Abstract

Weight suppression (WS) refers to the discrepancy between highest adult weight and current weight, and has been examined as a key construct related both to eating pathology and weight management. Despite increasing interest in WS as it relates to eating disorders and obesity, findings regarding the clinical implications of WS are often conflicting. Depending on the population examined, WS may be related to both adaptive and maladaptive outcomes. Moreover, results regarding the predictive utility of WS within clinical samples have been inconsistent. The current paper aims to provide a narrative review of existing investigation related to WS, highlight gaps in the field's understanding of this construct, and provide recommendations for future study.

Keywords: weight suppression, eating disorders, weight management, eating pathology, anorexia nervosa, bulimia nervosa

Weight Suppression and its Relation to Eating Disorder and Weight Outcomes: A Narrative Review

Weight suppression (WS) refers to the calculated difference between an individual's highest weight since reaching adulthood (outside of pregnancy) and current weight (Lowe, 1984, 1993). Originally introduced in the 1980's (e.g., Lowe, 1984), interest in WS and its links to eating pathology has burgeoned over the past decade, under the theoretical proposition that maintaining a body weight below a point that is most natural for one's body will create metabolic and psychological resistance and pressure to return to this natural, higher weight. Despite increased interest in WS in eating disorder and weight management research, along with a growing emphasis on metabolic and biological mechanisms underlying the development of eating pathology and obesity, no reviews to date provide a comprehensive summary of existing research on the construct of WS. The current review aims to provide a description of existing findings on WS across the spectrum of weight- and eating-related behaviors. To do so, we first describe findings regarding the role of WS in clinical and non-clinical populations, focusing on outcomes across samples of individuals with diagnoses of bulimia nervosa (BN), anorexia nervosa (AN), and multi-diagnostic samples, including individuals with binge eating disorder (BED). Next, we consider existing work on WS within college and community samples. Following review of existing data probing associations between WS and relevant eating and weight-related outcomes, we highlight controversies and several limitations of the current assessment of WS, as well as provide commentary on important areas for future study.

Weight Suppression and Associated Psychological Outcomes

Weight suppression in clinical samples. The majority of research on WS has evaluated its association with eating disorder (ED) -related clinical phenomena. A recent systematic review

of studies examining WS as a predictor of treatment in EDs determined that WS was generally associated with weight gain following treatment, but not with other treatment outcomes (e.g., treatment completion; symptom abstinence) (Jenkins, Lebow, & Rienecke, 2018). A more detailed investigation of the extant literature is warranted, as the hypothesis that WS negatively impacts treatment outcome is not consistently supported. In the following sections, we explore existing work linking WS with clinical symptoms, weight trajectory, and treatment response within BN and AN, and then consider some initial work investigating WS within mixed diagnostic samples and/or BED.

Bulimia nervosa.

ED pathology. Overall, research in samples of individuals with BN suggests that greater WS is associated with increased eating pathology. Specifically, cross-sectional studies of individuals seeking treatment for BN have indicated that elevated WS is associated with greater binge eating and purging frequency at baseline, prior to beginning treatment (Butryn, Juarascio, & Lowe, 2011; Lowe, Thomas, Safer, & Butryn, 2007). These findings support hypotheses that maintenance of bulimic symptoms may be influenced by the desire to avoid returning to premorbid body weights (e.g., Garner & Fairburn, 1988), as the behaviors needed to maintain a suppressed weight (e.g., caloric restriction) can also increase risk for binge eating and subsequent compensatory behaviors (Herman & Polivy, 1975; Lowe, 1986; Stice, Davis, Miller, & Marti, 2008).

Because mixed findings might indicate that there are other variables or individual difference factors that impact relations between WS and BN symptomology, some investigations have examined factors hypothesized to moderate this association. Butryn, Juarascio and Lowe (2011) tested whether the interaction between current body mass index (BMI) and WS at

pretreatment accounted for binge eating and purging frequency in women with BN, prior to beginning intensive outpatient cognitive behavioral therapy (CBT). This interaction was not significant for purging, as elevated WS demonstrated a significant, positive main effect on purging frequency regardless of pretreatment BMI; however, the WS x BMI interaction term predicted frequency of binge eating, such that individuals with low BMI and high WS reported significantly greater binge eating frequency than the other three groups (i.e., those with higher BMI, in combination with either low or high WS). While this finding suggests that individuals with lower body mass at treatment entry and greater suppressed weight may be at risk for binge eating, other research has failed to replicate this finding (Dawkins Watson, Egan, & Kane, 2013).

In addition to BMI at treatment onset, timing of WS assessment may present an important variable to consider, as some research suggests that differences in timing of highest weight attainment (premorbid vs. postmorbid) may differentially relate to age of onset and average duration of BN pathology (Shaw et al., 2012). Moreover, one study of adolescents with BN found that levels of *greatest* WS (difference between highest ever and lowest ever BMI z-score [BMIz]), rather than current WS, moderated the effect of BMIz on symptom outcomes, such that youth with higher levels of greatest WS and high current BMIz engaged in more frequency of binge eating than youth with low current BMIz (Accurso, Lebow, Murray, Kass, & Le Grange, 2016). Tests of additional moderators, including dietary restraint, difference between highest and lowest body weights, parental history of overweight, and patients' childhood body shape, have not yielded significant moderation effects on WS and BN symptoms (Dawkins et al., 2013; Lowe et al., 2007).

Treatment response. In a meta-analysis examining predictors of treatment outcome across studies of individuals with BN or BED, greater WS demonstrated a small effect on increased

treatment dropout (Vall & Wade, 2015). It should be noted that this effect was calculated from 5 studies, one of which included participants with BED (vs. only participants with BN), and did not include studies of patients with AN. Prospective research in treatment-seeking adults with BN also demonstrated that elevated pretreatment WS predicts greater bulimic symptom maintenance over the course of cognitive behavioral interventions in both adult outpatient (Butryn, Lowe, Safer, & Agras, 2006; Lowe et al., 2011; Herzog et al., 2010) and inpatient samples (Lowe, Davis, Lucks, Annunziato, & Butryn, 2006). Despite some evidence linking WS with BN symptom maintenance, results from several investigations do not support the role of WS in the maintenance of BN symptoms following treatment. For instance, although Butryn et al. (2006) determined that WS predicted treatment completion and symptom outcome in a large sample of women receiving outpatient treatment for BN, Carter, McIntosh, Joyce, and Bulik (2008) were unable to replicate these findings. A retrospective review of treatment outcome data among patients referred for CBT for BN over an eight-year period failed to find evidence of significant associations between WS and bulimic symptom outcomes, including abstinence from and frequency of binge eating and purging behaviors (Dawkins et al., 2013). Such discrepancies suggest additional research should strive to clarify whether and how WS may relate to bulimic symptomatology and maintenance over the course of treatment, and longitudinally thereafter, among individuals with BN.

Weight trajectory. Within an inpatient sample of females with BN, higher levels of WS predicted greater weight gain over treatment (Lowe et al., 2006). These findings were replicated among women with BN receiving outpatient CBT, where WS was positively associated with weight change (i.e., weight gain ≥5 kg over treatment) (Carter et al., 2008). Taken together, these studies provide preliminary evidence that WS predicts short-term weight gain in BN treatment.

Of note, although a study among inpatients with BN indicated WS was associated with weight gain over the course of treatment, with slightly more weight gain in patients with higher WS, these effects were not significant (Hessler et al., 2017). Investigating whether any significant weight trend might continue post-treatment, Herzog et al. (2010) found that among treatment-seeking women with BN, WS predicted faster weight gain over five years. Overall, most evidence supports an association between higher pretreatment/baseline WS and weight gain over the course of both inpatient and outpatient CBT treatment, an effect that may persist over time. Given that weight gain both during and after treatment might inspire dieting behavior that maintains BN or contribute to relapse, assessment of WS and specific weight trajectories in individuals with BN may have important treatment implications (Juarascio, Lantz, Muratore, & Lowe, 2017).

Anorexia nervosa.

Initial investigation of WS primarily focused on its presentation in individuals with BN; however, more recent research has established that WS may have similar utility in accounting for ED symptomatology among individuals with AN. Secondary lines of work have tested the utility of WS in accounting for treatment response, as well as long-term weight trajectory.

ED pathology. To date, several studies have evaluated associations between WS and eating disorder cognitions and behaviors within AN. First, within a sample of individuals receiving residential treatment for AN, WS demonstrated significant, cross-sectional correlations with several measures of ED symptoms, including shape concern, eating concern, restraint, global eating pathology, depression, bulimia, and drive for thinness (Berner, Shaw, Witt, & Lowe, 2013). Additionally, prospective analyses within this same sample indicated that an interaction between WS and BMI accounted for significant variance in symptom endorsement at

discharge; those individuals with high WS and low BMI at intake reported fewer symptoms at discharge, whereas women with high WS and high BMIs at intake reported higher symptomatology at discharge (Berner et al., 2013). Bodell, Racine and Wildes (2016) found that in a sample of women with AN, the interaction between WS and BMI predicted increases in ED symptoms following discharge from intensive treatment (i.e., inpatient only, outpatient day hospital, or a combination).

Related to WS, several studies have found that considering other aspects of an individual's weight history— specifically greatest WS, or his or her highest past weight— may account for variability in the onset and trajectory of AN symptoms. Within a sample of individuals with adolescent-onset AN and matched controls, Berkowitz et al. (2016) found that individuals with AN had significantly higher BMIs than healthy controls during infancy (as measured by ponderal index) and childhood. In addition to considering childhood and adolescent weight status, considering highest past weight may be important in determining symptom severity. For example, Swenne, Parling and Ros (2017) found that in a large sample of adolescents with restrictive EDs, degree and speed of weight loss— in addition to current weight status— accounted for significant variability in medical indices of symptom severity (e.g., cardiac function), even if individuals were currently in the normal BMI range. In a similar manner, Berner, Feig, Witt and Lowe (2017) investigated the association between weight history and amenorrhea in a sample of individuals seeking residential treatment for AN, and found that greater WS related to fewer menstrual periods in the four months prior to admission to treatment. Moreover, within this sample, higher past BMI related to loss of menses at a higher BMI, and higher past BMI related positively to BMI at menses resumption. Overall, these studies suggest

that considering an individual's weight history, in addition to current weight, may be important in accounting for symptom onset and severity in the context of AN.

Treatment response. In addition to symptom severity, other studies have evaluated links between WS and treatment outcome and dropout. Of note, understanding links between WS and treatment outcome are complex, given established links between WS and weight gain (i.e., a common target within AN treatment) *and* severity of psychopathology within bulimic-spectrum disorders. Accordingly, existing findings within study of AN have been somewhat mixed. The first study examining WS in AN suggested that higher levels of WS predicted greater total and faster rates of weight gain over the course of intensive inpatient behavioral treatment for AN, as well as increased likelihood of engaging in binge eating and purging behaviors at discharge (Wildes & Marcus, 2012). A recent study replicated results from Wildes and Marcus (2012), linking WS with greater speed of weight gain in an outpatient sample, demonstrating that WS predicted the total amount of weight gain, as well as rate of weight gain, over the course of treatment (Carter et al., 2015). Most recently, Swenne et al. (2017) found that WS accounted for significant variance in 12-month outcomes for family based treatment; specifically, lower WS was related to *better* outcomes, as defined by the absence of clinically-significant ED psychopathology and fulfillment of diagnostic criteria.

Other work has found no direct links between WS and treatment outcome, and thus has turned to evaluate other variables that may interact with WS to more precisely characterize WS as a prognostic indicator. Berner et al. (2013) found that while WS related to BMI at discharge and speed of weight gain throughout treatment, it did not relate to other ED symptoms (e.g., cognitive symptoms, bulimic-type behaviors) at discharge. Instead, the interaction between WS and BMI was a significant predictor of outcome among those with higher WS, such that those

individuals with higher BMI and higher WS at intake had higher symptoms at discharge, whereas those with lower BMI and higher WS at intake had better outcomes, reflected in lower symptoms at discharge. On the other hand, this same study failed to replicate findings linking WS with binge eating and purging at discharge.

Longer-term weight trajectory. Although there are only a few existing studies evaluating the links between WS and longer-term weight trajectory in samples of individuals with AN, findings from existing work consistently align with results from treatment research suggesting that WS is associated with greater weight gain over time. For example, in a community sample of individuals with AN, Witt et al. (2014) found that baseline WS was positively associated with BMI at 6- and 10-year follow-up; this effect was strongest among those with lower BMIs at baseline. Moreover, baseline WS was positively associated with BMI at 18-year follow-up. Bodell et al. (2016) found that in a sample of women with AN who discharged from intensive treatment, WS at discharge predicted change in BMI at 3-, 6-, and 12-month follow-up.

Mixed clinical samples.

Expanding from research conducted in samples of single diagnoses (e.g., BN-only, AN-only), other research has examined WS among samples of individuals with varying ED diagnoses. As in study samples with a single ED diagnosis, multi-diagnostic studies have included outcome variables that examine ED symptoms, treatment response, and weight trajectory. To date, only one study has explicitly studied WS in a sample of individuals with a primary diagnosis of BED (Zunker et al., 2011); as such, it is critical that future work include studies of WS specifically amongst individuals with a primary diagnosis of BED.

ED pathology. One investigation of interactions among WS, BMI, and DSM-IV-defined ED diagnostic groups (i.e., AN, BN, BED, EDNOS) found that after controlling for ED

diagnosis and current BMI, WS demonstrated a significant association with weight and shape concerns, exercise and restrictive behaviors, and weight control medication use; WS was not directly related to binge eating or vomiting (Lavender et al., 2015). Notably, the relation between WS and ED pathology in this study did not differ by diagnosis. Significant interaction effects included WS x BMI, but only in relation to weight and shape concerns, such that the positive association between WS and weight and shape concern was strongest at lower BMI. There were also stronger positive associations between BMI and weight control medication use among those with BN versus EDNOS, and stronger, positive associations between BMI and both binge eating/vomiting and exercise/restrictive eating behaviors among those with AN versus EDNOS. Given the varied presentation of individuals with EDNOS, it is difficult to draw specific conclusions from these interaction effects, but evidence supports a strong main effect of WS on various ED psychopathology dimensions (with the exception of binge eating/vomiting).

In a large, multisite sample of individuals with either BN or BED, individuals were assessed following various CBT interventions (i.e., group CBT for BED; individual CBT for BN, either face to face or via telemedicine). Results indicated that of the individuals who completed treatment, WS failed to predict abstinence from binge eating for BN and BED participants, and failed to predict abstinence from purging or binge eating and purging combined for those with BN (Zunker et al., 2011).

Only one investigation of WS and ED pathology in a mixed-diagnostic sample (in this case, individuals diagnosed with BN or BED) has studied WS as an outcome variable. Cook et al. (2015) found that both exercise frequency and BN/BED diagnoses were associated with WS. Additionally, exercise frequency moderated the relation between diagnosis and WS, such that WS was higher in BN than in BED among those who reported lower exercise frequency. In

contrast, no notable difference in WS was identified among those reporting higher exercise frequency in patients with BN and BED. Findings from this study suggest a potential subgroup of individuals with BED who may engage in more frequent exercise to maintain a suppressed body weight. As WS is the outcome in this study, it is difficult to draw conclusions about the relation between WS and maladaptive exercise behavior in individuals with BED or BN across other literature.

Treatment response. In the multisite trial conducted by Zunker et al. (2011) described above, for both individuals with BN or BED, WS did not predict treatment completion (Zunker et al., 2011). To date, no other studies have examined treatment response explicitly with a transdiagnostic ED sample.

Weight trajectory. Recent work with a combined sample of patients with AN and BN demonstrated a significant positive association between baseline WS and weight gain at the end of treatment (Miotto, Ciappini, Favaro, Santanastapso & Gallichio, 2017). In contrast, in the multisite trial conducted by Zunker et al. (2011) described above, WS failed to predict weight change during treatment both for participants with BN, as well as for those with BED. In sum, studies of the relation between WS and ED symptoms, treatment outcome and weight trajectory are minimal in transdiagnostic samples.

Weight suppression in non-treatment-seeking samples.

While initial interest in WS was prompted by findings within clinical ED samples, WS has also garnered interest as a correlate as well as predictor of eating and weight outcomes in non-treatment-seeking samples. Elevated WS has been reported to be suggestive of a history of consistent caloric restriction (Klem, Wing, McGuire, Seagle, & Hill, 1998). While the long-term goal of dieting is typically sustained weight loss, WS may have both adaptive and maladaptive

associations with various factors related to weight management in non-clinical populations. Individuals high in WS report higher levels of dietary restraint (Lowe, 1984), increased levels of physical activity (Cook et al., 2015; French & Jeffrey, 1997), and reduced food consumption following a laboratory preload (Lowe & Kleifield, 1988) than their counterparts with low WS, all of which may be adaptive for health outcomes and weight management. However, WS has also been identified as a predictor of negative eating and weight outcomes in healthy populations, including excess weight gain (Stice et al., 2011) and increased incidence of loss of control (LOC) and binge eating behavior (Van son, van der Meer, & Van Furth, 2013). Below, we explore links between WS and weight-relevant outcomes, including eating pathology and weight trajectory, in college and community-based samples.

Weight suppression in college samples.

ED pathology. Given typical age of ED onset and other psychosocial and developmental factors, undergraduate students are at increased risk for the development of eating pathology (Eisenberg, Nickett, Roeder & Kirz, 2011). Despite this vulnerability, few studies to date have directly tested associations between WS and eating behaviors in college samples. Early investigations of in-lab behavior were mixed in their findings. On one hand, Lowe and Kleinfield (1988) found that within a small sample of healthy, female undergraduates, individuals identified as weight suppressors reported higher restraint, and ate significantly less food following a milkshake preload. On the other hand, another investigation found no significant links between WS, cognitive restraint, and in-lab eating behaviors (Morgan & Jeffrey, 1999).

More recent research that has gauged associations between WS and self-reported eating pathology has also yielded conflicting results. Specifically, one examination of the relation between WS and disordered eating symptoms among college students determined that WS did

not predict increases in BN symptoms at the end of the first year of college (Stice et al., 2011). In another sample, WS was cross-sectionally related to dietary restraint and purging behaviors, but not to LOC eating; additionally, these effects may differentially impact eating behavior relative to gender (Burnette, Simpson, & Mazzeo, 2017). In this study, men with higher WS engaged in more frequent purging behaviors (i.e., vomiting and laxative use). These findings suggest that undergraduate men who have history of overweight or significant weight loss may be at increased risk for the development of more extreme weight control behaviors.

Weight trajectory. Greater WS is associated with increased weight gain over time among undergraduate student samples (Lowe et al., 2006; Lowe et al., 2007). Few studies have tested potential mechanisms of links between WS and weight gain within college samples. In a notable exception, one study found that increases in BMI did not appear to be affected by biological mechanisms such as resting metabolic rate, or total energy expenditure (Stice et al., 2011). Findings from this investigation suggest that psychological variables, such as perceived deprivation, and behavioral variables related to eating and exercise must be investigated further to examine the association between WS and weight gain.

Overall, while the work evaluating links between WS and weight trajectory within college samples remains limited in scope, findings from existing research tentatively support associations between WS and weight gain, and indicate that biological mechanisms may not wholly account for the nature of this relation. On the other hand, limited work evaluating links between WS and eating pathology, significant variability in methodological approaches, and mixed findings precludes any conclusions regarding relations between WS and eating behaviors in college students at this time.

Weight suppression in community-based samples.

In the following sections, we include studies of WS with associated eating pathology and weight outcomes in non-clinical, community-based samples.

ED pathology. Within a generally healthy community sample, weight suppressors reported higher physical activity levels and low-fat eating behaviors as compared to non-suppressors, suggesting that WS might predict successful weight loss maintenance (French & Jeffrey, 1997). More recent work examining associations between WS and disordered eating within these samples has identified significant positive links between WS and bulimic symptoms (Mitchell et al., 2011), even when controlling for dieting behavior (Keel & Heatherton, 2010). For example, one study found positive associations between WS, binge eating, and LOC eating behaviors, though effects were nonsignificant after accounting for dieting (Van son et al. 2013).

Drawing from a large population-based sample of female adult twin dyads, Mitchell et al. (2011) found that restraint and dieting during childhood related to increased WS across the sample. In addition, body dissatisfaction, restraint, age, and drive for thinness positively related to WS among non-binge eaters whereas only restraint, disinhibition, and dieting during childhood were positively linked to WS among individuals who endorsed binge eating and LOC eating. Noting these differences, the authors concluded that WS might differentially relate to various factors, depending on ED vulnerability status.

Further, prospective investigation of links between WS and bulimic symptoms have been examined in a community sample across the span of 20 years (Bodell, Brown & Keel, 2017; Keel & Heatherton, 2010). The first part of this study, conducted across two population-based cohorts (1982; 1992), suggested that WS was a significant predictor of changes in bulimic behaviors at 10-year follow-up, regardless of reported dieting frequency (Keel & Heatherton, 2010). Patterns in the data suggest that greater WS at baseline assessment predicted maintenance

of EDI bulimic symptoms, more gradual decrease in symptoms over time, or increases in symptoms from baseline to follow-up. Follow-up evaluation of diagnostic status at baseline determined that WS was the only significant predictor for bulimic syndrome onset at the 10-year follow-up for individuals who did not originally endorse bulimic symptoms at baseline, suggesting that WS may be a core risk factor for individuals at risk for BN eating pathology. Further examination of this study sample in 2012 revealed that higher WS at baseline predicted increased bulimic symptoms at 20-year follow-up, controlling for baseline bulimic symptoms, BMI, and drive for thinness. Elevated drive for thinness at 10-year follow-up mediated this effect (Bodell, Brown, & Keel, 2017). This finding seems to suggest that preoccupation with thinness may serve to perpetuate bulimic symptoms for those who report higher WS.

Weight trajectory. Prospective study of obese individuals attempting to lose weight has indicated difficulty in sustaining a suppressed weight for an extended period of time (Dombrowski, Knittle, Avenell, Araujo-Soares, & Sniehotta, 2014). Recent research indicates that strong neurobehavioral (Appelhans, French, Pagoto & Sherwood, 2016) and biological (Greenway, 2015; Fothergill et al., 2016) drives towards restoration of a previously higher weight may interfere with successful weight management. Notably, research that examined weight history and subsequent weight gain found that history of WS itself did not independently increase risk for longitudinal weight gain for men, and only marginally so for women (Wye, Dubin, Blair & Pietro, 2007). Additional evidence from trials investigating weight cycling show that weight cycling does not induce greater weight gain when compared to non-cycling individuals (Mason et al., 2013). Further, individuals with more previous weight loss attempts and larger previous weight losses actually perform better in self-help weight management (Latner & Ciao, 2014). Altogether, WS does not seem to independently produce weight regain

above that which would have been gained without rebound from loss related to a current diet (Greenway, 2015; Lowe, 2015).

Examinations of WS and associated maladaptive outcomes within community populations are limited in number, but demonstrate consistent links with negative outcomes associated with maintenance of ED symptoms (e.g., Keel & Heatherton, 2010). In particular, symptoms of binge eating, LOC eating, and drive for thinness may be more problematic in maintaining ED pathology over time, specifically symptoms related to BN (i.e., binge eating, and compensatory purging behaviors). Recent study of clinical impairment specifically related to bulimic symptoms in a community sample found that WS was significantly associated with clinical impairment (Hagan, Clark, & Forbush, 2017). Although WS did not demonstrate incremental validity above and beyond other factors tested (e.g., frequency of engagement in maladaptive compensatory behavior), it did demonstrate a medium effect size in independently predicting clinical impairment. In sum, preliminary findings from study of WS in community samples indicate value in considering the clinical impact WS might have in maintenance of symptoms and impairment associated with BN-related pathology.

Current Controversies and Future Directions

Future research is needed to clarify associations between WS and eating and weight-related outcomes, particularly in certain understudied populations (e.g., clinical samples of individuals with BED). Future work should pursue a consistent definition, operationalization, and calculation of WS. In the following sections, we outline important controversies and issues that should be the focus of study moving forward.

Method of calculation. To date, there has been marked inconsistency in the operationalization of WS used within existing literature, with some studies calculating WS by

subtracting an individual's current weight from his or her highest ever adult, non-pregnancy weight, in pounds or kilograms (e.g., Butryn et al., 2006; Herzog et al., 2010; Stice et al., 2011), others using BMI units (e.g., Berner et al., 2013; Witt et al., 2014), some choosing to dichotomize samples into "high" or "low" WS (e.g., Carter et al., 2008; Butryn et al., 2011; Zunker et al., 2011), and other studies using alternative methods of gauging weight fluctuation that consider lowest adult weight (e.g., Carter et al., 2008; Witt et al., 2014). Notably, inconsistent operationalization of WS may contribute to mixed findings across samples and limit researchers' ability to make comparisons across investigations, as different calculations would likely generate differing groups of individuals considered "weight suppressed." For instance, a study calculating WS using weight only would treat two cases with BN that have lost 10 pounds from their highest weight (e.g., 150) in a similar manner in statistical analyses, even if those women had significantly different heights (e.g., 58 inches, and one 67 inches, respectively). On the other hand, for a study using changes in BMI units, these women would be considered differentially weight suppressed, as the 58-inch woman would have a WS value of 2.0 and the 67-inch woman would have a WS value of 1.4. Although lack of clarity surrounding the precise mechanism through which WS relates to symptoms precludes determining the "best" way to classify individuals along a continuum of weight suppression, it is clear that varying calculations of the construct likely limit the ability to compare results of statistical tests.

Moreover, the majority of existing WS calculations make use of a change score and are collected in a self-report format, both of which may detract from the validity and reliability of the construct (Schaumberg, Anderson, Reilly, Gorrell, & Anderson, 2016). Preliminary work comparing methods of WS calculation determined that it is advisable to consider the impact of highest past weight within calculations (Schaumberg et al., 2016). Based on this

recommendation, in one recent study, current weight was subtracted from highest lifetime weight; this weight loss was then divided by lifetime highest weight to determine percentage of weight loss (Forney et al., 2017). Future work must better explore the psychometric properties of differing calculations of WS, and it is recommended that researchers operationalize the construct in a manner that maximizes reliability and validity, while facilitating consistency within the literature.

Developmental sensitivity of the weight suppression construct. Early conceptualizations of WS operationalized the construct as considering an individual's highest *adult* weight. However, some recent existing investigations of WS—particularly those within clinical samples—have included participants who are below the age of 18 and/or ask participants to report on highest post-puberty weight. Recent work on the relation between EDs and body weight throughout childhood reveals a complex developmental phenomenon. For example, genome-wide association studies find positive genetic correlations between risk for AN and lower BMI (Duncan et al., 2017). Additionally, in contrast to prior work showing that individuals with AN demonstrated higher childhood weight (Berkowitz et al., 2016), a recent epidemiological investigation indicated that individuals with AN may drop from expected growth curves very early in life (e.g., before age five; Yilmaz, Gottfredson, Zerwas, Bulik, & Micali, manuscript submitted for publication). Some individuals with AN may never have reached a developmentally appropriate highest adult weight, and therefore would not have experienced significant weight loss. In contrast, some observational studies indicate that higher childhood BMI is associated with risk for eating pathology during adolescence (Berkowitz et al., 2016), and an epidemiological study recently explored the causal role of BMI on later disordered eating and found that higher BMI at age seven predicted disordered eating during adolescence

(Reed, Micali, Bulik, Davey Smith, & Wade, 2017). These results suggest that individuals disposed to higher weight may be more likely to engage in disordered eating to induce weight loss to a suppressed, even if normative, weight. When considering WS in a developmental context, it is thus relevant to consider the construct with an eye towards deviations from expected growth patterns, which vary across individuals.

Importantly, although initial investigation indicates that WS may hold clinical utility across developmental stages (Accurso et al., 2016), there are several conceptual and methodological issues relevant when investigating WS across the lifespan. Mechanisms that place overweight individuals at risk for EDs may derive from factors related to WS, specifically related to expectations of weight and height relative to a growth curve. For example, an 8-year old girl who is 53 inches tall and 100 pounds would have a BMI of 25, BMI percentile of 98%, and would be considered obese. If this girl reached an adult height of 63 inches (i.e., maximum lifetime height) at 12 years old and maintained a weight of 110 pounds from 12 to 17 years, her BMI would remain stable at 19.5 throughout that time frame, but her BMI percentile would drop from 75% to 30%. While the child in this example is not weight suppressed according to the current calculation of WS, her BMI is significantly reduced throughout the measurement term with a profile that may indicate that she is at risk for future weight gain because of her "relative" WS. Further, the development of a developmentally-sensitive index of WS is also relevant for young adults (e.g., aged 21), who do not have an extensive adult weight history from which to draw their highest weight measurement. Following the conundrum of calculating WS in a developmentally sensitive manner through emerging adulthood, girls typically reach their adult height before age 15, and yet weight is expected to continue to increase as a normative part of development through age 20. Thus, if a girl reaches her "adult height" at age 14, begins to lose

weight at age 15, and maintains a suppressed weight at age 22, a traditional calculation of WS for this adult woman at age 22 would rely on this individuals highest weight since reaching adult height (at age 15), without considering her expected weight gain throughout later adolescence. As research attempts to evaluate mechanisms by which WS may relate to risk for eating pathology, defining how this variable may be captured in a developmentally-sensitive way will be critical.

Informing future intervention and prevention. While WS has received increased attention over the past decade as a key construct of interest among ED and obesity research, mixed findings regarding links between WS and weight and eating disorder related outcomes do not currently offer straightforward guidance for clinical assessment and intervention efforts. Closing the gap between research and practice may inform specific treatments, particularly as existing studies of WS suggest that incorporation of assessment of weight history may impact clinical decision-making. For instance, weight gain throughout intervention for eating pathology may be a likely outcome if an individual presents at a suppressed weight (Shaw et al., 2012), As such, assessing WS in combination with fear of weight gain may be clinically informative for ED treatment planning. While current treatments for BN are generally considered to be "weight neutral," individuals high in WS are more likely to gain weight than non-suppressed counterparts (e.g., Carter et al., 2008); as such, offering treatment options that encourage distress tolerance related to weight fluctuation may improve treatment engagement and adherence for those at risk. Weight gain within the context of treatment may be necessary for some individuals with AN or BN, but might be contraindicated for some individuals with atypical AN, BN or BED. Consideration of a patient's WS might suggest a weight which he/she might be biologically predisposed to return to, absent of ED behaviors. While need to gain or

lose weight to reach this eventual weight might differ transdiagnostically, it is important to acknowledge, with patients, the impact that WS might have had in the etiology and maintenance of his/her ED. Recent work identifies specific strategies that clinicians may put into place to address WS and related fear of weight gain within standard CBT treatment for BN (Juarascio et al., 2017). For example, these authors suggest that educating patients about WS (both generally, and in the context of their own weight history) might help patients to see that his/her prior efforts to lose weight may have contributed to BN disorder development. Further, given that WS is generally associated with weight gain in ED treatment, the authors suggest that it would be particularly beneficial to foster patient flexibility and acceptance of this phenomenon. Recent network analysis consistently identifies fear of weight gain as a central symptom to ED pathology in individuals with BN (Levinson et al., 2017). Towards this end, a clinician might help a patient to understand that slight weight gain may reduce urges to binge eat, or assist the patient in entertaining the pros and cons of weight gain in light of other aspects of their life and recovery. Juarascio and colleagues (2017) also called for future research to determine a level of WS that is clinically meaningful, one at which a patient may experience significant impact on treatment response, and symptom maintenance.

Increased consideration of detailed weight history (i.e., both the timing and amount of weight change) may also be particularly clinically meaningful in informing treatment. In one large prospective study of adolescent women assessed annually over eight years, significant weight change – either weight loss or weight gain (10% change in age adjusted BMI over a period of 1 year) related to development of subthreshold or threshold BN as compared to weight stable participants (Thomas, Butryn, Stice & Lowe, 2011). In addition, those who went on to develop subthreshold or threshold BN gained significantly more weight in the two years prior to

the onset of their ED. Other work studying youth with BN indicates that current BMI and current WS may be particularly problematic for predicting bulimic symptoms in slightly older adolescents (Accurso et al., 2016). In investigation of younger populations with AN, evaluation of weight history indicated that elevated BMI in grade school predicted adolescent onset of AN (Berkowitz et al., 2016). These findings suggest that predisposition toward elevated premorbid BMIs during childhood may characterize those who later develop AN or BN.

Informing future research. In consideration of the above-mentioned issues, further study of weight history that includes a developmentally sensitive measurement of WS is critical in understanding how EDs may develop throughout adolescence. Such calculations in both youth and young adults should be grounded in information provided by standardized growth curves over time. In so doing, researchers would be able to evaluate the impact of suppressed weight, relative to expected development.

Another important future direction for WS research involves better identifying the mechanisms through which WS may relate to weight and eating-related outcomes. To date, some researchers have posited that links between WS, weight trajectory, and eating behaviors can be accounted for by metabolic processes (Leibel et al., 1995). An initial investigation of physiological mechanisms, such as change in metabolic efficiency (i.e., the degree to which the body uses fat as an energy source), found that WS was moderately related to changes in resting metabolic rate and total energy expenditure, but such changes did not appear to underlie future increases in BMI (Stice et al., 2011). Alternatively, two studies have examined leptin as a potential biological mechanism that might drive WS-BN symptom associations. Produced by adipose cells, leptin is a hormone that serves to inhibit food intake and regulate the storage of fat; obesity is associated with decreased sensitivity to leptin (Crujeiras et al., 2015). Initial findings

from Bodell and Keel (2015) did not support a significant association between WS and leptin levels. However, in a mediation study examining associations between WS, leptin and duration of illness, Keel, Bodell, Haedt-Matt, Williams, and Applebaum (2017) found that greater WS, and lower leptin levels were related to longer duration of illness; in this study, leptin levels fully mediated the relation between WS and illness duration.

In addition to the need for research on biological factors that might drive WS-BN associations, further research is needed to identify potential psychological mediators that may account for links between WS and symptoms and weight trajectory. Also mentioned above, one longitudinal study of the link between WS and BN symptoms determined that elevated WS at baseline predicted greater report of bulimic symptoms at 20-year follow-up; increased drive for thinness at 10-year follow-up mediated this effect (Bodell et al., 2017). Of note, this study is the only investigation to date evaluating a proposed psychological mechanism related to the effects of WS.

One psychological mechanism that warrants further study involves relations between WS and appetitive regulation. Neurobiological theories of eating behavior include a hedonic-inhibitory model, in which a particular individual with a goal of weight loss might naturally engage in hedonic feeding (i.e., eating behavior that is based upon immediate reward, and susceptible to visual cue activation), but would then recruit inhibitory control in an effort to engage in dietary restraint (Appelhans, 2009). Currently, no work has explicitly examined the potential impact of sustained WS on the mechanism of inhibitory control (and successful dietary restraint) within this model. It is possible that self-control depletion (via sustained dietary restraint) might be compromised in individuals for whom WS is higher, resulting in difficulties in successful dieting. In contrast, some work has indicated that individuals who are able to

successfully maintain suppressed weights (e.g. successful restrainers, AN patients) may be less prone to self-control depletion as a result of the fact that the task of restraint has been transferred to automatic control mechanisms over time (Appelhans, French, Pagoto, & Sherwood, 2016; Gianini, Walsh, Steinglass, & Mayer, 2017). As such, evaluation of the degree to which food choice for a particular individual is reliant on automatic vs. executive control may be a fruitful avenue for research that attempts to clarify the relation between WS, weight trajectory, and eating pathology.

Other avenues of mechanistic research include examining psychological symptoms reported among individuals with ED, including a fear of weight gain. For those with a significant history of overweight or obese status, this fear may be substantially heightened. Particularly for individuals with this weight status history, elevated WS (and subsequently a current, much lower weight when presenting to treatment) might be particularly negatively reinforcing, in that the WS might alleviate fear of returning to a premorbid weight. To date, no studies have specifically examined fear of weight gain, relative to WS. For those with greater WS, fear of weight gain may be a more salient treatment target within clinical intervention. Further, individuals with this weight status history may also experience internalized weight stigma, potentially related to perceived social reward for having achieved a lower weight and/or internalization of a thin ideal. These psychological mechanisms warrant further investigation, particularly as they may serve as important clinical foci within treatment.

In sum, work investigating psychological mechanisms related to WS is nascent. Accordingly, additional longitudinal work that probes associations between WS, weight, and eating-related outcomes is critical in clarifying mixed findings and moving the field's understanding of the construct forward.

Conclusions

Over the past several decades, empirical interest in WS has flourished. Findings from recent investigation into WS suggests that across ED diagnoses, WS may relate to symptoms and treatment outcome, specifically in increased weight gain, and maintenance of BN symptoms. Within non-clinical samples, research into WS is limited, but tentative results from this line of work also suggest that WS may relate to disordered eating behaviors. However, across both clinical and non-clinical samples, findings are mixed. The reasons for the existing mixed findings may be manifold; however, pursuit of a robust, developmentally-sensitive definition and calculation of WS, an increased emphasis on translating empirical work on WS into the clinical setting, and completion of longitudinal work focused on the mechanisms of observed effects will undoubtedly provide critical information for better understanding this important construct and its implications for weight and eating-related outcomes.

References

Accurso, E. C., Lebow, J., Murray, S. B., Kass, A. E., & Le Grange, D. (2016). The relation of weight suppression and BMIz to bulimic symptoms in youth with bulimia nervosa. *Journal of Eating Disorders, 4*(1). doi:10.1186/s40337-016-0111-5

Appelhans, B. M. (2009). Neurobehavioral inhibition of reward-driven feeding: implications for dieting and obesity. *Obesity, 17*(4), 640-647. doi:10.1038/oby.2008.638

Appelhans, B. M., French, S. A., Pagoto, S. L., & Sherwood, N. E. (2016). Managing temptation in obesity treatment: a neurobehavioral model of intervention strategies. *Appetite, 96,* 268-279. doi:10.1016/j.appet.2015.09.035

Berkowitz, S. A., Witt, A. A., Gillberg, C., Råstam, M., Wentz, E., & Lowe, M. R. (2016). Childhood body mass index in adolescent-onset anorexia nervosa. *International Journal of Eating Disorders, 49*(11), 1002-1009. doi:10.1002/eat.22584

Berner, L. A., Feig, E. H., Witt, A. A., & Lowe, M. R. (2017). Menstrual cycle loss and resumption among patients with anorexia nervosa spectrum eating disorders: Is relative or absolute weight more influential?. *International Journal of Eating Disorders, 50*(4), 442-446. doi:10.1002/eat.22697

Berner, L. A., Shaw, J. A., Witt, A. A., & Lowe, M. R. (2013). The relation of weight suppression and body mass index to symptomatology and treatment response in anorexia nervosa. *Journal of Abnormal Psychology, 122,* 694-708. doi:10.1037/a0033930

Bodell, L. P., Brown, T. A., & Keel, P. K. (2017). Weight suppression predicts bulimic symptoms at 20-year follow-up: The mediating role of drive for thinness. *Journal of Abnormal Psychology, 126*(1), 32-37. doi:10.1037/abn0000217

Bodell, L. P., & Keel, P. K. (2015). Weight suppression in bulimia nervosa: Associations with biology and behavior. *Journal of Abnormal Psychology, 124*, 994-1002. doi:10.1037/abn0000077

Bodell, L. P., Racine, S. E., & Wildes, J. E. (2016). Examining weight suppression as a predictor of eating disorder symptom trajectories in anorexia nervosa. *International Journal of Eating Disorders, 49*(8), 753-763. doi:10.1002/eat.22545

Burnette, C. B., Simpson, C. C., & Mazzeo, S. E. (2017). Exploring gender differences in the link between weight suppression and eating pathology. *Eating Behaviors, 27*, 17-22. doi:10.1016/j.eatbeh.2017.10.001

Butryn, M. L., Juarascio, A., & Lowe, M. R. (2011). The relation of weight suppression and BMI to bulimic symptoms. *International Journal of Eating Disorders, 44*, 612-617. doi:10.1002/eat.20881

Butryn, M. L., Lowe, M. R., Safer, D. L., & Agras, W. S. (2006). Weight suppression is a robust predictor of outcome in the cognitive-behavioral treatment of bulimia nervosa. *Journal of Abnormal Psychology, 115*, 62-67. doi:10.1037/0021-843X.115.1.62.

Carter, F. A., Boden, J. M., Jordan, J., McIntosh, V. V., Bulik, C. M., & Joyce, P. R. (2015). Weight suppression predicts total weight gain and rate of weight gain in outpatients with anorexia nervosa. *International Journal of Eating Disorders, 48*, 912-918. doi:10.1002/eat.22425

Carter, F. A., McIntosh, V. V., Joyce, P. R., & Bulik, C. M. (2008). Weight suppression predicts weight gain over treatment but not treatment completion or outcome in bulimia nervosa. *Journal of Abnormal Psychology, 117*, 936-940. doi:10.1037/a0013942.

Cook, B. J., Steffen, K. J., Mitchell, J. E., Otto, M., Crosby, R. D., Cao, L., ... & Powers, P.

(2015). A pilot study examining diagnostic differences among exercise and weight suppression in bulimia nervosa and binge eating disorder. *European Eating Disorders Review*, *23*, 241-245. doi:10.1002/erv.2350

Crujeiras, A. B., Carreira, M. C., Cabia, B., Andrade, S., Amil, M., & Casanueva, F. F. (2015). Leptin resistance in obesity: an epigenetic landscape. *Life sciences*, *140*, 57-63. doi:10.1016/j.lfs.2015.05.003

Dawkins, H., Watson, H. J., Egan, S. J., & Kane, R. T. (2013). Weight suppression in bulimia nervosa: relationship with cognitive behavioral therapy outcome. *International Journal of Eating Disorders*, *46*, 586-593. doi:10.1002/eat.22137

Dombrowski, S. U., Knittle, K., Avenell, A., Araujo-Soares, V., & Sniehotta, F. F. (2014). Long term maintenance of weight loss with non-surgical interventions in obese adults: systematic review and meta-analyses of randomised controlled trials. *BMJ*, *348*, g2646–g2646. doi:10.1136/bmj.g2646

Duncan, L., Yilmaz, Z., Gaspar, H., Walters, R., Goldstein, J., Anttila, V., ... & Hinney, A. (2017). Significant locus and metabolic genetic correlations revealed in genome-wide association study of anorexia nervosa. *American Journal of Psychiatry*, *174*(9), 850-858. doi: 10.1176/appi.ajp. 2017.16121402

Eisenberg, D., Nicklett, E. J., Roeder, K., & Kirz, N. E. (2011). Eating disorder symptoms among college students: Prevalence, persistence, correlates, and treatment-seeking. *Journal of American College Health*, *59*(8), 700-707. doi:10.1080/07448481.2010.546461

Forney, K. J., Brown, T. A., Holland-Carter, L. A., Kennedy, G. A., & Keel, P. K. (2017). Defining "significant weight loss" in atypical anorexia nervosa. *International Journal of Eating Disorders, 50*(8), 952-962. doi:10.1002/eat.22717

Fothergill, E., Guo, J., Howard, L., Kerns, J. C., Knuth, N. D., Brychta, R., ... & Hall, K. D. (2016). Persistent metabolic adaptation 6 years after "The Biggest Loser" competition. *Obesity, 24*(8), 1612-1619. doi:10.1002/oby.21538

French, S. A., & Jeffery, R. W. (1997). Current dieting, weight loss history, and weight suppression: behavioral correlates of three dimensions of dieting. *Addictive behaviors, 22*(1), 31-44. doi:10.1016/S0306-4603(96)00002-0

Gianini, L.M., Walsh, B.T., Steinglass, J., Mayer, L. (2017) Long-term weight loss maintenance in obesity: Possible insights from anorexia nervosa. *International Journal of Eating Disorders, 50*(4), 341-342. doi:10.1002/eat.22685

Greenway, F. L. (2015). Physiological adaptations to weight loss and factors favouring weight regain. *International Journal of Obesity, 39*(8), 1188-1196. doi:10.1038/ijo.2015.59

Hagan, K. E., Clark, K. E., & Forbush, K. T. (2017). Incremental validity of weight suppression in predicting clinical impairment in bulimic syndromes. *International Journal of Eating Disorders, 50*(6), 672-678. doi:10.1002/eat.22673.

Herman, C. P., & Polivy, J. (1980). Restrained eating. *Obesity*, 208-225. doi:10.1016/b978-0-12-374387-9.00011-8

Herzog, D. B., Thomas, J. G., Kass, A. E., Eddy, K. T., Franko, D. L., & Lowe, M. R. (2010). Weight suppression predicts weight change over 5 years in bulimia nervosa. *Psychiatry Research, 177*, 330-334. doi:10.1016/j.psychres.2010.03.002

Hessler, J. B., Diedrich, A., Greetfeld, M., Schlegl, S., Schwartz, C., & Voderholzer, U. (2017). Weight Suppression But Not Symptom Improvement Predicts Weight Gain During Inpatient Treatment for Bulimia Nervosa. *European Eating Disorders Review*. doi:10.1002/erv.2573

Jenkins, P. E., Lebow, J., & Rienecke, R. D. (2018). Weight Suppression as a Predictor Variable in the Treatment of Eating Disorders: A Systematic Review. *Journal of psychiatric and mental health nursing*. doi:10.1111/jpm.12462

Juarascio, A., Lantz, E. L., Muratore, A. F., & Lowe, M. R. (2017). Addressing Weight Suppression to Improve Treatment Outcome for Bulimia Nervosa. *Cognitive and Behavioral Practice*. doi:10.1016/j.cbpra.2017.09.004

Keel, P. K., Bodell, L. P., Haedt-Matt, A. A., Williams, D. L., & Appelbaum, J. (2017). Weight suppression and bulimic syndrome maintenance: Preliminary findings for the mediating role of leptin. *International Journal of Eating Disorders. 50*(12), 1432–1436. doi:10.1002/eat.22788

Keel, P. K., & Heatherton, T. F. (2010). Weight suppression predicts maintenance and onset of bulimic syndromes at 10-year follow-up. *Journal of Abnormal Psychology, 119*, 268-275. doi:10.1037/a0019190

Klem, M. L., Wing, R. R., McGuire, M. T., Seagle, H. M., & Hill, J. O. (1998). Psychological symptoms in individuals successful at long-term maintenance of weight loss. *Health Psychology, 17*(4), 336-345. doi:10.1037/0278-6133.17.4.336

Latner, J. D., & Ciao, A. C. (2014). Weight-loss history as a predictor of obesity treatment outcome: Prospective, long-term results from behavioral, group self-help treatment. *Journal of Health Psychology*, *19*(2), 253-261.

doi:10.1177/1359105312468191

Lavender, J. M., Shaw, J. A., Crosby, R. D., Feig, E. H., Mitchell, J. E., Crow, S. J., ... & Lowe, M. R. (2015). Associations between weight suppression and dimensions of eating disorder psychopathology in a multisite sample. *Journal of Psychiatric Research, 69*, 87-93. doi:10.1016/j.jpsychires.2015.07.021

Leibel, R. L., Rosenbaum, M., & Hirsch, J. (1995). Changes in energy expenditure resulting from altered body weight. *New England Journal of Medicine, 332*(10), 621-628. doi:10.1056/nejm199503093321001

Levinson, C. A., Zerwas, S., Calebs, B., Forbush, K., Kordy, H., Watson, H., ... & Runfola, C. D. (2017). The core symptoms of bulimia nervosa, anxiety, and depression: A network analysis. *Journal of abnormal psychology*, *126*(3), 340. doi:10.1037/abn0000254

Lowe, M. R. (1984). Dietary concern, weight fluctuation and weight status: Further explorations of the Restraint Scale. *Behaviour Research and Therapy, 22*(3), 243- 248. doi:10.1016/0005-7967(84)90004-4

Lowe, M. R. (1986). Dieting and binging: Some unanswered questions. American Psychologist, 41(3), 326–327. doi:10.1037/0003-066x.41.3.326

Lowe, M. R. (1993). The effects of dieting on eating behavior: A three-factor model. *Psychological bulletin, 114*(1), 100-121. doi:10.1037/0033-2909.114.1.100

Lowe, M. R. (2015). Dieting: proxy or cause of future weight gain?. *Obesity Reviews, 16*, 19-24. doi:10.1111/obr.12252.

Lowe, M. R., Annunziato, R. A., Markowitz, J. T., Didie, E., Bellace, D. L., Riddell, L., ... & Stice, E. (2006). Multiple types of dieting prospectively predict weight gain during the freshman year of college. *Appetite, 47*, 83-90. doi:10.1016/j.appet.2006.03.160

Lowe, M. R., Berner, L. A., Swanson, S. A., Clark, V. L., Eddy, K. T., Franko, D. L., ... & Herzog, D. B. (2011). Weight suppression predicts time to remission from bulimia nervosa. *Journal of Consulting and Clinical Psychology, 79,* 772-776. doi:10.1037/a0025714.

Lowe, M. R., Davis, W., Lucks, D., Annunziato, R., & Butryn, M. (2006). Weight suppression predicts weight gain during inpatient treatment of bulimia nervosa. *Physiology & Behavior, 87,* 487-492. doi:10.1016/j.physbeh.2005.11.011

Lowe, M. R., & Kleifield, E. I. (1988). Cognitive restraint, weight suppression, and the regulation of eating. *Appetite, 10,* 159-168. doi:10.1016/0195-6663(88)90009-8.

Lowe, M. R., Thomas, J. G., Safer, D. L., & Butryn, M. L. (2007). The relationship of weight suppression and dietary restraint to binge eating in bulimia nervosa. *International Journal of Eating Disorders, 40,* 640-644. doi:10.1002/eat.20405

Mason, C., Foster-Schubert, K. E., Imayama, I., Xiao, L., Kong, A., Campbell, K. L., ... & Blackburn, G. L. (2013). History of weight cycling does not impede future weight loss or metabolic improvements in postmenopausal women. *Metabolism, 62*(1), 127-136. doi:10.1016/j.metabol.2012.06.012

Miotto, G., Chiappini, I., Favaro, A., Santonastaso, P., & Gallicchio, D. (2017). Assessing the role of weight suppression (WS) and weight loss rate (WLR) in eating disorders. *European Psychiatry, 41,* S71-S72. doi:10.1016/j.eurpsy.2017.01.230

Mitchell, K. S., Neale, M. C., Bulik, C. M., Lowe, M., Maes, H. H., Kendler, K. S., & Mazzeo, S. E. (2011). An investigation of weight suppression in a population-based sample of female twins. *International Journal of Eating Disorders, 44,* 44-49. doi:10.1002/eat.20780

Morgan, P. J., & Jeffrey, D. B. (1999). Brief report restraint, weight suppression, and self-report reliability: how much do you really weigh?. *Addictive Behaviors, 24*, 679- 682. doi:10.1016/S0306-4603(98)00051-3

Polivy, J., & Herman, C. P. (1985). Dieting and binging: A causal analysis. *American Psychologist, 40*(2), 193-201. doi:10.1037/0003-066x.40.2.193

Reed, Z. E., Micali, N., Bulik, C. M., Davey Smith, G., & Wade, K. H. (2017). Assessing the causal role of adiposity on disordered eating in childhood, adolescence, and adulthood: a Mendelian randomization analysis. *The American Journal of Clinical Nutrition*, *106*(3), 764-772. doi:10.3945/ajcn.117.154104

Schaumberg, K., Anderson, L. M., Reilly, E. E., Gorrell, S., Anderson, D. A., & Earleywine, M. (2016). Considering alternative calculations of weight suppression. *Eating Behaviors, 20*, 57-63.doi:10.1016/j.eatbeh.2015.11.003

Shaw, J. A., Herzog, D. B., Clark, V. L., Berner, L. A., Eddy, K. T., Franko, D. L., & Lowe, M. R. (2012). Elevated pre-morbid weights in bulimic individuals are usually surpassed post-morbidly: Implications for perpetuation of the disorder. *International Journal of Eating Disorders, 45*, 512-523. doi:10.1002/eat.20985

Stice, E., Davis, K., Miller, N. P., & Marti, C. N. (2008). Fasting increases risk for onset of binge eating and bulimic pathology: a 5-year prospective study. *Journal of abnormal psychology*, *117*(4), 941-946. doi:10.1037/a0013644

Stice, E., Durant, S., Burger, K. S., & Schoeller, D. A. (2011). Weight suppression and risk of future increases in body mass: effects of suppressed resting metabolic rate and energy expenditure. *The American Journal of Clinical Nutrition, 94*, 7-11. doi:10.3945/ajcn.110.010025

Swenne, I., Parling, T., & Ros, H. S. (2017). Family-based intervention in adolescent restrictive eating disorders: early treatment response and low weight suppression is associated with favourable one-year outcome. *BMC Psychiatry*, *17*(1), 333. doi:10.1186/s12888-017-1486-9

Thomas, J. G., Butryn, M. L., Stice, E., & Lowe, M. R. (2011). A prospective test of the relation between weight change and risk for bulimia nervosa. *International Journal of Eating Disorders, 44,* 295-303. doi:10.1002/eat.20832

Vall, E., & Wade, T. D. (2015). Predictors of treatment outcome in individuals with eating disorders: A systematic review and meta-analysis. *International Journal of Eating Disorders, 48,* 946-971. doi:10.1002/eat.22411

Van Son, G. E., van der Meer, P. A., & Van Furth, E. F. (2013). Correlates and associations between weight suppression and binge eating symptomatology in a population-based sample. *Eating Behaviors, 14,* 102-106. doi:10.1016/j.eatbeh.2012.11.003

Wildes, J. E., & Marcus, M. D. (2012). Weight suppression as a predictor of weight gain and response to intensive behavioral treatment in patients with anorexia nervosa. *Behaviour Research and Therapy*, *50*(4), 266-274. doi:10.1016/j.brat.2012.02.006

Witt, A. A., Berkowitz, S. A., Gillberg, C., Lowe, M. R., Råstam, M., & Wentz, E. (2014). Weight suppression and body mass index interact to predict long-term weight outcomes in adolescent-onset anorexia nervosa. *Journal of Consulting and Clinical Psychology, 82,* 1207-1211. doi:10.1037/a0037484

Wye, G., Dubin, J. A., Blair, S. N., & Pietro, L. (2007). Weight Cycling and 6-Year Weight Change in Healthy Adults: The Aerobics Center Longitudinal Study. *Obesity*, *15*(3), 731-739. doi:10.1038/oby.2007.598

Yilmaz, Z., Gottfredson, N.C., Zerwas, S.C., Bulik, C.M., & Micali, N. Developmental premorbid BMI trajectories of adolescents with eating disorders in a longitudinal population cohort (manuscript submitted for publication)

Zunker, C., Crosby, R. D., Mitchell, J. E., Wonderlich, S. A., Peterson, C. B., & Crow, S. J. (2011). Weight suppression as a predictor variable in treatment trials of bulimia nervosa and binge eating disorder. *International Journal of Eating Disorders, 44*, 727-730. doi:10.1002/eat.20859