Received: 19 March 2021 | Revised: 2 July 2021 | Accepted: 2 August 2021

DOI: 10.1002/oby.23285

Obesity A Research Journal | THE OBESITY SOCIETY | WILEY

ORIGINAL ARTICLE

Epidemiology/Genetics

Drive for thinness in adolescents predicts greater adult BMI in the Growth and Health Study cohort over 20 years

Barbara A. Laraia[1] | **Cindy W. Leung**[2] | **A. Janet Tomiyama**[3] | **Lorrene D. Ritchie**[4] | **Patricia B. Crawford**[4] | **Elissa S. Epel**[5]

[1]School of Public Health, University of California, Berkeley, Berkeley, California, USA

[2]School of Public Health, University of Michigan, Ann Arbor, Michigan, USA

[3]Department of Psychology, University of California, Los Angeles, California, USA

[4]Division of Natural Resources, Nutrition Policy Institute, University of California, Oakland, California, USA

[5]Department of Psychiatry, University of California, San Francisco, California, USA

Correspondence
Barbara A. Laraia, Public Health Nutrition, School of Public Health, University of California, Berkeley, 2121 Berkeley Way West, Berkeley, CA 94720-7360, USA.
Email: blaraia@berkeley.edu

Funding information
Eunice Kennedy Shriver National Institute of Child Health and Human Development, Grant/Award Number: R01HD073568; National Heart, Lung, and Blood Institute, Grant/Award Number: R56HL141878; National Institute on Aging, Grant/Award Number: R01AG059677 and R56AG059677.

Abstract

Objective: In youth, a preoccupation with weight and the desire to be thinner, or drive for thinness, might persist into adulthood and predict reward-based compulsive eating and greater weight status.

Methods: A total of 623 women were enrolled from a prospective cohort study starting at 10 years old and assessed up to 20 years later. Drive for thinness was measured five times during adolescence. In adulthood (mean age = 39.5), drive for thinness, reward-based eating drive, and BMI were measured.

Results: Structural equation modeling found cumulative adolescent drive for thinness predicted higher scores for both adult drive for thinness and reward-based eating drive. Youth drive for thinness was not directly associated with adult BMI but rather indirectly through adult drive for thinness. Reward-based eating drive was not associated with adult BMI.

Conclusions: Drive for thinness during the critical developmental years may exert long-term effects on adulthood eating behaviors tied to greater weight gain, potentially reflecting an important early target of intervention.

INTRODUCTION

In our modern food environment, a drive to restrict eating may be necessary to avoid weight gain, but excessive thoughts and emotions around dieting and weight gain can have unintended consequences. A strong drive to restrict eating and to lose weight may paradoxically result in weight gain among normal-weight women (1). In survey and longitudinal cohort studies, self-reports of dieting and weight control are prospective predictors of weight gain, rather than loss, in adolescents and adults (1-3). For example, in adolescent females, both self-reported dieting status and dietary restraint were related to prospective risk of obesity (2). Dieting is also prospectively positively associated with obesity in women (4,5). In a review, Lowe and colleagues (1) found that self-reported weight loss dieting in the past year was related to prospective weight gain in 15 out of 20 studies.

A preoccupation with thinness and negative emotions after eating certain foods may lead to counterproductive dieting. This preoccupation is well measured by the concept and scale for "drive for thinness." The drive for thinness scale, developed as part of the Eating Disorder Inventory (EDI) (6), captures negative emotions, thoughts and preoccupation with weight, and extreme pursuit of thinness. Drive for thinness is associated with dieting behaviors (7) and is highly correlated with body dissatisfaction, independent of eating disorder or depression (8).

There are several pathways through which drive for thinness might lead to dieting and ultimately dysregulated eating behavior, such as reward-based eating drive, and, in some cases, weight gain. Dieting is associated with impaired cognitive performance (9,10) and negative effects on cognitive function (11). When attempting excessive cognitive restraint to chronically manage food intake, people's attention is taxed, and they can easily lose restraint and inadvertently increase intake (12). Chronic self-monitoring of food intake is a source of cognitive load, and in some but not all studies, this leads to less self-control over eating and other impulsive behaviors (13-15). Rigid, "all-or-none" dieting, restricting certain comfort foods, puts individuals at risk of disinhibited overeating when common dietary lapses occur (16) and leaves individuals vulnerable to overeating in response to emotions or external cues (17,18), possibly because of the commonly found low prefrontal cortex/limbic balance under states of stress (19,20).

Dieting can also serve as a chronic source of stress. In one study, restricting caloric intake using calorie counting in order to lose weight increased perceived stress and elevated activity of the hypothalamic–pituitary–adrenal axis as reflected by daily salivary cortisol (21). These experiences of stress may make one more vulnerable to dysregulated eating, creating a cycle of unhealthy behaviors that promote weight gain (22).

Dieting, especially in the presence of stress, may increase the reinforcement value of food, leading to excessive drive to eat (23,24). Reward-based eating is conceptualized as a common compulsive eating behavior, but one that drives overeating and obesity. Reward-based eating drive (RED) is measured by three issues: feeling one cannot stop overeating, eating rapidly or mindlessly without perceiving fullness or satiation, and excessive thoughts of food and eating. RED is associated with eating hyperpalatable foods, food craving, weight gain, and, at extremes, developing obesity (24,25).

We hypothesize that psychological drive for thinness, which includes cognitive and behavioral measures indicative of constant dieting, may drive RED and obesity. In fact, previous results from the longitudinal NHLBI Growth and Health Study (NGHS) (26) children found that drive for thinness was higher for Black girls at age 10 but was associated with higher BMI for both White and Black girls in a cross-sectional analysis (7). Using data from NGHS, we found that the strongest predictors of BMI increase in girls from age 10 through 19 were family socioeconomic position (income and parent education) and drive for thinness (27).

It is unclear whether childhood drive for thinness might also have long-term consequences: Does it track through life, manifesting as a high drive for thinness in adulthood? Does it lead to both reward-based eating and adulthood obesity? Here we conducted a follow-up of this cohort, allowing us to address these questions. The objective of this investigation was to determine the long-term consequences of adolescent drive for thinness in women who participated in NGHS as children, investigating adult eating behaviors and BMI approximately 30 years later. We asked: (1) Does adolescent drive for thinness predict adult drive for thinness and elevated reward-based eating? (2) Does adolescent drive for thinness predict higher adulthood BMI? If so, is it a direct relationship, or does it work through adult drive for thinness or adult reward-based eating?

Study Importance

What is already known?

- ► Dieting and weight control are associated with weight gain in the short term.
- ► Dieting has been found to be a source of stress.

What does this study add?

- ► Drive for thinness during critical developmental periods has long-term effects on adulthood eating behaviors.
- ► Drive for thinness throughout life leads to greater weight gain in midlife.

How might these results change the direction of research or the focus of clinical practice?

- ► Preoccupation with thinness and negative emotions about eating may be important predictors of metabolic health.
- ► Interventions are needed to help women manage negative thoughts and emotions about weight and eating.

METHODS

Study sample

NGHS is a population-based cohort study of White and Black girls recruited from two communities (Cincinnati, Ohio, and Richmond, California) and from families enrolled in a health maintenance organization in Washington, DC. A total of 2,379 girls (1,213 Black and 1,166 White) who were 9 or 10 years old were recruited between 1987 and 1988 and assessed in 10 annual measurement points. The original objective of NGHS was to investigate racial differences in dietary intake, physical activity, family, and psychosocial factors associated with the development of obesity from preadolescence through maturation. NGHS also sought to examine effects of obesity on cardiovascular disease risk factors. NGHS procedures included a physical examination, anthropometric measurements, dietary information, physical activity, lipoprotein profiles, family socioeconomic status, and psychosocial information. Extensive information on this study and sample are available elsewhere (26).

NGHS follow-up study

We conducted the follow-up study from January 2016 to September 2019 with women from the Richmond, California, cohort (n = 882) in the original NGHS study. We found that 29 years after enrollment

into the study, there was still satisfactory recruitment and retention of this sample of women: 624 women (73.3% response rate) of 850 eligible participants (32 women were deceased, lost to follow-up after the initial year of the study, or institutionalized).

The analytic sample included 614 women (300 Black, 314 White) with complete eating and BMI data (98.6%). Using baseline demographic information, we found that a higher percentage of participants were White, were from two-parent households, had the highest parental education level, and had the highest income level, compared with eligible nonparticipants. There was no difference in mean baseline BMI between participants and nonparticipants. All procedures were approved by the Committee on Human Research of the University of California, Berkeley.

Dysregulated eating behaviors

We measured drive for thinness in both childhood and in adulthood and RED only in adulthood. The EDI was administered when the girls were ages 12, 14, 16, 18, and 19 years; therefore, adolescent drive for thinness was averaged across these time points. We found that the mean and median values did not change much across these time points, and at each time point, the childhood drive for thinness measure was associated with adult BMI. Sample items are, "I think about dieting," "I am terrified of gaining weight," "If I gain a pound, I worry that I will keep gaining," "I feel extremely guilty after overeating," and "I am preoccupied with the desire to be thinner." Respondents are asked to choose a value on a 6-point Likert-type scale from Never = 0 to Always = 5, for a range of 0 to 35. The drive for thinness scale has been found to have psychometric properties for adolescents similar to those of adults (28). Fabian and Thompson (29) found that among female adolescents aged 10 to 15 years, drive for thinness significantly increased among postmenarcheal compared with premenarcheal females and that it was significantly correlated with lower body esteem.

Drive for thinness was assessed again in the follow-up study at ages 37 to 43 years. The internal reliability for the drive for thinness scale was good (Cronbach $\alpha = 0.86$).

RED was assessed only in the follow-up adult study. The RED scale (24) is an 11-item scale that focuses on aspects of compulsive eating (feeling out of control when eating, lack of satiety, and preoccupation with thoughts of food) and predicts weight gain. Higher RED scores are correlated with food craving (25). The Cronbach α was 0.92, demonstrating excellent internal reliability.

BMI

During the original 10 years of the study, annual health assessments were conducted, including height and weight measurements. In the follow-up study, all participants self-reported their weight and height on the baseline questionnaire. Participants received a home visit or visited the University of California, Berkeley to complete anthropometric measurements for those who lived within 60 miles of campus. A Tanita Professional digital scale and a Portable Adult/Infant Measuring Unit stadiometer from Perspective Enterprises were used for local participants. Participants who lived more than 60 miles from campus self-reported their weight measured on a scale and height measured using a paper copy of the QuickMedical Wall Growth Chart, which were mailed to the participants at home (30). Each weight was recorded in pounds to one decimal place, and height was recorded in centimeters to one decimal place. The measured weight and height compared with the self-reported weight and height based on the survey resulted in a near-perfect correlation for weight ($\rho = 0.99$), with a mean difference of 2.4 lb, and an almost-perfect correlation for BMI ($\rho = 0.98$), with a mean difference of 0.6 BMI units. BMI, calculated as weight in kilograms divided by height in meters squared, was based on measured weight and height for 503 participants (82%) and self-reported height and weight for 111 participants (18%).

Covariates

To avoid model overfitting and to limit covariates on the pathway, we limited the number of covariates included in the analytic model, using a priori assumptions and evidence from prior NGHS studies to guide our variable selection. Covariates for the final model included participant's race (White or Black), baseline household income ($0 to $9,999, $10,000 to $19,999, $20,000 to $39,999, ≥$40,000), baseline household educational attainment (high school or less, some college, college degree or more), and baseline BMI when the participant was aged 9 or 10. A missing indicator was used to account for 20 girls with missing household income data.

Statistical methods

We examined the trajectories of drive for thinness throughout adolescence. We first calculated the interquartile range at each time point. We then estimated the association between drive for thinness and BMI for each time point that it was measured to identify whether there was a linear relationship across time or if there was a critical period during adolescence that had the greatest association with adult BMI. As correlations were relatively constant across time points, we concluded that a summary adolescent drive for thinness score would be most relevant in predicting adult BMI, rather than drive for thinness at any particular age.

Structural equation modeling was used to examine the relationships between the variables of interest. We fit four equations simultaneously for the outcomes of adolescent drive for thinness, adult drive for thinness, adult RED, and adult BMI, using the data's inherent temporality to predict variable paths (Figure 1).

For example, adolescent drive for thinness was predicted by baseline BMI, baseline household income, and race. Adult drive for thinness and RED were both predicted by baseline BMI and adolescent drive for thinness. Adult BMI was predicted by all of the model covariates.

Obesity A Research Journal THE OBESITY SOCIETY –WILEY–

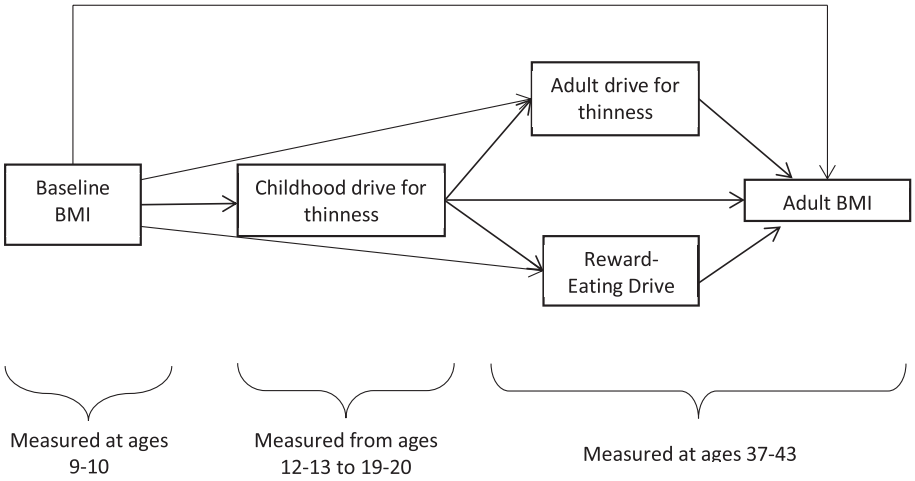

FIGURE 1 Conceptual model of adolescent drive for thinness and BMI in adulthood

Total, direct, and indirect effects were estimated. Model fit was evaluated using the comparative fit Index >0.90, root mean square error of approximation <0.08, and standardized root mean squared residual <0.05 (31). We report both unstandardized (B) and standardized (β) estimates and focus our interpretations on the unstandardized estimates.

For comparison, we also conducted a sensitivity analysis by omitting participants who met the criteria of being above the 95th percentile for age and sex at baseline ($n = 112$, 18%) to establish that any effect of adolescent drive for thinness on adult BMI was not due to obesity at baseline. Baseline obesity was determined using age- and gender-specific cut points for child overweight and obesity from the Centers for Disease Control and Prevention growth charts (32). All statistical tests were two-sided and statistical significance considered at $p < 0.05$. Statistical analyses were conducted using Stata Statistical Software/SE 14 (StataCorp LP).

RESULTS

Women were ages 37 to 43 at the time of the follow-up study. Table 1 shows the baseline characteristics and outcome variables. The average weight of the participants in the follow-up study was 190.0 lb (SD = 55.6, range: 103 to 444). Calculated mean BMI was 31.8 (9.4). Most women (79%) had children; on average, they had 2.3 (1.2) children, with a mean age of 10.1 years (5.7). The mean score for drive for thinness in the follow-up sample was 9.3 (7.3). The mean score for RED was 0.77 (0.77). Drive for thinness and RED were correlated at $\rho = 0.41$.

During adolescence, the mean score for drive for thinness was 4.8 (4.8), ranging from a minimum of 4.2 at age 15/16 to a peak of 4.9 at age 19/20 (Table 2).

A significant association between drive for thinness and adult BMI was found starting at age 10 and it remained significant until the end of adolescence, after adjusting for race, baseline household income, and baseline BMI. The cumulative (average) drive for thinness had the largest association with adult BMI ($\beta = 0.52$), compared with any single measurement in time, suggesting that increasing exposure may be the most relevant pathway to adult BMI. The proposed structural equation model fit the data well: comparative fit index was 0.962; root mean square error of approximation was 0.10; and standardized root mean squared residual was 0.04 (Figure 2).

Table 3 shows both unstandardized and standardized regression coefficients from the full structural equation model. From the unstandardized model, adolescent drive for thinness was positively predicted by baseline child BMI (B = 0.61, 95% CI: 0.53-0.70) independent of race and household income. Higher adolescent drive

TABLE 1 Characteristics of the NHLBI Growth and Health Study follow-up participants ($n = 614$)

	n	%
Baseline characteristics (age 9-10 years)		
Race		
White	314	51.1
Black	300	48.9
Parental education		
High school diploma or less	131	21.3
Any college	286	46.7
College graduate	196	32.0
Household income		
$0 to $9,999	112	19.0
$10,000 to $19,999	104	17.7
$20,000 to $39,999	172	29.3
≥$40,000	200	34.0
BMI (mean ± SD)	18.8 ± 3.7	
Follow-up characteristics (age 37-43 years)		
BMI (mean ± SD)	31.8 ± 9.4	
≥30 BMI	311	50.3
Reward-based eating drive (mean ± SD)	0.77 ± 7.7	
Drive for thinness (mean ± SD)	9.3 ± 7.3	

TABLE 2 Relation between adolescent drive for thinness and BMI in adulthood

	Drive for thinness score				Relation with BMI in adulthood[a]			
	n	Mean	Median	IQR	*r*	*β*	SE	*p*
Age 12 to 13 (Year 3)	569	4.5	2.0	7.0	0.28	0.47	0.07	0.0001
Age 14 to 15 (Year 5)	540	4.2	2.0	6.0	0.22	0.37	0.07	0.0001
Age 16 to 17 (Year 7)	520	4.7	2.0	8.0	0.18	0.29	0.07	0.0001
Age 18 to 19 (Year 9)	564	4.4	2.0	7.0	0.19	0.31	0.07	0.0001
Age 19 to 20 (Year 10)	596	4.9	3.0	9.0	0.21	0.34	0.06	0.0001
Adolescent average	615	4.8	2.8	8.0	0.27	0.52	0.08	0.0001

IQR, interquartile range.

[a]Adjusted for race, baseline household income, baseline household education, and baseline BMI.

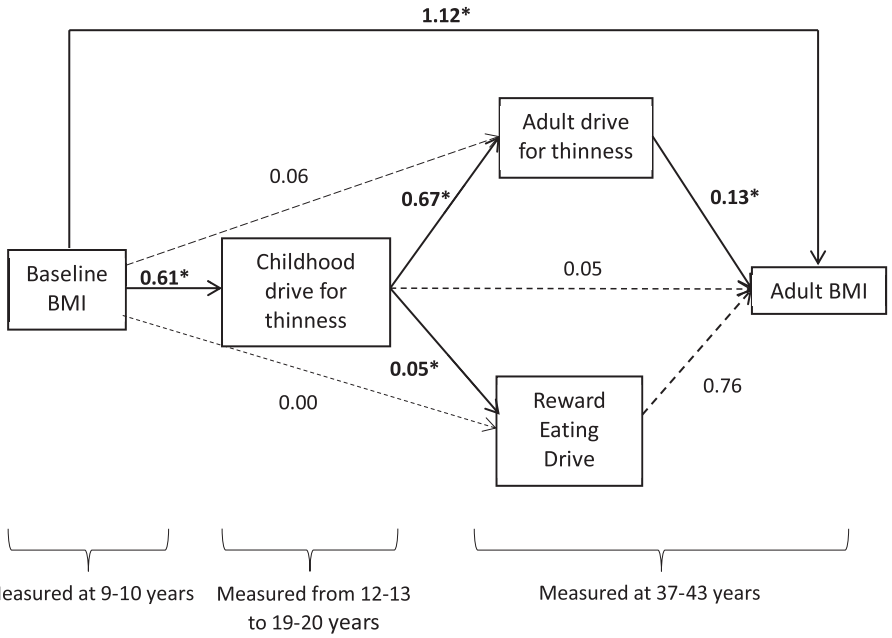

FIGURE 2 Results of structural equation modeling for the association between adolescent drive for thinness and BMI in adulthood. Coefficients are unstandardized estimates (B). Solid lines represent paths significant at $p < 0.05$. Dashed lines represent nonsignificant paths. **P* value significant at <0.05

for thinness significantly predicted higher adult drive for thinness (B = 0.67, 95%: CI 0.54-0.81) and adult RED (B = 0.05, 95% CI: 0.04-0.07) and was only associated with adult BMI through adult drive for thinness (B = 0.13, 95% CI: 0.03-0.22), controlling for race, maternal education, household income, and baseline BMI.

Childhood BMI was the largest predictor of adulthood BMI and it worked through direct means (B = 1.12, 95% CI: 0.98-1.34), as well as indirect means, through drive for thinness. Childhood BMI was not directly associated with either adult drive for thinness or adult RED. The total effect of adolescent drive for thinness on adult BMI (β = 0.17) was composed of a small direct effect (β = 0.05) and a larger indirect effect that was mediated through adult drive for thinness (β = 0.13) (data not shown). The results did not differ in sensitivity analyses excluding women who were considered to have obesity at baseline when they were 9 to 10 years old (*n* = 112).

DISCUSSION

Drive for thinness is a critical concept to understand as it is common and it can start early in life (29). As we found in an earlier study, this was a predictor of weight gain during childhood, up to 19 years old, the last time participants were followed during youth (27). Here we assess participants' eating behavior in their 40s. We found, as we hypothesized, that the cumulative drive for thinness during childhood led to a continued strong drive for thinness 20 years later in adulthood, as well as higher adulthood RED scores. Furthermore, adolescent drive for thinness, independent of childhood BMI, had a significant indirect effect on adult BMI that was mediated through adult drive for thinness (but not through RED). Drive for thinness is easy to assess and it is an important predictor of adult metabolic health. More than half of the participants in this sample had BMI ≥30

TABLE 3 Associations between averaged adolescent drive for thinness during adolescence and adult eating behavior and BMI using structural equation modeling in the NHLBI Growth and Health Study (n = 577)

	Unstandardized B	95% CI	Standardized β	95% CI
Adolescent drive for thinness				
Baseline BMI	0.61[a]	0.53, 0.70	0.50[a]	0.44, 0.56
Black race	−2.25[a]	−2.97, −1.53	−0.24[a]	−0.31, −0.17
Baseline household income: $10,000 to $19,999	−1.27[a]	−2.36, −0.18	−0.10[a]	−0.19, −0.02
Baseline household income: $20,000 to $39,999	−0.58	−1.60, 0.43	−0.06	−0.16, 0.04
Baseline household income: ≥$40,000	−0.66	−1.73, 0.40	−0.07	−0.18, 0.04
Baseline household education: some college	−0.19	−1.05, 0.67	−0.02	−0.11, 0.07
Baseline household education: college or more	−0.14	−1.13, −0.84	−0.01	−0.11, 008
Adulthood drive for thinness				
Baseline BMI	0.06	−0.10, 0.22	0.03	−0.05, −0.11
Adolescent drive for thinness	0.67[a]	0.54, 0.81	0.43[a]	0.35, 0.51
Reward−based eating drive				
Baseline BMI	0.00	−0.01, 0.02	0.02	−0.07, 0.11
Adolescent drive for thinness	0.05[a]	0.04, 0.07	0.32[a]	0.23, 0.40
Adulthood BMI				
Baseline BMI	1.16[a]	0.98, 1.34	0.48[a]	0.41, 0.55
Adolescent drive for thinness	0.05	−0.11, 0.21	0.02	−0.06, 0.10
Adulthood drive for thinness	0.13[a]	0.03, 0.22	0.10[a]	0.03, 0.18
Black race	4.13[a]	2.76, 5.51	0.22[a]	0.15, 0.30
Baseline household income: $10,000 to $19,999	−0.88	−2.86, 1.10	−0.04	−0.12, 0.05
Baseline household income: $20,000 to $39,999	−0.97	−2.82, 0.88	−0.05	−0.14, 0.04
Baseline household income: ≥$40,000	−1.32	−3.26, 0.62	−0.07	−0.17, 0.03
Baseline household education: some college	−1.52	−3.08, 0.04	−0.08	−0.17, 0.02
Baseline household education: college or more	−1.95[a]	−3.74, −0.16	−0.10[a]	−0.19, −0.01
Reward−based eating drive	0.76	−0.09, 1.34	0.06	−0.01, 0.14

Likelihood ratio test of model vs. saturated: $\chi^2 = 94.50$, $p < 0.0001$ for both models.

[a]$p < 0.05$.

and thus high risk for current or future obesity-related metabolic disease.

The results from this follow-up study also suggest that adolescent drive for thinness may be a major determinant of not only continued drive for thinness but also of reward-driven compulsive eating, such as preoccupation with food, feeling unsatiated, and feeling out of control. Surprisingly, RED was not cross-sectionally associated with adult BMI in this sample, whereas drive for thinness was. Perhaps the drive for thinness indicates behaviors that are more obesogenic over time. Drive for thinness may tap a more challenging set of behaviors that contribute to excess weight: highly restrictive eating, typically followed by overeating (as measured by the item asking about "struggling with weight"), compared with the behaviors captured by RED, which does not include any dieting behavior.

Roughly half of this sample of midlife women met the BMI cut-off for obesity. Interventions are needed to help address unhealthy eating behaviors early in life, especially such constructs as drive for thinness, which captures the desire to diet, to help women manage weight and reduce risk of weight-related chronic diseases. Such interventions may need to start before puberty and continue throughout adolescence, given that we saw consistent relationships between drive for thinness and BMI across childhood to adolescence and cumulative drive for thinness was related to outcomes at age ~40.

Drive for thinness is a potential target for early intervention because it is an important predictor of a variety of eating disorders as well as weight gain (33). Cognitive behavioral therapy has been used to address drive for thinness among young adults who score high on internalized weight bias (33). The implications of this study are also important to parents who can play a role in helping children develop a balanced or healthy restraint—not too high or low—early in life. Parental behaviors likely shape eating and dietary behaviors. For example, parents setting limits on and restricting young girls at age 5 from snacking were associated with higher scores on eating in the absence of hunger and BMI at age 7 compared with setting limits but not restricting or no limit setting (34). However, permitting child self-serve portions at mealtime may need to be coupled with guidance and rules, especially among young children with higher BMI (35). Interventions are also needed to help mothers manage weight and eating-related thoughts, emotions, and behaviors because these

maternal behaviors can increase child restrained eating behaviors (36).

These findings should be taken in light of the limitations to this study. A larger percentage of participants from the California site came from families of higher socioeconomic status compared with eligible nonparticipants. Therefore, this may bias the results away from or toward a stronger finding.

We are also aware that there are many studies that suggest dieting behavior is a strong predictor of weight gain, not drive for thinness per se (1). This study did not have a measure of dieting behavior, so we were not able to compare the two. However, results suggest that drive for thinness in adolescence is by no means benign. Drive for thinness is shaped during childhood, tracks through adulthood, and predicts greater weight status.

Another limitation is that we studied only females; however, dieting behaviors are much higher in girls than boys (37,38). Dieting has also been associated with mental health, which was not part of the current analysis (39). Future research is needed to examine the extent to which these findings apply to boys and identify appropriate interventions. Despite these weaknesses, our study had notable strengths, such as prospectively collecting information on drive for thinness multiple times throughout adolescence, measured weight and height, and the long time frame of the cohort.

CONCLUSION

Adolescent drive for thinness in girls has a long shadow on adulthood health. It tracks throughout adulthood, and it predicts compulsive eating behavior and greater weight mass independent of childhood weight. Given the associations with obesity-related diseases, drive for thinness is a serious early risk factor for early morbidity. Further understanding of these relationships may help to design obesity prevention programs to address drive for thinness in adolescents.O

ACKNOWLEDGMENTS

This study was supported by the Eunice Kennedy Shriver National Institute of Child Health and Human Development grant “Race, Stress and Dysregulated Eating: Maternal to Child Transmission of Obesity” (R01HD073568), the National Heart, Lung, and Blood Institute grant “Neighborhood Environments and Intergenerational Transmission of Cardiovascular Health” (R56HL141878), and the National Institute on Aging grants “Early Life Adversity, Cumulative Life Stress, Race, and Cellular Aging in Midlife Women and Offspring” (R56AG059677 and R01AG059677). We thank the Nutrition Policy Institute which provided consultation and support with historical study data. Most of all, we thank our incredible study participants.

CONFLICT OF INTEREST

The authors declared no conflict of interest.

AUTHOR CONTRIBUTIONS

BAL, LDR, PBC, and ESE conceived and carried out the study design and data collection; BAL and CWL conducted the data analysis; BAL, CWL, AJT, and ESE completed the data interpretation. All authors were involved in writing the paper and had final approval of the submitted and published versions.

ORCID

Barbara A. Laraia https://orcid.org/0000-0002-0493-2900

REFERENCES

1. Lowe MR, Doshi SD, Katterman SN, Feig EH. Dieting and restrained eating as prospective predictors of weight gain. *Front Psychol*. 2013;4:577. doi:10.3389/fpsyg.2013.00577
2. Stice E, Cameron RP, Killen JD, Hayward C, Taylor CB. Naturalistic weight-reduction efforts prospectively predict growth in relative weight and onset of obesity among female adolescents. *J Consult Clin Psychol*. 1999;67(6):967-974.
3. van Strien T, Herman CP, Verheijden MW. Dietary restraint and body mass change. A 3-year follow up study in a representative Dutch sample. *Appetite*. 2014;76:44-49.
4. Hill AJ. Does dieting make you fat? *Br J Nutr*. 2004;92(supp 1): S15-S18.
5. Haines J, Neumark-Sztainer D. Prevention of obesity and eating disorders: a consideration of shared risk factors. *Health Educ Res*. 2006;21(6):770-782.
6. Garner DM, Olmstead MP, Polivy J. Development and validation of a multidimensional eating disorder inventory for anorexia-nervosa and bulimia. *Int J Eat Disord*. 1983;2(2):15-34.
7. Striegel-Moore RH, Schreiber GB, Pike KM, Wilfley DE, Rodin J. Drive for thinness in black and white preadolescent girls. *Int J Eat Disord*. 1995;18(1):59–69.
8. Wiederman MW, Pryor TL. Body dissatisfaction, bulimia, and depression among women: the mediating role of drive for thinness. *Int J Eat Disord*. 2000;27(1):90-95.
9. Green MW, Rogers PJ, Elliman NA, Gatenby SJ. Impairment of cognitive performance associated with dieting and high levels of dietary restraint. *Physiol Behav*. 1994;55(3):447-452.
10. Green MW, Rogers PJ. Impaired cognitive functioning during spontaneous dieting. *Psychol Med*. 1995;25(5):1003-1010.
11. Hart KE, Chiovari P. Inhibition of eating behavior: negative cognitive effects of dieting. *J Clin Psychol*. 1998;54(4):427-430.
12. Bellisle F, Dalix AM. Cognitive restraint can be offset by distraction, leading to increased meal intake in women. *Am J Clin Nutr*. 2001;74(2):197-200.
13. Baumeister RF, Bratslavsky E, Muraven M, Tice DM. Ego depletion: Is the active self a limited resource? *J Pers Soc Psychol*. 1998;74(5):1252-1265.
14. Hagger MS, Panetta G, Leung CM, et al. Chronic inhibition, self-control and eating behavior: test of a 'resource depletion' model. *PLoS One*. 2013;8(10):e76888. doi:10.1371/journal.pone.0076888
15. Ward A, Mann T. Don't mind if I do: disinhibited eating under cognitive load. *J Pers Soc Psychol*. 2000;78(4):753-763.
16. Westenhoefer J, Broeckmann P, Munch AK, Pudel V. Cognitive control of eating behaviour and the disinhibition effect. *Appetite*. 1994;23(1):27-41.
17. Gibson EL. Emotional influences on food choice: sensory, physiological and psychological pathways. *Physiol Behav*. 2006;89(1):53-61.
18. Greeno CG, Wing RR. Stress-induced eating. *Psychol Bull*. 1994;115(3):444-464.

19. Epel EE, Tomiyama AJ, Dallman MF. Stress and reward neural networks, eating, and obesity. In: Brownell KD, Gold MS, eds. *Food and Addiction: A Comprehensive Handbook*. Oxford University Press; 2012:266-272. doi:10.1093/med:psych/9780199738168.003.0040
20. Arnsten AF. Stress signalling pathways that impair prefrontal cortex structure and function. *Nat Rev Neurosci*. 2009;10(6):410-422.
21. Tomiyama AJ, Mann T, Vinas D, Hunger JM, Dejager J, Taylor SE. Low calorie dieting increases cortisol. *Psychosom Med*. 2010;72(4):357-364.
22. Tomiyama AJ. Stress and obesity. *Annu Rev Psychol*. 2019;70:703-718.
23. Adam TC, Epel ES. Stress, eating and the reward system. *Physiol Behav*. 2007;91(4):449-458.
24. Epel ES, Tomiyama AJ, Mason AE, et al. The reward-based eating drive scale: a self-report index of reward-based eating. *PLoS One*. 2014;9(6):e101350. doi:10.1371/journal.pone.0101350
25. Mason AE, Epel ES, Aschbacher K, et al. Reduced reward-driven eating accounts for the impact of a mindfulness-based diet and exercise intervention on weight loss: data from the SHINE randomized controlled trial. *Appetite*. 2016;100:86-93.
26. Obesity and cardiovascular disease risk factors in black and white girls: the NHLBI Growth and Health Study. *Am J Public Health*. 1992;82:1613-1620. doi:10.2105/ajph.82.12.1613
27. Rehkopf DH, Laraia BA, Segal M, Braithwaite D, Epel E. The relative importance of predictors of body mass index change, overweight and obesity in adolescent girls. *Int J Pediatr Obes*. 2011;6(2-2):e233-e242. doi:10.3109/17477166.2010.545410
28. Williams RL, Schaefer CA, Shisslak CM, Gronwaldt VH, Comerci GD. Eating attitudes and behaviors in adolescent women - discrimination of normals, dieters, and suspected bulimics using the Eating Attitudes Test and Eating Disorder Inventory. *Int J Eat Disord*. 1986;5(5):879-894.
29. Fabian LJ, Thompson JK. Body-image and eating disturbance in young females. *Int J Eat Disord*. 1989;8(1):63-74.
30. Wardle J, Carnell S, Haworth CM, Plomin R. Evidence for a strong genetic influence on childhood adiposity despite the force of the obesogenic environment. *Am J Clin Nutr*. 2008;87(2):398-404.
31. Hooper D, Coughlan J, Mullen M. Evaluating model fit: a synthesis of the structural equation modelling literature. Presented at: 7th European Conference on Research Methodology for Business and Management Studies, Regent's College, London, United Kingdom; 2008. Accessed May 3, 2021. https://arrow.tudublin.ie/cgi/viewcontent.cgi?article=1046&context=buschmancon
32. Kuczmarski RJ, Ogden CL, Guo SS, et al. 2000 CDC growth charts for the United States: methods and development. Vital and Health Statistics, series 11, no. 246. National Center for Health Statistics; 2002.
33. Marshall RD, Latner JD, Masuda A. Internalized weight bias and disordered eating: the mediating role of body image avoidance and drive for thinness. *Front Psychol*. 2020;10:2999. doi:10.3389/fpsyg.2019.02999
34. Rollins BY, Loken E, Savage JS, Birch LL. Maternal controlling feeding practices and girls' inhibitory control interact to predict changes in BMI and eating in the absence of hunger from 5 to 7 y. *Am J Clin Nutr*. 2014;99(1):249-257.
35. Savage JS, Haisfield L, Fisher JO, Marini M, Birch LL. Do children eat less at meals when allowed to serve themselves? *Am J Clin Nutr*. 2012;96(1):36-43.
36. Francis LA, Birch LL. Maternal influences on daughters' restrained eating behavior. *Health Psychol*. 2005;24(6):548-554.
37. French SA, Story M, Downes B, Resnick MD, Blum RW. Frequent dieting among adolescents: psychosocial and health behavior correlates. *Am J Public Health*. 1995;85(5):695-701.
38. Neumark-Sztainer D, Paxton SJ, Hannan PJ, Haines J, Story M. Does body satisfaction matter? Five-year longitudinal associations between body satisfaction and health behaviors in adolescent females and males. *J Adolesc Health*. 2006;39(2):244-251.
39. Crow S, Eisenberg ME, Story M, Neumark-Sztainer D. Psychosocial and behavioral correlates of dieting among overweight and nonoverweight adolescents. *J Adolesc Health*. 2006;38(5):569-574.

How to cite this article: Laraia BA, Leung CW, Tomiyama AJ, Ritchie LD, Crawford PB, Epel ES. Drive for thinness in adolescents predicts greater adult BMI in the Growth and Health Study cohort over 20 years. *Obesity (Silver Spring)*. 2021;29:2126–2133. https://doi.org/10.1002/oby.23285