result in a greater BMI. Moreover, an early age at dieting onset may be a particular risk factor for higher adult BMI and risky dieting behaviors.

**Methods:** Female twins ages 18-60 years (N=950) from the University of Washington Twin Registry completed a survey in 2006. A subsequent follow-up survey was completed approximately 3-years later. Overall and within-twin pair analyses, which included all twin participants and statistically accounted for twin’s relatedness, were conducted in order to quantify associations between the age at dieting onset and 1) BMI at time of baseline assessment, 2) change in BMI between two surveys, and 3) risky eating behaviors.

**Results:** Overall, in adjusted models significant associations were found between an earlier age at dieting onset and BMI at baseline (p=.003), and with use of dieting methods such as restrained eating (p<0.001), risky dieting behaviors (p=0.016), and weight cycling (p=0.011). However, in within-pair models that adjusted for shared genetic and family environment factors, there were no significant associations between age at dieting onset and any of the above measures.

**Conclusion:** Among adult female twins, an earlier age at dieting onset was associated with an increased BMI, and greater use of dieting behaviors including restraint, risky dieting methods, and weight cycling in overall, cross-sectional analyses. However, when genetic and family environment factors were accounted for using within-twin pair analyses, the above associations were no longer significant. Thus, genetics and the family environment may influence both the age at dieting onset and BMI, as well as the adoption of risky eating behaviors.
Introduction

Dieting

As our society struggles with the prevailing obesity epidemic, many Americans have attempted dieting as a means to control and hopefully lose excess weight. The most common method for weight loss in overweight and obese individuals is dieting; unfortunately it has not been shown to be successful for long-term weight loss maintenance (MacLean et al., 2011). The average age at dieting onset was 14 in the 1970’s. However, the average age of dieting dropped dramatically to age 8 by 1990 (Eating Disorder Foundation 2005).

There is a large gender discrepancy in dieting as evidenced by a much greater prevalence of dieting in females as compared to males (Field et al., 2007). Estimates from the most recent Youth Risk Behavior Surveillance System (YRBSS) survey found that the prevalence of dieting was 61.2% among female high-school students as compared to 31.6% in male high-school students (CDC 2012). Bulik and colleagues conducted a national survey detailing eating and diet behaviors of 25-45 year-old women and found that 67% of women are trying to lose weight, 53% of dieters are a normal weight but continue to diet, and 75% of women report disordered eating behaviors (Reba-Harrelson and Bulik et al., 2009).

The prevalence of dieting in women rises with increasing BMI; 24% of women with a normal BMI reported dieting, 49% with an overweight BMI, and 58% with an obese BMI (Kruger et al., 2004). Although a greater prevalence of dieting is
evidenced in heavier individuals, almost a ¼ of normal-weight women are on a diet. Moreover, it has been documented that women who diet are more likely to have lower body satisfaction independent of their weight status (Spear 2006).

Dieting is largely ineffective in the long-term for significant weight loss as evidenced by the growing number of women who diet and the unrelenting rise in obesity nationwide. In fact, most dieters are successful only in the short-term and tend to gain back what they lost, if not more weight, in the long run (Safer 1991). This is known as weight cycling, which can result in an accumulation of abdominal fat associated with multiple attempts at weight loss followed by weight regain (Cereda 2011). Weiss and colleagues demonstrated that overweight and obese individuals who had experienced substantial weight loss (>10% of body weight) from their maximum body weight were largely unsuccessful maintaining their weight loss 1 year later, as evidenced by weight regain in 33.5% of subjects (Weiss et al., 2007).

*The Relationship Between Dieting and BMI*

It has been well established that dieting is associated with greater weight gain over time and a greater BMI. Female dieters gained more weight over time compared to female nondieters (Fields et al., 2007). A 6-year follow-up study conducted by Savage and colleagues observed women who reported dieting at the beginning of the study were heavier and gained more weight throughout the duration as compared to nondieters (Savage, Hoffman and Birch 2009).
The exact mechanism by which dieting influences weight gain is still unknown. However, multiple possibilities have been proposed for the relationship between dieting and increased BMI, which include the adoption of specific unhealthy dietary habits, physiological changes to metabolism, and genetic influences. Findings from the Project EAT study indicate that the adoption of unhealthy lifestyle behaviors is common in adolescent dieters, including decreased breakfast consumption, lower physical activity levels and fruit and vegetable intake, as well as increased binge eating (Neumark-Sztainer et al. 2006). Therefore, individuals who diet may be more prone to weight gain by engaging in these unhealthy behaviors long-term. Additionally, the act of dieting has been related to a compensatory decrease in an individual's metabolic rate, driving weight re-gain (Leibel, 1995). Lastly, those who diet may be genetically predisposed to obesity and more likely to diet due to genetic and familial influences. It is unknown if heavier people are more likely to diet and remain heavier, or if dieting itself makes one gain weight and become heavier.

Few longitudinal studies have observed the long-term effects of dieting at an earlier age. Some have found that weight control behaviors used in adolescence and preadolescence are correlated with a higher BMI later in life (Neumark-Sztainer et al., 2011; Viner 2007; Field 2003; Neumark-Sztainer et al., 2006; Larson 2007). Results from the National Longitudinal Study of Adolescent Health found that female adolescents who reported dieting had significantly larger gains in BMI over 5-years as compared to nondieters (Field 2007). Dieting adolescents are not only
predisposed to greater weight gain and obesity in adulthood, but also to disordered eating behaviors that often result in clinical eating disorders (Neumark-Sztainer et al. 2006).

*Genetic and Familial Influences on Eating Behaviors and BMI*

Various studies indicate that there is a considerable amount of heritability and genetic predisposition for BMI. One study in particular analyzed the genetic and environmental influences on BMI from adolescence into adulthood in twins (Ortega-Alonso 2012). Heritability accounted for 80% of BMI growth during adolescence, but later decreased to 70% as the twins progressed into young adulthood (age 24) (Ortega-Alonso, 2012). The rate-of-change in BMI over the course of adolescence was not associated with initial BMI, indicating that specific genes may influence the maintenance of BMI whereas others are responsible for the rate-of-change in BMI (Ortega-Alonso 2012). These genetic components not only interact with each other but also are most likely dependent on numerous environmental factors such as physical activity, diet, and body stressors (Ortega-Alonso 2009).

While genetic influences are strong, other familial factors such as eating habits likely also contribute to obesity. This is especially pertinent because the dietary behaviors learned as a child generally continue into adulthood (Scaglioni et al., 2008). In particular, the parent’s behavior, especially that of the mother, can have a strong influence on a child’s eating behavior including specific eating styles,
food selection and preference, and the self-regulation of food and energy intake (Scaglioni et al., 2008).

Parental encouragement for their child to diet, especially on the part of mothers, was predictive of increased adolescent unhealthy weight control behaviors and added weight gain (Neumark-Sztainer et al., 2008; 2010). A follow-up study from Project EAT discovered that the girls who reported greater maternal eating and weight-related concerns and behaviors at baseline were associated with a greater prevalence of dieting at the 5-year follow-up (Van den Berg et al., 2010). Additionally, adolescent females who perceived a thinner weight was important to their mother were frequent dieters compared to those who perceived their weight to be unimportant to their mother (Field, et al., 2005).

*Dieting in Twins*

There remains an ongoing debate concerning the relationship between dieting and weight gain. Researchers have hypothesized that individuals may be predisposed to a greater weight and more likely to engage in dieting behaviors that are unsuccessful. Therefore, individuals predisposed to obesity may diet more often in order to control for their affinity towards weight gain (Pietilainen et al., 2012). On the other hand, dieting may actually induce weight gain. A recent twin study found that the twin with the highest BMI at baseline also had the most instances of intentional weight loss and highest amount of weight gain, compared to his or her monozygotic co-twin (Pietilainen et al., 2012). A different analysis using
monozygotic twins discordant for obesity or overweight found that the heavier twins took part in unhealthy eating behaviors such as restrictive eating, overeating, and had fewer healthy food choices despite a shared genetic background (Keski-Rahkonen et al., 2007).

It is still unclear whether individuals diet as a means to counter their genetic tendency towards a greater weight or if dieting behaviors arise from environmental factors, which ultimately leads to subsequent weight gain. Twin studies provide an opportunity to examine these questions.

Unhealthy Weight-Control Behaviors and BMI

Dieting can be composed of healthy or risky behaviors. Healthy dieting behaviors are associated with lifestyle changes such as drinking water instead of soda, decreasing sweets, or incorporating exercise into a normal routine. These types of behaviors may lead to improvements in overall health and the maintenance of a healthy weight (Larson, Nicole et al. 2009). On the other hand, risky dieting behaviors such as fasting, use of diet pills, vomiting, laxatives, and excessive exercising are associated with less healthful dietary patterns and a greater tendency to remain or become overweight (Neumark-Sztainer et al., 2011; Larson et al., 2009). Adolescents who reported using risky dieting behaviors such as meal skipping and diet pills were at 3 times greater risk of becoming overweight over 5-years in one longitudinal study (Neumark-Sztainer et al., 2006).
Adolescents who engage in unhealthy weight control behaviors have greater BMI values to start with and change in BMI over 10-years (Neumark-Sztainer et al., 2011). In this study, the persistent use of unhealthy weight control behaviors was predictive of larger weight gain compared to those who did not use any unhealthy weight control behaviors (Neumark-Sztainer et al., 2011).

Although unhealthy weight control behaviors are known to be dangerous and largely ineffective, 43.7% of female middle school and high-school students reported the use of unhealthy weight control behaviors (Neumark-Sztainer et al., 2011). Results from the nationwide YRBSS survey indicate that the use of unhealthy weight control behaviors is a concern among female high-school students, based on the following prevalence estimates: 17.4% had fasted, 5.9% had taken diet pills, and 6.0% reported the use of laxatives or vomiting (CDC, 2012). Fortunately, the use of risky dieting behaviors in adolescent females has decreased in recent years (Neumark-Sztainer et al., 2012; CDC 2012).

The proposed research will highlight the role of genetic predisposition on dieting and weight gain by studying twins. It remains unclear why people choose to diet and if this is due to their propensity for weight gain or if their actions to diet result in unintentional weight gain. Furthermore, if those who diet at a younger age are more prone to chronic weight problems, including a higher BMI in adult life, then this could be useful for educating families to watch for signs of dieting in their children. Interventions could then be implemented to decrease chronic weight
problems experienced later in life, which are associated with adverse health outcomes such as cardiovascular disease, type II diabetes, hypertension and stroke (Field et al., 2001). Lastly, young children concerned with their weight and attempt to diet may also be more prone to riskier dieting behaviors as adolescents and adults. Not only are these associated with a greater weight gain in the long-term, but also increase one’s risk for developing an eating disorder (Larson et al., 2009).

The primary aim of this study is to determine the relationship between age at dieting onset and BMI in female twins. I hypothesize that an earlier age at dieting onset is associated with a greater BMI in both cross-sectional (i.e., at the time of first survey completion) and longitudinal (i.e., at the time of second survey completion) analyses.

The secondary aim will be to test the association between an earlier age of dieting with unhealthy dieting behaviors in female twins. I hypothesize that individuals who begin dieting at an earlier age will be more likely to engage in risky dieting behaviors.

The third aim will be to test the above analyses within twin pairs. These analyses will assess the influence of genetics and familial factors for age at onset of dieting and dieting outcomes, including BMI.

Lastly, I will investigate the age at onset of dieting in women by age group, to determine which dieting age group is associated with the greatest risk for elevated BMI. This will be helpful to determine if dieting at specific ages puts individuals at a
greater risk for greater BMI and risky eating behaviors compared to others who diet later.

Ultimately, the knowledge gained from the proposed study may be useful for designing interventions to alleviate chronic conditions associated with disordered eating behaviors and weight gain.
Methods

Subjects

Participants were female twins, aged 18 to 60 years old from the University of Washington Twin Registry. Twins of both male and female gender from the state of Washington were recruited from driver’s license applications and the University of Washington Twin Registry Health Survey of 2006 was mailed out to everyone in the registry. This self-reported survey achieved a 55% response rate. A subsequent follow-up survey, the Health and Wellbeing Questionnaire, was mailed out and collected in 2009 and 2010 for an average interim of follow-up of approximately 3 years. Only the individual’s self-reported height and weight from the follow-up survey were used.

Overall, 1,545 women completed the Twin Registry Health Survey of 2006. The exclusion criteria for this analysis consisted of age greater than 60 years of age (n=358), twins who did not live together prior to age 18 (n=227), and individuals with gastric bypass surgery (n=8). Two subjects were excluded from the analysis for an implausible age at dieting onset. After exclusions, 950 eligible women who also completed a second survey were available in the present analyses.

Measures:

Age at onset of dieting for weight loss was measured from the self-reported, retrospective question “How old were you when you first attempted to lose weight?”
BMI (kg/m²) at Time 1 was calculated from self-reported weight and height given in the 2006 survey, “how tall are you without shoes?” and “how much do you weight without clothes or shoes?” BMI at time 2 was calculated from self-reported height and weight of the Health and Wellbeing Survey (2009/2010). The question asked, “What is your current height (in feet and inches) and weight (in pounds)?” Change in BMI was then calculated as the difference between BMI at Time 2 to Time 1.

Disordered eating behaviors were measured via risky dieting behavior methods used in the past 12 months. The questions asked included, “During the past 12 months, how did you try to lose weight? Skipped meals/fasted? Took diet pills prescribed by a doctor? Took laxatives or vomited?” These measures have all been associated with risky dieting behaviors and unhealthy weight control habits (Larson et al., 2009; Neumark-Sztainer et al., 2006; CDC 2012). Other dieting methods were asked but not used in this analysis because they were not considered risky dieting behaviors. Individuals who engaged in one or more risky dieting behaviors were grouped together and compared to those who did not use any of the above methods for weight loss.

The Restraint Scale has been used as a measure for restrained eating and assessment for frequent dieting, disinhibited eating, and body dissatisfaction in women (Herman and Polivy et al., 1980; van Strien et al., 2007). The survey included 10-questions regarding weight fluctuations and feelings about eating behaviors and
current weight such as “Would a weight fluctuation of 5 pounds affect the way you live your life? How conscious are you of what you are eating? Do you have feelings of guilt after overeating? What is the maximum amount of weight you have ever lost in 4 weeks?” Questions were ranked on a scale from 0 to 3 or 4 and all scores were summed to give an overall restraint scale score from 0 to 35. The validity of the Restraint Scale has been assessed, and while it was predictive of many behaviors, it may overestimate restraint in overweight individuals (van Strien et al., 2007). Regardless, it is a fairly accurate measure of restrained eating behaviors that are associated with dieting behaviors.

Weight cycling was assessed from the following question, “In your lifetime, how many times have you lost 10 or more pounds on purpose?” The response options were: 0, 1-2, 3-4, and 5+. Individuals who had lost 10 or more pounds on purpose at least one time were compared to those who had never reported weight cycling.

Analyses

Statistical analyses were performed on STATA 11C. Descriptive statistics including the means and standard deviations were completed for continuous variables, whereas the percentage of use was assessed for binary variables.

Generalized estimating equations (GEE) with robust standard errors were used to examine the relationship between age at dieting onset to BMI at time of first survey completion, change in BMI, risky eating behaviors, restrained eating, and
weight cycling of individuals. In addition, adjusted models controlled for the covariates determined \textit{a priori}. GEE was used to account for the twin’s relatedness, so that these analyses mimic those of unrelated individuals.

Within-pair analyses were conducted using linear regression equations, which accounted for each twin pair. These analyses were used to assess the difference in age at dieting onset to the differences between twins for the dependent variables used in the overall analyses. By comparing twins to each other, these analyses are independent of age as well as genetic and familial factors.

The final analysis used GEE to account for twin relatedness in order to determine the association between percentiles for age at onset of dieting with BMI at time of assessment. Age groups were categorized based on quartiles, 25\% (ages 15-17), 50\% (ages 18-24) and 75\% (ages 25+) and compared to those who dieted the earliest (younger than 15).
Results

The means and standard deviations of participant characteristics are given in Table 1. Participating women had a mean BMI of 25.8 ± 5.5 kg/m² with a range of 15.6 kg/m² to 50.63 kg/m². The mean age of dieting onset was 20.8 ± 9 years, with a range of 8 to 59 years of age. On average, participants experienced a mean change in BMI of 0.76 kg/m², with a range of -9.0 kg/m² to 10.8 kg/m² between completion of the two surveys. The overall restraint scale score was 15.7 ± 4.84 and included a range of scores from 3 to 31.

There were also specific participant characteristics for the percentiles at age of dieting onset (Table 1). Individuals who recalled their first attempt to diet prior to age 25 were on average between the ages of 31-34 at the time of assessment. However, those who recalled their first attempt to diet older than 25 years of age were significantly older at the age of assessment, with a mean age of 47 years old. This is important because the three groups who dieted <15 years of age, between 15 and 17 years of age, and those 18-24 had a fairly even age at time of assessment. On the other hand, the older individuals tended to report their age at dieting onset older than 25 may not be as reliable as the others.

Aside from the variance in age at assessment between the age at dieting onset percentiles there were minimal differences in racial demographics between the groups. The sample population was predominantly Caucasian (89.5%), followed by Hispanic/Latino (4.4%), Other (3.3%), Asian (3.0%), American-Indian/Alaskan
Native (2.9%), African-America (1.5%), and American Hawaiian/Pacific Islander (0.7%).

**Overall association of age at dieting onset with BMI at time of baseline assessment:**

Unadjusted models show no relationship between the age at dieting onset with BMI at time of baseline assessment (Table 2). When adjusted for age at time of the assessment, there was a significant association between age at dieting onset with BMI at time of baseline assessment (p=0.003) (Table 2). The results indicate that for every year increase in age at dieting onset there was a .06 kg/m² decrease in BMI. A subsequent model adjusted for ethnicity and income remained significant (p=0.004).

**Association between age at dieting onset to change in BMI:**

Change in BMI between the two surveys was not associated with age at dieting onset in either unadjusted (p=0.21) or adjusted (p=0.91) models (Table 2).

**Within-pairs association for difference in age at dieting onset to difference in BMI:**

In within-pair analyses, there were no significant associations between age at dieting onset and BMI at time of baseline assessment (p=0.21) or change in BMI (p=0.24) (Table 3). A subsequent within-pair analysis of BMI at time of baseline assessment stratified by zygosity similarly did not yield significant results.
**Association between age percentiles for age at dieting onset and BMI at time of baseline assessment:**

Next, age at dieting onset was divided into quartiles, creating the following age groups: ages 15-17, ages 18-24, and ages 25+, which were all compared to the earliest group of dieters younger than 15.

A significant association existed for the relationship between age at dieting onset and BMI for the groups who began dieting at ages 18-24 (p=.004) and those 25 years and older (p=.002) when compared to the youngest dieting group (Table 4). The mean BMI of individuals who began dieting younger than 15 years-old was 26.4 ± 6 kg/m², compared to those who began dieting from 18-24 years-of-age, 25.3 ± 5.4 kg/m², and those greater than 25 years-old, 25.7 ± 5.4 kg/m² (Table 4). The results indicate that individuals who began dieting at age 18 or older tend to have lower BMI’s than individuals who began dieting prior to age 15. There was no significant association for the group who began dieting from 15-17 years of age as compared to the youngest (reference) age group (p=0.689)(Table 4).

**Association between the age at dieting onset to risky dieting behaviors:**

The measure of risky dieting behaviors was analyzed between individuals who engaged in any number of risky behaviors compared to those who used none. In the unadjusted model, an earlier age at dieting onset was associated with the use of risky dieting behaviors (β=-.018 and p=.025) (Table 5). Adjusted models showed
a stronger association between an earlier age dieting onset and the use of risky behaviors (p=0.016) (Table 5).

*Association between age at dieting onset and Restraint Scale score:*

There was a significant association between higher restraint scale scores and a younger age at dieting onset in both unadjusted and adjusted models (p=<0.001)(Table 5). For every year increase in age at dieting onset, the restraint scale score decreased by .13. There was no significant association for the within-pair analyses (Table 5).

*Association between weight cycling and age at dieting onset:*

The unadjusted model showed no significant relationship between an earlier age at dieting onset and episodes of weight cycling within the past year (Table 5). However, once adjusted for age at baseline assessment, there was a significant association between weight cycling and earlier age at dieting onset (p=.011)(Table 5). The within-pair analyses were not significant when adjusted for genetics and familial factors between twin pairs (p=.059).
Discussion

Among a large group of female twins, we found that an earlier age at dieting onset was associated with a greater BMI at baseline, higher restrained eating, the use of risky dieting behaviors, and a greater number of weight cycling episodes when accounting for baseline age. Subsequent within-pair analyses, which controlled for the twin’s shared genetic and family environment factors found no significant associations between age at dieting onset and our outcome measures of interest.

The use of within-pair analyses, in addition to the overall analyses, allowed for the opportunity to analyze the influence of genetics and familial factors on the above associations. Because there no longer was a significant association once these shared factors were accounted for, our findings demonstrate that genetics and familial factors influence the age at dieting onset as well as BMI and dieting behaviors.

Similar to the overall association in this analysis, prior studies support the association between dieting and weight gain (Neumark-Sztainer et al., 2006; Larson et al., 2009; Kruger et al., 2004; Neumark-Sztainer et al., 2007). However, there is an ongoing debate as to the exact mechanism by which dieting leads to weight gain. The two predominant hypotheses supported in the literature are that dieting itself leads to an increased BMI, or that those who diet are genetically predisposed to weight gain regardless of their attempts to diet for weight loss.
A number of studies indicate that a heavier weight precedes dieting. Girls who were already considered overweight at age 5 had higher levels of disinhibited eating, dietary restraint, overweight concern, and body dissatisfaction by age 9 (Shunk et al., 2004). Overweight female adolescents who reported dieting and use of unhealthy weight control behaviors had greater increases in BMI compared to normal weight females at 10-year follow-up (Neumark-Sztainer et al., 2011). Additionally, a survey of binge eating adults concluded that the majority reported being overweight prior to dieting and binge eating (Emmons 1994).

On the other hand, the majority of dieters are unsuccessful at losing weight in the long term and dieting methods have been associated with weight gain. A recent twin study from Finland, in which intentional weight loss episodes between MZ and DZ twins was analyzed, may shed some light on this issue (Pietilainen et al., 2012). Twins with more than one episode of intentional weight loss had a greater weight gain compared to those with no instances of intentional weight loss. In MZ pairs discordant for intentional weight losses, the twin with more intentional weight loss episodes was heavier at follow-up compared to their non-dieting twin although there was no significant difference between the twins BMI at baseline (Pietilainen et al., 2012). Other studies found the use of specific dieting methods to be associated with weight gain. For example, in one study highly restrained eating behaviors in women was associated with weight gain over six years (Drapeau, et al., 2003). Schur and colleagues discovered a significant relationship between restrained eating and a higher BMI and weight gain in twins (Schur et al. 2010). Together these
studies provide evidence that certain dieting behaviors may be related to weight gain regardless of the individual’s genetic predisposition for BMI.

Although dieting has been associated with weight gain, not all women who diet are overweight, and many women of normal weight diet. Results from NHANES demonstrated that dieting was related to an individual’s perceived weight rather than their actual weight (Strauss 1999). In fact, 2/3 of adolescents who viewed themselves as overweight reported having dieted in the past year regardless of their normal-weight status (Strauss 1999). Similarly, a study comparing the influences between dieters and nondieters from a group of high school seniors found a majority of the dieters were not overweight, although they perceived themselves to be overweight (Reas 2007). Therefore, many normal weight individuals may diet because of body dissatisfaction rather than a genetic predisposition for a larger BMI. It is therefore critical to understand whether dieting behaviors among normal weight individuals constitute a risk factor for excess weight gain.

Although the significant findings from this study conclude an earlier age at dieting onset is associated with a greater BMI and use of risky dieting behaviors, it does not directly support either of preexisting hypotheses for the mechanism by which dieting contributes to weight gain. The first possible hypothesis is dieting individuals are predisposed for weight gain and unsuccessfully diet. The results of this study concluded that genetics and the family environment influences an individual’s age at dieting onset and also their BMI and use of dieting behaviors.
However, we cannot make any conclusions for how an individual’s genetic predisposition or propensity for weight gain would influence their age at dieting onset or BMI from this analysis. We do not have the weight of the individual at their reported time of dieting onset. Therefore, we cannot make any associations regarding how one’s weight status influenced age at dieting onset or BMI. Additionally, although it was concluded that an earlier age at dieting onset is associated with a greater BMI we do not know if dieting itself contributed to this weight gain. Based on the participant characteristics there were many individuals of a normal weight-status who reported dieting and use of dieting behaviors. However, it was out of the scope of this study to determine how one’s weight status influenced their BMI or dieting behaviors.

Ultimately, it appears that the complex relationship between dieting and weight gain is embedded in not only one’s genetic predisposition for BMI, but also familial and environmental factors. Individuals choose to diet regardless of their weight status and likely for a multitude of other reasons that would require further research in order to unravel the true relationship. The within-pair analysis for age at dieting onset and BMI was insignificant and we can infer that this relationship is influenced by genetic and familial factors such as growing up with unhealthy eating habits, low physical activity levels, and the influence of other family members regarding their weight and dieting habits. These influences are evident as individuals of both normal and overweight status diet. Whether that is the influence of genetics, family, social factors, or a combination cannot be elucidated from our
data. Needless to say, individuals who are predisposed to a higher weight are probably more influenced by their genetic and family environment to diet to lose weight and engage in risky dieting behaviors that are also associated with weight gain. Therefore, subsequent studies are necessary in order to elucidate the true mechanism by which dieting leads to weight gain.

*Age at dieting onset and BMI*

Although these data are not conclusive, they do support an association between dieting earlier with a higher weight status. Because of this study's retrospective design, it is difficult to accurately assess the individual's true age at onset of dieting as well as their natural propensity for weight gain because we have no measures from when they reported their first attempt to diet. The only concrete results from this study indicate that genetics and family environment contribute to the relationship between age at dieting onset and BMI. Future longitudinal studies should follow adolescent twins and document when they first began dieting, their initial BMI, use of weight control behaviors, and subsequent changes in BMI.

*Change in BMI*

Although there was a significant difference between those who dieted earlier and their BMI at time of baseline assessment, there was no significant relationship for change in BMI between the two surveys. This may be attributed to a short follow-up between surveys of only three years, hence an insufficient amount of time for significant weight gain. Additionally, it is well known that individuals, especially
those who are overweight, tend to underestimate self-reported weight and may have elected to put a lower weight at follow-up (Nawaz et al., 2001).

*DiETING METHODS (RISKY DIETING BEHAVIORS, RS, AND WEIGHT CYCLING)*

In overall, adjusted analyses, an earlier age at dieting onset was associated with the use of dieting methods such as, risky dieting behaviors, a higher Restraint Scale score, and weight cycling episodes, compared to those who dieted later. However, results from within-pairs analyses found no association between the above dieting measures and age at dieting onset when genetic and familial factors were accounted for. Therefore, the use of dieting methods is highly influenced by the family environment. In particular, mother’s preoccupied with their weight and dieting has been associated with unhealthy dieting behaviors in adolescents (Neumark-Sztainer et al., 2009). Additionally, a mother’s preoccupation with her own weight and encouraging her daughter to lose weight has been associated with restrained eating behavior in their daughters (Francis and Birch 2005).

It was out of the scope of this analysis to assess how the number of risky dieting behaviors differed by age at onset of dieting or how they changed over time. Fortunately, there is existing data for adolescents’ dieting methods. Project EAT followed adolescents ages 12-16 over a 10-year period and documented their weight and weight control habits (Neumark-Sztainer et al., 2011). They found that the younger group of adolescents (ages 12-13) maintained a constant prevalence of unhealthy weight control behaviors (fasted, skipped meals, ate very little food, ate a
food substitute) throughout the duration, whereas the older girls (15-16) decreased their use of unhealthy weight control habits as they progressed into young adulthood (Neumark-Sztainer et al., 2011). However, the prevalence of extreme dieting behaviors (use of diet pills, laxatives, diuretics, and vomiting) increased for all females as they matured into young adulthood (Neumark-Sztainer et al., 2011).

The use of more extreme dieting methods and greater body dissatisfaction into adulthood is not only related to weight gain but also to disordered eating habits, which could be a predictor for eating disorders. The dieting individuals in Project EAT who used unhealthy weight control behaviors had outcomes related to obesity and disordered eating at just 5 years of follow-up (Neumark-Sztainer et al., 2006). The adolescents who reported use of unhealthy weight control behaviors at Time 1 were at the greatest risk for disordered eating behaviors, and at twice the risk for an eating disorder at Time 2 (Neumark-Sztainer et al., 2006). A 3-year cohort study performed by Patton and colleagues found dieting was the most important predictor of new eating disorders in female adolescents and those who dieted at a more severe level were 18 times more likely to develop an eating disorder compared to nondieters (Patton 1999).

In the present analysis, only the top quartile of participants documented the use of risky dieting behaviors. This resulted in a small sample size to investigate the association between age at dieting onset and risky dieting behaviors. A future study could use a population that engages in more risky behaviors as a whole, as well as
analyze how the number of risky dieting behaviors compares between ages of dieting onset. Also, because this study only asked retrospective questions regarding behaviors used in the past year, we were unable to deduce any type of association between an individual's previous or future use of risky dieting behaviors.

Quartiles for age at dieting onset and BMI

An important aim of this study was to analyze if specific quartiles for age at dieting onset placed one at a higher or lower risk for increased BMI. The upper three quartiles of age at dieting onset were compared to the youngest quartile group, defined as those who dieted younger than 15 years-of-age. There was a significant association between those who dieted later, specifically individuals in the third and fourth quartile compared to the reference group, which translates to a dieting onset of 18 years or older and decreased BMI. Thus, an onset of dieting as an adult compared to a pre-teen may be protective of additional weight gain.

There could be numerous explanations for why the majority of women begin dieting as an adult and consequently have a lower BMI. First, women may begin dieting at this age to avoid or mitigate the dreaded “freshman 15,” the term used to describe the common weight gain associated in the first year of entering college, while also attempting to make healthier, more sustainable dietary and exercise choices as they mature into their adult years. In actuality, the average weight gain in females after the first year of college is 4 pounds (Holm-Denoma et al., 2008). Secondly, the body’s natural metabolism begins to slow from its peak in adolescence
(Ortega-Alonso et al., 2012). Dieting may be an attempt to offset the natural decline in metabolism and increase in fat mass that comes with increasing age.

Overall, these results illustrate that the delay of dieting onset until adulthood may be associated with lower BMIs. Largely because those who begin to diet later do so for very different reasons from those who diet earlier. Thus, it may be that two separate populations of individuals make up the groups who diet earlier, and those who diet later. It is plausible that the reasons why an individual chooses to diet are more important than the act of dieting itself in determining their future BMI.

Limitations

These analyses are not without limitations. The sample population was demographically homogenous and cannot be extrapolated to other populations. The mean BMI of participating individuals was barely considered overweight, which is significantly underweight compared to other populations. Therefore, their involvement in dieting and use of various dieting behaviors may differ dramatically from other populations and may not show as strong of an association. Because this was a self-reported, at-home survey, all of the participants self-selected to participate in both surveys, which could result in selection bias. Individuals who chose to participate in the survey may have different backgrounds and answers than those who chose not to participate. Although we do not know the characteristics of those who did not respond they likely vary from those who did.
There were additional potential sources of bias with the study design. A retrospective study can be influenced by inaccurate or biased recall on the part of the respondent's. Each question pertained to certain behaviors, thoughts, or feelings, which occurred over either the past 4 weeks or the previous 12 months. The inconsistency between the two time periods could make it difficult to answer correctly if one was not paying attention to each individual question.

The primary variable of interest, age at dieting onset, required the individual to recall when they first engaged in dieting behavior in order to lose weight. Unfortunately, due to the recall nature of this question the exact age that one answered could be inaccurate. Because the question only asked “How old were you when you first attempted to lose weight?” there is no information regarding how successful their dieting attempt was, how many subsequent dieting attempts they had, or if they were continually dieting at the time they took the survey. Future studies should take into account how dieting behaviors change over time and if there is a difference between those who diet only a few times versus those who are continually dieting. Lastly, we had no measure of the individual’s BMI at their reported age at dieting onset. Therefore, we cannot extrapolate these results to show how weight status influenced the age at dieting onset nor future BMI or risky eating behavior outcomes.

Height and weight measures were self-reported with no assessment for the accuracy of these responses. It has been documented extensively that individuals,
especially overweight individuals, have a tendency to underreport their weight (Nawaz et al., 2001). This could once again bias the results by underestimating the true effect an earlier age at dieting onset can have on subsequent weight gain.

Lastly, there were limitations regarding the sample sizes used in the various analyses. The overall analyses used 950 individuals, whereas when these were performed within-pairs the number dropped drastically to 236 or 224 pairs. Therefore, many individuals were part of the overall analyses but not within-pairs, and if we had a larger sample size of twin pairs to assess their genetic and family environmental factors, our results may have been different.

Conclusions

The results of the overall and within-pair analyses demonstrate some significant findings. For example, a younger age at dieting onset is associated with increased BMI, and use of negative outcomes such as higher restraint scale scores, risky dieting behaviors, and episodes of weight cycling. However, this relationship was not independent of genetics. The within-pair analyses demonstrate that genetic and family factors influence the relationship between age at dieting onset and both BMI and dieting behaviors.
Tables

Table 1: Participant characteristics by ages of dieting onset (years)\(^1\)

<table>
<thead>
<tr>
<th>N=</th>
<th>Overall</th>
<th>&lt;15</th>
<th>15-17</th>
<th>18-24</th>
<th>25+</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>36.3 (14.5)</td>
<td>32.2 (12.6)</td>
<td>31.3 (12.8)</td>
<td>34.5 (14.2)</td>
<td>47.5 (12.3)</td>
</tr>
<tr>
<td>Zygosity(%)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MZ (0)</td>
<td>54.6</td>
<td>57.5</td>
<td>56.9</td>
<td>52.4</td>
<td>52.7</td>
</tr>
<tr>
<td>DZ (1)</td>
<td>42.1</td>
<td>39.2</td>
<td>41.7</td>
<td>43.0</td>
<td>43.5</td>
</tr>
<tr>
<td>IZ</td>
<td>3.2</td>
<td>3.2</td>
<td>1.2</td>
<td>4.5</td>
<td>3.6</td>
</tr>
<tr>
<td>BMI(^2) (kg/m(^2))</td>
<td>25.8 (5.5)</td>
<td>26.4 (6.0)</td>
<td>26.1 (5.9)</td>
<td>25.3* (5.4)</td>
<td>25.7* (4.7)</td>
</tr>
<tr>
<td>Change BMI (kg/m(^2))</td>
<td>0.76 (2.48)</td>
<td>0.815 (2.77)</td>
<td>0.90 (2.6)</td>
<td>0.72 (2.4)</td>
<td>0.63 (2.2)</td>
</tr>
<tr>
<td>Age at dieting onset (years)</td>
<td>20.8 (9.0)</td>
<td>12.4 (1.55)</td>
<td>15.9 (.77)</td>
<td>19.9 (1.8)</td>
<td>34.6 (8.4)</td>
</tr>
<tr>
<td>Restraint Scale</td>
<td>15.7 (4.84)</td>
<td>17.8 (4.87)</td>
<td>15.9 (5.15)</td>
<td>14.9 (4.4)</td>
<td>14.8 (4.4)</td>
</tr>
<tr>
<td>Weight cycling #times (%)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>0</td>
<td>21.0</td>
<td>13.9</td>
<td>21.3</td>
<td>26.6</td>
<td>18.9</td>
</tr>
<tr>
<td>1-2</td>
<td>44.2</td>
<td>41.9</td>
<td>42.7</td>
<td>43.5</td>
<td>49</td>
</tr>
<tr>
<td>3-4</td>
<td>20.8</td>
<td>22</td>
<td>20.5</td>
<td>18.5</td>
<td>23.6</td>
</tr>
<tr>
<td>5+</td>
<td>13.7</td>
<td>22</td>
<td>15.3</td>
<td>11.3</td>
<td>8.3</td>
</tr>
<tr>
<td>Risky behaviors(^3) (%)</td>
<td>41.6</td>
<td>50.5</td>
<td>43.1</td>
<td>38.6</td>
<td>36.6</td>
</tr>
</tbody>
</table>

\(^1\)All values are given as means (s.d), unless otherwise specified
\(^2\)BMI = Body Mass Index (kg/m\(^2\))
\(^3\)Risky Behaviors = % reported the use of at least one risky dieting behavior
Table 2: Overall analyses for age at dieting onset to initial and change in BMI

|                  | N   | Coefficient | z    | p>|z|   | CI             |
|------------------|-----|-------------|------|-------|----------------|
| **Unadjusted**   |     |             |      |       |                |
| BMI^2            | 938 | -0.003      | -0.20| 0.845 | (-0.039 0.032) |
| Change BMI       | 936 | -0.009      | -1.09| 0.277 | (-0.026 0.007) |
| **Adjusted^1**   |     |             |      |       |                |
| BMI              | 938 | -0.06       | -2.95| 0.003 | (-0.106 -0.021)|
| Change BMI       | 936 | -0.00       | -0.12| 0.905 | (-0.020 0.018)|

^1 Adjusted for age at time of assessment

^2 BMI= Body Mass Index

^3 P-values were derived from GEE with robust standard errors

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Table 3: Within-pair analyses for age at dieting onset to initial and change in BMI

|                  | N   | Coefficient | z    | p>|z|   | CI             |
|------------------|-----|-------------|------|-------|----------------|
| BMI              | 237 | -0.035      | -1.27| 0.207 | (-0.090 .019)  |
| Change BMI       | 236 | 0.024       | 1.10 | 0.273 | (-0.019 .068)  |

^1 P-values were derived from simple and multiple linear regression models

^2 BMI= Body mass Index
Table 4: Quartiles for age at dieting onset and BMI

<table>
<thead>
<tr>
<th>Mean BMI CI</th>
<th>&lt;15</th>
<th>15-17</th>
<th>18-24*</th>
<th>25+*</th>
</tr>
</thead>
<tbody>
<tr>
<td>(22.58-25.12)</td>
<td>23.85</td>
<td>23.63</td>
<td>22.27</td>
<td>21.93</td>
</tr>
<tr>
<td>(22.53-24.72)</td>
<td>(21.20-23.34)</td>
<td>(20.73-23.13)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>0.689</td>
<td>0.004</td>
<td>0.002</td>
<td></td>
<td></td>
</tr>
<tr>
<td>-0.40</td>
<td>-2.90</td>
<td>-3.14</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Significantly different from group who dieted younger than 15 years of age
** Adjusted for age at time of assessment

1P-values were derived from GEE with robust standard errors

Table 5: Overall and within-pair analyses for age at dieting onset to dieting behaviors

| N | Coefficient | z | p>|z| | CI |
|---|-------------|---|-------|-----|
| Restraint Scale Scores: | | | | |
| Overall | 913 | -.081 | -4.62 | 0.000 | (-.116 -.047) |
| Adjusted | 913 | -.129 | -6.41 | 0.000 | (-.169 -.090) |
| Within-pair | 224 | -.034 | -1.39 | 0.167 | (-.082 .014) |
| Risky Behaviors | | | | |
| Overall | 950 | -.018 | -2.24 | 0.025 | (-.034 -.002) |
| Adjusted | 950 | -.021 | -2.41 | 0.016 | (-.039 -.004) |
| Within-pair | 184 | .96 (OR) | -1.34 | 0.181 | (.919 1.01) |
| Weight cycling: | | | | |
| Overall | 944 | -.002 | -0.34 | 0.733 | (-.019 .013) |
| Adjusted | 944 | -.030 | -2.54 | 0.011 | (-.054 -.007) |
| Within-pair | 241 | -.013 | -1.89 | 0.059 | (-.028 .000) |

1P-values were derived from simple and multiple linear regression models, and GEE with robust standard errors
References


Shunk, Jennifer et al., “Girls at risk for overweight at age 5 are at risk for dietary restraint, disinhibited overeating, weight concerns, and greater weight gain from 5 to 9 years” J Am Diet Assoc. 104 (2004):1120-1126.


