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# Weight suppression and its relation to eating disorder and weight outcomes: a narrative review

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## Abstract

Weight suppression (WS) refers to the discrepancy between highest adult weight and current weight, and has been examined as a key construct related to both: eating pathology and weight management. However, despite increasing interest in WS, findings regarding the clinical implications of WS are often conflicting. For instance, WS has been associated with both adaptive and maladaptive outcomes across various populations. 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 outline recommendations for future study.

Weight suppression (WS) refers to the difference between an individual's highest weight since reaching adulthood (outside of pregnancy) and their current weight (Lowe, 1984, 1993). Interest in WS and its links to eating pathology has burgeoned over the past decade, under the proposition that maintaining a relatively low weight for one's body will create metabolic and psychological resistance and pressure to return to a higher weight. Reflecting increased interest in WS in eating disorder (ED) and weight management research, along with a growing emphasis on metabolic and biological mechanisms underlying the development of eating pathology and obesity, recent reviews of WS have been published that examined the predicted outcome of psychological treatment for ED (e.g., Jenkins, Lebow, & Rienecke, 2018). However, no reviews to date have provided a more extensive examination of WS, across a wide range of individuals with and without eating and weight disorders. The current paper reviews existing findings on WS across the spectrum of weight- and eating-related behaviours (Table 1). First, we describe findings regarding WS in clinical and non-clinical populations, including the 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 review findings on WS within college and

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community samples. Finally, we highlight controversies and limitations of the current assessment of WS, as well as important areas for future study.

## Weight suppression and associated outcomes

### Weight suppression in clinical samples

The majority of research on WS has evaluated its association with ED symptoms. A recent systematic review, which focused on the role of WS as a predictor of treatment outcome in EDs, determined that WS was associated with weight gain following treatment, but not with other treatment outcomes (e.g., treatment completion; symptom abstinence) (Jenkins et al., 2018). A more detailed investigation of the existing literature is warranted, as the hypothesis that WS impacts treatment outcome is not consistently supported. In the following, we explore existing work linking WS to clinical symptoms, weight trajectory, and treatment response across diagnostic samples.

### Bulimia nervosa

**ED pathology.—**Research in samples of individuals with BN suggests that greater WS is associated with increased eating pathology. In treatment-seeking individuals with BN, elevated WS is cross-sectionally associated with greater binge eating and purging frequency at treatment admission (Butryn, Juarascio, & Lowe, 2011; Lowe, Thomas, Safer, & Butryn, 2007). These findings suggest individuals may engage in bulimic behaviours to avoid returning to premorbid body weights (e.g., Garner & Fairburn, 1988), as the behaviours needed to maintain a suppressed weight (e.g., caloric restriction) may increase risk for binge eating and compensatory behaviours (Herman & Polivy, 1975; Lowe, 1986; Stice, Davis, Miller, & Marti, 2008).

Investigations have also examined factors that may moderate the association between WS and BN symptoms. Butryn et al. (2011) tested whether the interaction between current body mass index (BMI) and WS at pretreatment accounted for binge eating and purging frequency in treatment-seeking women with BN. This interaction was not significant for purging, as elevated WS related positively to purging frequency regardless of pretreatment BMI; however, the WS × BMI interaction term predicted binge eating, such that individuals with low BMI and high WS reported more frequent binge eating than those with higher BMIs. Whilst this result suggests individuals with lower BMI and greater WS at treatment entry may be at risk for binge eating, other research has not replicated this finding (Dawkins Watson, Egan, & Kane, 2013).

Other research has suggested that the timing of highest weight attainment (premorbid vs. postmorbid) may relate to the age of onset and duration of BN pathology (Shaw et al., 2012). One study of adolescents with BN found that *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 endorsed more frequent 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 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 in BN or BED, greater WS related to increased treatment dropout (Vall & Wade, 2015). This effect was calculated from five 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 has also demonstrated that elevated pretreatment WS predicts greater bulimic symptom maintenance over the course of cognitive behavioural (CBT) interventions in both outpatient (Butryn, Lowe, Safer, & Agras, 2006; Herzog et al., 2010; Lowe et al., 2011) and inpatient samples (Lowe, Davis, Lucks, Annunziato, & Butryn, 2006). However, other results do not consistently support the role of WS in the maintenance of BN symptoms following treatment (e.g., Carter, McIntosh, Joyce, & Bulik, 2008). A review of treatment outcome data amongst patients referred for CBT for BN over an 8-year period failed to find the evidence of significant associations between WS and bulimic symptom outcomes, including abstinence from and frequency of binge eating and purging behaviours (Dawkins et al., 2013). Additional research should strive to clarify whether and how WS might relate to bulimic symptomatology over the course of treatment, and longitudinally thereafter, amongst individuals with BN.

**Weight trajectory.—**In both inpatient (Lowe, M. R., Davis, W., Lucks, D., Annunziato, R., & Butryn, M. 2006), and outpatient (Carter et al., 2008) females with BN, WS positively related to weight gain over treatment. A recent study of inpatient females with BN sought to replicate these findings, and, although 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). Examination of the rate of weight gain in treatment-seeking women with BN has indicated that higher baseline WS may relate to faster weight gain over 5 years (Herzog et al., 2010). Overall, existing evidence supports associations between higher pretreatment/baseline WS and weight gain during both inpatient and outpatient CBT treatments, an effect that may persist over time, though additional replication of these effects is needed. Given that weight gain during and after the treatment might inspire dieting behaviour that maintains BN or contributes to relapse, the 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 associations with BN symptoms; however, WS might have similar utility in accounting for ED symptomatology amongst individuals with AN.

**ED pathology.—**Within a sample of individuals receiving residential treatment for AN, WS demonstrated significant, cross-sectional correlations with 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 ED 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), such that amongst those with high BMI at discharge, higher WS predicted increased purging after discharge compared to lower WS.

Several studies indicate that weight history may account for variability in the onset and trajectory of AN symptoms. Within individuals with adolescent-onset AN and matched controls, individuals with AN had significantly higher BMIs than healthy controls during infancy (as measured by ponderal index) and childhood (Berkowitz et al., 2016), suggesting that examining highest weight at various developmental periods may inform ED work. In addition, consideration of rate and magnitude of prior weight loss may also 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 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 4 months prior to treatment admission. Moreover, higher past BMI related to the loss of menses at a higher BMI, and higher past BMI related positively to BMI at menses resumption. Overall, findings 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.—**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 AN samples have been mixed. Initial study of WS in AN suggested that higher levels of WS predicted greater total and faster rates of weight gain over the course of inpatient behavioural treatment, as well as an increased likelihood of engaging in binge eating and purging behaviours at discharge (Wildes & Marcus, 2012). Other research replicated these results, 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 the 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 at intake was related to *better* outcomes, as defined by the absence of clinically significant ED psychopathology.

Other work has found no direct links between WS and AN treatment outcome, and thus has prompted the evaluation of other variables that may interact with WS to more precisely account for treatment outcomes. Berner et al. (2013) found that whilst 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 behaviours) at discharge. Instead, the interaction between WS and BMI was a significant predictor of outcome amongst those with

higher WS, such that individuals with higher BMI and higher WS at intake had greater symptoms at discharge; whereas, those with lower BMI and higher WS at intake had fewer symptoms at discharge. However, this study failed to replicate findings linking WS with binge eating and purging at discharge.

**Longer-term weight trajectory.—**The few existing studies evaluating links between WS and longer-term weight trajectory amongst individuals with AN consistently suggest that higher baseline WS is associated with greater weight gain over time. For example, amongst individuals with AN identified through community-based screening, Witt et al. (2014) found that baseline WS was positively associated with BMI at 6- and 10-year follow-up; this effect was strongest amongst those with lower baseline BMIs. Moreover, baseline WS positively related to BMI at 18-year follow-up. WS at discharge has also related to weight trajectory, as Bodell et al. (2016) found that WS at discharge from intensive treatment for AN predicted positive change in BMI at 3-, 6-, and 12-month follow-up.

### Mixed clinical samples

**ED pathology.—**One investigation of interactions amongst 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 significant associations with weight and shape concerns, exercise and restrictive behaviours, and weight control medication use; WS was not directly related to binge eating or vomiting (Lavender et al., 2015). Relations between WS and ED pathology in this study did not differ by diagnosis. Significant interaction effects included WS × 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 significant positive associations between BMI and weight control medication use amongst those with BN versus EDNOS, and stronger, positive associations between BMI and both binge eating/vomiting and exercise/restrictive eating behaviours amongst those with AN versus EDNOS.

In another 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). Results indicated that WS failed to predict abstinence from binge eating for treatment completers with BED or BN, and failed to predict abstinence from purging or binge eating/purging episodes 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 amongst those who reported lower exercise frequency. In contrast, WS did not differ amongst 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. Unlike most studies that identify WS as a predictor of ED outcomes, WS was

the outcome in this study. Therefore, additional work is first needed to draw conclusions regarding how these findings coincide with exercise-related research in the literature.

**Treatment response.—**In the multisite trial conducted by Zunker et al. (2011) described earlier, for both individuals with BN or BED, WS did not predict treatment completion. 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 positive association between baseline WS and weight gain at the end of treatment (Miotto, Ciappini, Favaro, Santanastapso, & Gallichio, 2017). In contrast, Zunker et al. (2011) found no association between WS and weight change during treatment for participants with BN or BED.

## Weight suppression in non-treatment-seeking samples

Greater WS is linked with a history of consistent caloric restriction (Klem, Wing, McGuire, Seagle, & Hill, 1998). Whilst the long-term goal of dieting is sustained weight loss, WS may have both adaptive and maladaptive associations with 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 than their counterparts with low WS (Lowe & Kleifield, 1988), which may be adaptive for weight-management-related health outcomes. However, WS has also predicted detrimental eating and weight outcomes in healthy populations, including excess weight gain (Stice, Durant, Burger, & Schoeller, 2011) and more frequent loss of control (LOC) and binge eating behaviour (Van Son, Van Der Meer, & Van Furth, 2013).

## Weight suppression in college samples

**ED pathology.—**Despite the increased risk for the development of eating pathology within undergraduates (Eisenberg, Nickett, Roeder & Kirz, 2011), few studies have directly tested associations between WS and eating behaviours in college samples. Early investigations of in-lab behaviour revealed mixed findings. Lowe and Kleinfield (1988) found that within healthy, female undergraduates, self-identified weight suppressors reported higher restraint and ate less food following a milkshake preload. However, another investigation found no significant links between WS, cognitive restraint, and in-lab eating behaviours (Morgan & Jeffrey, 1999).

Research gauging associations between WS and self-reported eating pathology has also yielded conflicting results. One examination of the relation between WS and disordered eating symptoms 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 behaviours, but not to LOC eating; these effects appeared to differentially impact eating behaviour relative to gender (Burnette, Simpson, & Mazzeo, 2017). In this study, men with higher WS engaged in more frequent purging behaviours (i.e., vomiting; laxative use).

**Weight trajectory.—**Greater WS is associated with weight gain over time in undergraduates (Lowe, M. R., Annunziato, R. A., Markowitz, J. T., Didie, E., Bellace, D. L., Riddell, L., … Stice, E. 2006; Lowe et al., 2007); however, few studies have tested potential mechanisms of these effects. 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). Thus, psychological variables (e.g., perceived deprivation) and behavioural variables related to eating and exercise should be investigated further to examine links between WS and weight gain.

## Weight suppression in community-based samples

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

Within a population-based sample of female adult twin dyads, Mitchell et al. (2011) found that childhood restraint and dieting related to increased WS. Additionally, body dissatisfaction, restraint, age, and drive for thinness positively related to WS amongst non-binge eaters; whereas, only restraint, disinhibition, and dieting during childhood were positively linked to WS amongst individuals who endorsed binge eating and LOC eating. The authors concluded that WS might differentially relate to various factors, depending on ED vulnerability status.

Prospective associations 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, suggested that greater WS at baseline assessment predicted maintenance of bulimic symptoms, more gradual decreases in symptoms over time, and increases in symptoms from baseline to 10-year follow-up (Keel & Heatherton, 2010). 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 might be a core risk factor for individuals vulnerable for BN eating pathology. Further examination of this sample revealed that higher baseline WS 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 et al., 2017).

Limited examinations of WS and associated maladaptive outcomes within community populations 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 might be more problematic in maintaining ED pathology over time. 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 behaviour), it did demonstrate a medium effect size in independently predicting clinical impairment.

**Weight trajectory.—**Prospective study of obese individuals has indicated long-term difficulty in sustaining a suppressed weight (Dombrowski, Knittle, Avenell, Araujo-Soares, & Sniehotta, 2014). Recent research indicates that strong neurobehavioural and biological drives towards restoration of a previously higher weight may interfere with successful weight management (Appelhans, French, Pagoto, & Sherwood, 2016; Fothergill et al., 2016; Greenway, 2015).

Whilst weight-suppressed individuals may be at risk for weight regain to a previous highest weight, research has also investigated whether WS drives weight gain *beyond* this set point. History of WS did not independently increase risk for longitudinal weight gain for men, and only marginally so for women (Wye, Dubin, Blair, & Pietro, 2007). Additional evidence indicates 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).

## Current controversies and future directions

To date, only one study has explicitly studied WS in a sample of individuals with a primary diagnosis of BED (Zunker et al., 2011); therefore, it is critical that future work clarifies associations between WS and eating and weight-related outcomes in understudied populations. Overall, 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 inconsistency in the operationalization of WS, with some studies calculating WS by subtracting an individual's current weight from his or her highest ever adult, non-pregnancy weight (Butryn et al., 2006; Herzog et al., 2010; Stice et al., 2011), others using BMI units (Berner et al., 2013; Witt et al., 2014), some choosing to dichotomize samples into "high/low" WS (Butryn et al., 2011; Carter et al., 2008; Zunker et al., 2011), and other studies using alternative methods of gauging weight fluctuation that consider lowest adult weight (Carter et al., 2008; Witt et al., 2014). Inconsistent operationalization of WS may contribute to mixed findings and limit researchers' ability to compare results across investigations, as different calculations will 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 lb from their highest weight in a similar manner in statistical analyses, even if those women had significantly different heights and weights. Alternatively, for a study using changes in BMI units, these women would be considered differentially weight suppressed. 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 WS, varying calculations of the construct limit the ability to compare results of statistical tests (Schaumberg, Anderson, Reilly, Gorrell, & Anderson, 2016). Moreover, existing WS calculations often use a change score and are collected in a self-report format, both of which may decrease reliability and validity. Preliminary work comparing methods of WS calculation determined that it was 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, Brown, Holland-Carter, Kennedy, & Keel, 2017). It is recommended that researchers consider operationalizing WS in a manner that maximizes reliability, validity, and 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, as adolescence and young adulthood is a high-risk period for the development of eating pathology, defining WS in a way that is developmentally sensitive is paramount to ED research. Recent work on the relation between EDs and body weight throughout childhood reveals 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 work showing that individuals with AN demonstrated higher childhood weight (Berkowitz et al., 2016), a recent epidemiological investigation indicated that individuals with AN might drop from expected growth curves very early in life (e.g., before age five; Yilmaz, Gottfredson, Zerwas, Bulik, & Micali, in press). Some individuals with AN might have never reached a developmentally appropriate highest adult weight, and, therefore, would not have experienced significant weight loss. In contrast, other studies indicate that higher childhood BMI was 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 engage in disordered eating to induce weight loss to a suppressed, even if normative, weight. When considering WS in a developmental context, it is relevant to consider deviations from expected growth patterns.

Although initial investigation indicates that WS may be clinically relevant 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” tall and 100 lb would have a BMI of 25, BMI

percentile of 98%, and would be considered obese. If this girl reached her maximum lifetime height of 63” at 12 years old and maintained a weight of 110 lb from 12 to 17 years, her BMI would remain stable at 19.5 throughout that timeframe, but her BMI percentile would drop from 98% at age 10 to 75% at age 12 to 30% at age 19.5. Whilst this child would not be weight suppressed according to the current calculation of WS, her BMI is significantly reduced throughout the measurement term with a profile that may indicate a 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. Notably, 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 her highest weight since reaching adult height (at age 15), without considering her expected weight gain throughout later adolescence. As the research attempts to evaluate mechanisms by which WS may relate to risk for eating pathology, capturing WS in a developmentally sensitive way will be critical.

## Informing future intervention and prevention

Mixed findings regarding links between WS and weight- and ED-related outcomes do not offer a straightforward guidance for clinical assessment and intervention efforts. However, existing studies of WS suggest that incorporation of assessment of weight history may impact clinical decision-making (Table 2). For instance, although treatments for BN are generally considered to be “weight neutral,” individuals with high WS are more likely to gain weight in treatment (Shaw et al., 2012). Accordingly, assessing WS in combination with fear of weight gain may be clinically informative, and promoting distress tolerance techniques to cope with weight fluctuation may improve treatment engagement and adherence. Weight gain within the context of treatment may be necessary for individuals with AN and many individuals with BN. Consideration of a patient’s WS may suggest a weight that he/she may be biologically predisposed to return to, the absent of ED behaviours. Whilst need to gain or lose weight may differ across individuals, 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 employ to address WS and related fear of weight gain within CBT treatment for BN (Juarascio et al., 2017). Educating patients about WS (both generally, and in the context of individual weight history) may help patients understand that prior efforts to lose weight may have contributed to BN disorder development (Juarascio et al., 2017). Further, given that WS is associated with weight gain, the authors suggest that it would be particularly beneficial to foster patient flexibility and acceptance of this phenomenon. They 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. 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 may target fear of weight gain using exposure-based techniques, provide psychoeducation that

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 his/her life and recovery.

Increased consideration of weight history (i.e., the timing/amount of weight change) may also be clinically meaningful. In a large prospective study of adolescent women assessed annually over 8 years, significant weight loss or 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 more weight in the 2 years prior to the onset of their ED. Other work studying youth with BN indicates that current BMI and WS may specifically predict bulimic symptoms in older adolescents (Accurso et al., 2016).

## Informing future research

Overall, development of a developmentally sensitive measurement of WS will be critical in studying WS and EDs throughout adolescence. Ideally, this calculation will be informed by standardized growth curves over time.

Another important future research direction involves identifying 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 behaviours can be accounted for by metabolic processes (Leibel, Rosenbaum, & Hirsch, 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 changes did not appear to underlie future increases in BMI (Stice et al., 2011). Alternatively, two studies have examined leptin as a potential 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, and leptin levels mediated the relation between WS and illness duration.

In addition to a focus on biological influences, further research should identify potential psychological mediators of the association between WS, ED symptoms, and weight trajectory. Also mentioned earlier, one longitudinal study of the link between WS and BN symptoms determined that baseline WS related to bulimic symptoms at 20-year follow-up; increased drive for thinness mediated this effect (Bodell et al., 2017). To date, this study is the only investigation that has tested a psychological mechanism of the WS-ED association.

One psychological mechanism that warrants further study is appetitive regulation. Neurobiological theories of eating behaviour include a hedonic-inhibitory model, in which an individual with a goal of weight loss may naturally engage in hedonic feeding (i.e., eating behaviour that is based upon immediate reward, and susceptible to visual cue activation), but will then recruit inhibitory control in an effort to engage in dietary restraint (Appelhans,

2009). Currently, no work has explicitly examined the impact of sustained WS on the mechanism of inhibitory control within this model. It is possible that self-control depletion (via sustained dietary restraint) may be compromised in individuals with high WS, resulting in difficulties in successful dieting. Alternatively, 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 et al., 2016; Gianini, Walsh, Steinglass, & Mayer, 2017). Accordingly, considering the degree to which restraint is reliant on automatic versus executive control may help to clarify the associations between WS, weight trajectory, and eating pathology.

Other avenues of mechanistic research include examining psychological symptoms reported amongst individuals with ED, including a fear of weight gain. Particularly, for individuals with a history of weight-related teasing or experienced stigma, elevated WS might be negatively reinforcing, in that WS might alleviate fear of returning to a premorbid weight. To date, no studies have specifically examined fear of weight gain, relative to WS, a potentially salient treatment target within clinical intervention. These psychological mechanisms warrant further investigation; particularly, as they may serve as important clinical foci within treatment.

## Conclusions

Existing research suggests that WS may relate to ED symptoms and treatment outcome, and longitudinal findings are most consistent for weight trajectory and the maintenance of BN symptoms. In non-clinical samples, WS research is limited, but initial results support associations between WS and disordered eating behaviours. However, across both clinical and non-clinical samples, findings are mixed. The reasons for mixed findings may be manifold; however, pursuit of a robust, developmentally sensitive definition and calculation of WS, an increased emphasis on translating work on WS into the clinical setting, and longitudinal study focused on the mechanisms of observed effects will undoubtedly provide critical information for better understanding of this important construct and its implications for weight and eating-related outcomes.

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### Clinical Implications

- Results from study of weight suppression (WS) are inconsistent
- WS might relate to increased weight gain, and maintenance of bulimic symptoms
- Within non-clinical samples, WS might relate to disordered eating behaviors
- Future work should include developmentally-sensitive definition and calculation of WS
- Study of mechanisms related to WS, and translation into clinical settings is needed

**Table 1.**

Studies with weight suppression as a variable of interest.

| Study | Sample | Study Design | Variables of Interest | Main Findings |
|---|---|---|---|---|
| Accurso et al., 2016 | Treatment-seeking youth, aged 12–17 years, diagnosed with BN ($n$ = 85, 91.6% female | Cross-sectional | WS (current and greatest), BMIz, objective binge eating, compensatory behaviours, dietary restraint in past month | Higher levels of greatest WS (but not current WS) were positively associated with age ($r$ = .311, $p$ = .007), duration of illness ($r$ = .423, $p$ = .0003), and weight and shape concern ($r$ = .221, $p$ = .048). Adjusting for BMIz, WS (current or greatest) was not significantly associated with bulimic behaviours or dietary restraint in the full sample. For older participants (≥ 16 years) current WS moderated effects of BMIz on binge eating ($B$ = 4.865, $p$ = .037) and compensatory behaviours ($B$ = −8.987, $p$ = .013). Adjusting for age, duration of illness, and weight/shape concern, there was a significant main effect of current WS on dietary restraint ($B$ = 0.374, $p$ = .003) in the full sample. |
| Berner et al., 2013 | Women diagnosed with AN in inpatient treatment who completed measures at admission ($n$ = 350) and discharge ($n$ = 238) | Longitudinal | WS, BMI, and WS × BMI interaction as predictors of psychological symptoms and weight in AN | At admission, correlations between baseline BMI and WS were significant ($r$ = −.22, $p$ < .001) and WS positively correlated with all measures except Weight Concern and Body Dissatisfaction subscales. At discharge, the admission WS × BMI interaction consistently predicted posttreatment psychopathology (e.g., EDE-Q global scores: $B$ = 0.14, $p$ < .01). Higher admission WS predicted lower discharge scores amongst those with lower BMIs when controlling for weight gain; amongst those with higher BMIs, higher WS predicted higher discharge scores. |
| Bodell et al., 2015 | Women diagnosed with BN ($n$ = 32), and non-eating disorder controls ($n$ = 30) | Cross-sectional | WS, leptin levels, reinforcing value of food | Individuals with BN had greater WS (Cohen's $d$ = 0.81, $p$ < .01) and reinforcing food value (Cohen's $d$ = 0.63, $p$ < .05) compared to controls. WS was associated with lower leptin levels in the full sample ($B$ = −0.24, $p$ < .05) and BN sample ($B$ = −0.34, $p$ < .04). WS was significantly associated with reinforcing value of food in the full sample ($r$ = .35, $p$ < .01). WS was also significantly associated with higher frequency of compensatory behaviours, larger binge-episode size, and longer duration of illness in BN. Relations between WS and binge size remained significant whilst controlling for BMI ($B$ = 0.46, $p$ < .01). |
| Bodell et al., 2016 | Women diagnosed with AN ($n$ = 180) | Longitudinal | WS, BMI, and WS × BMI interaction as predictors of the trajectory of ED symptoms | WS at discharge predicted change in BMI. WS × BMI interaction predicted growth in eating disorder severity and purging frequency over time ($b$ = .39, se($b$) = .03, $p$ = .03). Neither WS nor its interaction with BMI predicted growth in LOC eating frequency. |
| Bodell et al., 2017 | Community sample of women ($n$ = 1,190) and men ($n$ = 509) | Longitudinal | WS, bulimic symptoms, drive for thinness (DT) | Higher WS at baseline predicted higher bulimic symptoms at 20-year follow-up (partial $r^2$ = .012, $p$ < .001). At 10-year follow-up, increased DT mediated this effect such that the ratio of the indirect effect to the total effect indicates that approximately 67% of the effect of WS on bulimic symptoms occurs indirectly through DT, and approximately 3% of variance in 20-year bulimic symptoms is attributable to this indirect effect ($R^2 med$ = .026). |
| Burnette et al., 2017 | Undergraduates ($n$ = 859, 71.7% female) | Cross-sectional | WS, gender, eating pathology | Higher levels of WS were associated with more dietary restraint ($b$ = 0.38, $p$ < .001). WS was indirectly related to LOC eating through dietary restraint across gender ($b$ = 1.75, $p$ < .001). For purging behaviours, gender x WS was statistically significant ($b$ = −0.0323, $p$ = 0.036) indicating that men with higher WS were more likely to engage in extreme weight control behaviours. |
| Butryn et al., 2006 | Women diagnosed with BN in outpatient treatment ($n$ = 188) | Longitudinal | WS, treatment outcomes of a CBT intervention for BN | WS at pretreatment was a significant predictor of treatment completion ($B$ = −0.37, $p$ < .01). For treatment completers, those who continued to engage in binge/purge behaviours had significantly higher levels of WS than those who abstained ($B$ = −0.14, $p$ < .01). |
| Butryn et al., 2011 | Women diagnosed with full or sub-threshold BN beginning intensive outpatient treatment ($n$ = 64) | Cross-sectional | WS, BMI, binge eating, purging behaviours | Both WS ($B$ = .35, $p$ = .01) and BMI × WS ($B$ = −0.41, $p$ = .01) predicted frequency of binge eating. Higher levels of WS also predicted purging behaviours ($B$ = 0.27, $p$ = .03). |

| Study | Sample | Study Design | Variables of Interest | Main Findings |
|---|---|---|---|---|
| Carter et al., 2008 | Women diagnosed with BN in outpatient treatment ($n$ = 132 | Longitudinal | WS, treatment completion, treatment outcome | WS significantly predicted weight change (i.e., weight gain) over the course of treatment ($B$ = 0.100, $p$ = .01). Amongst treatment completers, WS did not significantly predict binge eating and purging post-treatment. |
| Carter et al., 2015 | Women diagnosed with AN participating in outpatient RCT psychotherapy trial ($n$ = 56) | Longitudinal | WS at pretreatment, total weight gain, faster weight gain, bulimic symptoms | WS was positively associated with total weight gain ($B$ = 0.35, $p$ < .05) and rate of weight gain ($B$ = 0.35, $p$ < .05). WS not significantly associated with bulimic symptoms in the month prior to post-treatment assessment. |
| Cook et al., 2015 | Individuals seeking treatment for eating disorders (BN = 774, 95.9% female; BED = 285, 90.9% female) | Cross-sectional | WS, exercise frequency, BN/BED diagnosis | Exercise frequency ($B$ = 1.55, $p$ = .015) and ED diagnosis ($B$ = 5.55, $p$ = .006) were both associated with WS. The interaction of diagnosis and exercise frequency was significant ($B$ = −2.04, $p$ = .006), indicating that the combined effects of exercise frequency and BED were significantly associated with WS. Exercise duration ($B$ = 3.74, $p$ = .037) and diagnosis ($B$ = 4.27, $p$ = .036) were both associated with WS; however, the interaction of diagnosis and exercise duration was not significant indicating that exercise duration does not moderate the relation of diagnosis and WS. |
| Dawkins et al., 2013 | Women; CBT outpatients with bulimic disorders ($n$ = 117 | Longitudinal | WS, treatment completion, treatment outcome for BN | WS did not significantly predict treatment completion or outcome. |
| French & Jeffery, 1997 | Community sample of women ($n$ = 999) | Cross-sectional | WS, current dieting, history of dieting, behaviours related to energy balance | Twenty-eight percent of participants were classified as weight suppressors. WS was associated with higher physical activity levels ($F$(6,983) = 3.1, $p$ < .005) and low-fat eating behaviours ($F$(5,984) = 2.9, $p$ < .01). |
| Hagan et al., 2017 | Community sample of adults ($N$ = 101; 80.2% female) with full- ($n$ = 51) or subthreshold ($n$ = 50) BN | Cross-sectional | WS, impairment for bulimic syndromes | WS was significantly associated with clinical impairment ($p$ = .011); however, WS did not demonstrate incremental validity over other independent variables. WS explained an additional 1.7% of the variance and had a medium-sized effect (Cohen's $d$ = 0.521). |
| Herzog et al., 2010 | Treatment-seeking women diagnosed with BN ($n$ = 97) | Longitudinal | WS, long-term weight change | Participants' weight increases over the study course were moderated by baseline WS (*coefficient* = .009, $p$ < .001) such that higher WS predicted more rapid weight gain. |
| Hessler et al., 2017 | Women diagnosed with BN in inpatient treatment ($n$ = 179) | Longitudinal | WS, BMI, treatment response | WS was associated with treatment outcome (partial $r^2$ = 0.05, $p$ < .044); however, no association noted between change in BMI and treatment outcome. |
| Keel & Heatherton, 2010 | Undergraduate men ($n$ = 369) and women ($n$ = 968) | Longitudinal | WS, bulimic syndrome maintenance and onset | Among participants with bulimic syndrome at baseline, greater WS predicted maintenance ($B$ = 0.11, 95% CI: 1.01, 1.08). Amongst those without bulimic syndrome at baseline, greater WS predicted onset at 10-year follow-up ($B$ = 0.11, 95% CI: 1.05, 1.19). |
| Keel et al., 2017 | Women; community sample with bulimic syndrome ($n$ = 53) | Cross-sectional | WS, leptin, illness duration | Significant associations noted amongst WS, lower leptin levels, and longer duration of illness. Mediation analyses indicated WS was a significant predictor of illness duration ($b$ = .81, $p$ < .01) and leptin mediated the link between WS and illness duration. |
| Lavender et al., 2015 | Individuals diagnosed with ED ($n$ = 1,748, 94% female) | Cross-sectional | WS, ED diagnosis, BMI, dimensions of ED psychopathology (i.e., weight/ shape concerns, binge eating/ vomiting, exercise/restrictive eating behaviours, and weight control medication use) | WS was uniquely associated with all ED dimensions except binge eating/vomiting [i.e., weight/shape concerns ($B$ = .103, $p$ < .001), exercise/restrictive eating behaviours ($B$ = 0.088, $p$ = .002), and weight control medication use ($B$ = 0.081, $p$ < .001)]. The WS × BMI interaction was significant for only weight/shape concerns ($B$ = −0.092, $p$ = .003) whereas the WS × ED interaction diagnosis was not significant for any ED dimensions. |
| Lowe & Kleifield, 1988 | Undergraduate women ($n$ = 42) | Experimental | WS, cognitive restraint, regulation of eating | Participants with higher WS ate significantly less food than those with lower or no WS (*means* 94.1 g and 49.3 g, respectively) despite the fact that they weighed more, were highly restrained, and had eaten significantly less prior to study. |
| Lowe et al., 2006 | Women in inpatient treatment who met criteria for BN or EDNOS ($n$ = 146) | Longitudinal | WS, pre-treatment BMI, current dieting, and purging as predictors of weight gain during treatment | WS and pre-treatment BMI independently predicted weight gain when entered together in regression analysis. Higher levels of WS predicted greater weight gain after controlling for |

| Study | Sample | Study Design | Variables of Interest | Main Findings |
|---|---|---|---|---|
| | | | | length of stay ($B$ = 0.21, $p$ = .03), current dieting ($B$ = 0.19, $p$ = .03), and purging frequency ($B$ = 0.24, $p$ = .01). |
| Lowe et al., 2007 | Women diagnosed with BN in outpatient treatment ($n$ = 182 | Cross-sectional | WS, dietary restraint, desire to lose weight, binge eating frequency | WS was positively related ($b$ = 0.319, $p$ < .05) and dietary restraint was inversely related ($b$ = 2.03, $p$ < .05) to frequency of binge eating. |
| Lowe et al., 2011 | Women diagnosed with BN ($n$ = 110) | Longitudinal | WS, time to first full remission | WS was significantly associated with time to first full remission [Hazard Ratio = .89, CI (0.82, 0.97), $p$ = .01] suggesting that those with higher WS took longer to recover. Age, highest past weight, baseline weight, and weight rebound were tested as covariates and only slightly changed the association between WS and time to remission. Similarly, weight change did not mediate the relation between WS and time to remission. |
| Miotto et al., 2017 | Patients diagnosed with ED ($n$ = 62 with AN-BP, 146 with AN-R, and 206 with BN) | Longitudinal | WS, weight loss rate | No significant relation noted between WS, weight loss rate, and variables collected at baseline; however, WS and weight gain were significantly associated at the end of treatment. High weight loss rate predicted remission of BE and compensatory behaviours in BN patients. |
| Mitchell et al., 2011 | Women; same-sex twins aged 18–54 ($n$ = 1,503; Monozygotic = 614, Dizygotic = 410) | Longitudinal | WS, eating disorder-related variables, and additive genetic (A), common (C), and unique (E) environmental contributions | Biometrical modelling yielded similar results for participants reporting no binge eating and those reporting binge eating plus loss of control yielded with 20–25% of variance in WS due to A and 70–75% due to E. Restraint ($B$ = 0.14) drive for thinness ($B$ = 0.09), body dissatisfaction ($B$ = −0.08), and dieting during child ($B$ = 0.10) adulthood ($B$ = 0.15) were significantly ($p$ < .05) associated with WS amongst participants reporting no binge eating. Restraint ($B$ = 0.25), disinhibition ($B$ = 0.25), and dieting during childhood ($B$ = 0.22) were significantly ($p$ < .05) associated with WS in participants reporting binge eating plus loss of control. |
| Morgan & Jeffrey, 1999 | Women; restrained eaters who self-reported high or low WS ($n$ = 58) | Experimental | WS, eating behaviours in a standard restraint taste-test paradigm | A 2 × 2 analysis of variance indicated no significant differences between groups (i.e., high WS no-preload group, low WS no pre-load group, high WS preload group, low WS preload group). |
| Stice et al., 2011 | Undergraduate women with elevated BD ($n$ = 91) | Longitudinal | WS, BMI, bulimic symptoms, resting metabolic rate (RMR), suppressed total energy expenditures (TEE) | WS predicted future increases in BMI (semipartial $R$ = 0.09, $p$ = .001). WS correlated inversely with suppressed RMW and TEE. However, when suppressed RMR and TEE were controlled for, WS still predicted increases in BMI (semipartial $R$ for all = 0.09, $p$ = .001). WS did not predict future increases in bulimic symptoms. |
| Swenne et al., 2017 | Adolescents, aged 9.4 −17.8 years, with restrictive ED symptoms in family-based treatment ($n$ = 201, 93.9% female) | Longitudinal | WS, early treatment response, eating disorder symptom recovery | An EDE-Q global score of < 2.0 was independently associated with lower EDE-Q score at presentation (Odds Ratio = 0.66, $p$ = .001), higher weight gain after 3 months of treatment (Odds Ratio = 1.26, $p$ = .002), and lower WS at follow-up (Odds Ratio = 0.57, $p$ = .0013). Not fulfilling criteria for an ED at follow-up was associated with the same factors, as well as BMI at presentation. Higher WS at follow-up was associated with poorer prognosis. |
| Thomas et al., 2011 | Adolescent females aged 12 – 15 years ($n$ = *496*) | Longitudinal | Weight gain, weight loss, bulimic pathology | Substantial weight gain or weight loss over the course of the study increased risk for future onset of bulimic symptoms; however low incidence rate limited statistical power. Participants who showed onset of bulimic symptoms experienced greater increases in weight in the 2 years before the onset of the ED compared to healthy participants ($F$(2, 878) = 3.72, $p$ = .008, $\eta^2$ = 0.01). |
| Van Son et al., 2013 | Community-based sample ($n$ = 3,512, 99% female) | Cross-sectional | WS, binge eating, LOC, diet activity, BD, BMI. | WS was not associated with current binge eating with LOC. High intensity of dieting ($B$ = 1.08, $p$ < .001), being overweight or obese during childhood ($B$ = 3.49, $p$ < .001), and weighing in a moderate ($B$ = 1.36, $p$ < .001) or high frequency ($B$ = 1.72, $p$ < .001) were all positively related to WS. |
| Wildes & Marcus, 2012 | Inpatients diagnosed with AN, aged ≥ 16 years ($n$ = 185, 97% female) | Longitudinal | WS, weight gain, clinical outcomes at discharge | WS at admission was unrelated to achievement in minimally adequate body weight and treatment dropout. Higher levels of WS at admission to intensive treatment predicted greater total weight gain ($B$ = 0.10, $p$ < .05) and rate of weight gain ($B$ = 0.16, $p$ < .05). Higher levels of WS also predicted bulimic symptoms at discharge ($B$ = −0.32, $p$ = .03). |

| Study | Sample | Study Design | Variables of Interest | Main Findings |
|---|---|---|---|---|
| Witt et al., 2014 | Individuals diagnosed with adolescent-onset AN ($n$ = 51; 94.1% female) | Longitudinal | WS and BMI at lowest weight (WS-LW and BMI-LW), BMI at follow-up, duration of AN, total ED duration. | High WS-LW was associated with higher BMI at 6-year and 10-year follow up ($B$ = 0.91, $p$ < .001; $B$ = 0.75, $p$ = .004). BMI-LW was positively associated with BMI at 18-year follow up ($B$ = 0.76, $p$ = .018). No association with WS-LW and AN duration were noted. |
| Zunker et al., 2011 | CBT treatment seeking adults (87.6% female) who met criteria for BED ($n$ = 259) or BN ($n$ = 128) | Longitudinal | WS, treatment completion, weight change during treatment, symptomatic abstinence, and percent reduction, and binge and purging frequency | WS was not a predictor for treatment completion, weight change during treatment, or abstinence and reduction in binge eating and purging episodes amongst BED and BN individuals. |

*Note*. ED = eating disorder; WS = weight suppression; BMI = body mass index; BN = bulimia nervosa; AN = anorexia nervosa; BED = binge eating disorder; DT = drive for thinness; LOC = loss of control; CBT = cognitive behavioural therapy; EDE-Q = Eating Disorder Examination - Questionnaire, BD = body dissatisfaction.

**Table 2.**

Hypothetical clinical presentations with proposed recommendations.

| Diagnosis | Hypothetical Patient Demographics | Hypothetical Clinical Presentations | Proposed Recommendations |
|---|---|---|---|
| Bulimia Nervosa (Low WS) | Female; 28 y.o.; 5'4"<br>***Highest weight***: 180 lbs<br>***Current weight***: 174 lbs, BMI = 29.9 (overweight)<br>***Duration of illness***: 3 months; purging 2× daily | **WS = 6**<br>This patient is currently at an obese BMI, where traditional health parameters would recommend weight loss. Being told by medical weight management staff that she was obese with fatty liver disease triggered the onset of her compensatory behaviours. | **Consider BMI Category from a Patient Perspective**: For this patient, her perception of her BMI classification is impacting her maintenance of ED symptoms. Her WS is low, but the result of this BMI class change holds value, and may complicate treatment (e.g., patient resistant to returning to her potentially genetically and biologically determined obese weight). For this patient, de-emphasizing BMI during treatment might be helpful. |
| Bulimia Nervosa (High WS) | Male; 59 y.o.; 5'9"<br>***Highest weight***: 280 lbs<br>***Current weight***: 169 lbs, BMI = 25.0 (normal)<br>***Duration of illness***: 42 years; over-exercise, muscle dysmorphia | **WS = 111**<br>This patient was bullied related to his weight as a teenager, which he reports triggered his eating disorder onset. Currently, he reports extreme fear of returning to his former size. He sought employment at a gym 4 years ago to facilitate his exercise behaviour and reports no other activities other than his full-day workouts. He was referred to treatment by his orthopedist, treating him for several injuries resulting from over-exercise. | **Consider WS and ED within gender and cultural norms**: Exercise is currently culturally sanctioned, but motivation for weight loss for some individuals can be problematic. For this individual, some WS may have improved his metabolic function, but his current behaviour that maintains his WS has led to loss of quality of life, and risk for Relative Energy Deficiency in Sport. Consider that treatment may involve some weight gain and changes in body composition as patient move towards adaptive exercise and eating patterns. |
| Binge Eating Disorder (Low WS) | Male; 33 y.o.; 5'8"<br>***Highest weight***: 250 lbs<br>***Current weight***: 246 lbs, BMI = 38 (obese)<br>***Duration of illness***: 6 years with history of extreme weight cycling; binge eating three times per week | **WS = 4**<br>This patient has minimal WS. However, he is at risk for complications related to his eating disorder, as well as metabolic and related disease given his weight status. | **Consider current WS *and* history of WS in treatment**: This patient demonstrates low current WS. However, if this patient has had extensive experience of weight cycling, he may be at a comparatively high lifetime weight currently. A treatment provider should consider weight history, as well as current presentation in guiding clinical recommendations within the context of intervention. |
| Binge Eating Disorder (High WS) | Female; 48 y.o.; 5'4"<br>***Highest weight (pre-Roux-n-Y surgery)***: 299 lbs<br>***Current weight***: 145 lbs, BMI = 24.9 (normal)<br>***Duration of illness***: 4 months; binge eating once per week | **WS = 154**<br>This patient presents for treatment at her nadir for post-surgical weight loss. Her BMI is within the normal range, and her pre-surgical obesity related comorbidities (hyperlipidemia, Diabetes mellitus) have resolved. She gets consistent praise from family, friends, and her medical providers for the progress she has made in achieving her current weight. Patient is compliant with post-surgical dietary and lifestyle recommendations, other than when she engages in binge eating. She also reports life history of major depressive disorder, with recent psychosocial stressors. | **Consider WS within specific populations**: This patient is at risk for post-surgical complications resulting from her eating behaviour, with potential for substantial weight regain. Whilst her WS is high, there are other factors that may be more salient in impacting her eating behaviour, including her history of, and current experience of depression which should be clinically treated. |
| Anorexia Nervosa | Female; 41 y.o.; 5'6"<br>***Highest weight***: 125 lbs<br>***Current weight***: 110 lbs, BMI 17.8 (underweight)<br>***Duration of illness***: 6 years; primary restriction | **WS = 15**<br>This patient had a low premorbid BMI (20.2), so reductions in her weight do not appear substantial. However, her current weight is significantly below a healthy range for her height. We might say that her WS is low, but her overall risk for maintenance of ED and related complications is high. | **Establishing a cutoff below which a %BWL in the context of WS might increase risk**: Patient has 12% body weight loss (BWL). Future research might establish a cutoff below which a WS as reflected by %BWL reflects higher risk, and predicts poorer outcome. |

| Diagnosis | Hypothetical Patient Demographics | Hypothetical Clinical Presentations | Proposed Recommendations |
|---|---|---|---|
| Atypical Anorexia Nervosa | Male; 30 y.o.; 5'10"<br>***Highest weight***: 180 lbs<br>***Current weight***: 140 lbs, BMI = 20.1 (normal)<br>***Duration of illness***: 2 years; restriction, overexercise | **WS = 40**<br>This patient is currently at a normal BMI; however for a man of this height, he is at risk of losing more weight given the cognitive and behavioural features of his ED. If we are considering WS and current BMI, we might say he is at a normal BMI, but we have no cutoff to determine if this is an unhealthy amount of WS that might impact his clinical profile going forward. | **Need for assessment of weight history/WS**: With a 40lb weight loss, this patient may present with a fear based in learning history of returning to a premorbid weight. In this case, screening for weight history is very important; presenting at a normal weight within standard medical appointments (e.g., in primary care), this patient would not necessarily be screened for potential ED. |
| Bulimia Nervosa (with developmental considerations) | Female; 11 y.o.; 4'3"<br>***Highest weight***: 92 lbs (1 year ago)<br>***Current weight***: 83 lbs, BMI = 22.4 (normal)<br>***Duration of illness***: 1 year; binge eating and purging, average 3 times per day | **WS = 9**<br>This patient is not at her adult height (her pediatrician estimates projected height of 5'8"). Considering her age and expected growth, she is at high risk for endocrine dysfunction and menstrual disorder, as well as issues with bone density and growth. | **Consider Using Difference in Weight %ile, according to a growth curve**: Patient has history of above 97$^{th}$ %ile in weight for her age, obese BMI by both pediatric and adult standards. Currently she is at an overweight BMI by childhood BMI percentile standards; as she continues to grow, she will experience greater WS. She has likely been commended by medical professionals, family and friends, for her weight loss. Any treatment provider should consider both her weight history, as well as her relative growth in guiding clinical recommendations for both weight maintenance, and eating disorder prevention. |
| Anorexia Nervosa (with developmental considerations) | Female; 24 y.o.; 5'8"<br>***Highest weight***: 119 lbs<br>***Current weight***: 109 lbs, BMI = 16.6 (underweight)<br>***Duration of illness***: 10 years; binge-purge presentation | **WS = 10**<br>This patient has had a lower weight throughout her early adult life, and has maintained a weight within the "underweight" BMI category for several years. Whilst her BMI of 16.6 was within a normal range at age 12 (even with a 10lb weight loss), she has transitioned to underweight status over time even as she maintained this weight. Whilst weight suppression from age 14 "highest ever weight at adult height" is 10lb, expected weight as an adult would be higher. | **Consider longer-term metabolic dysfunction, lack of true data point for expected adult weight**: Being weight suppressed for 10 years, and never having achieved normal weight as an emerging or young adult, this patient may have more pronounced metabolic factors that influence her weight gain, and maintenance of symptoms in treatment. |