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A new, developmentally-sensitive measure of weight suppression

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Abstract

Objective: Weight suppression (WS) has demonstrated associations with numerous indices of eating behavior, psychopathology and eating disorder prognosis. However, because WS has traditionally been measured as a simple subtraction of current weight from highest past weight at adult height, this calculation is problematic for most individuals with disordered eating, who usually reach their highest past weight during adolescence. Here we propose a new method for computing WS to address this shortcoming, termed “developmental weight suppression” (DWS), and provide a web-based tool for ease of calculation.

Method: DWS is calculated as the difference between one's highest premorbid z-BMI (i.e., BMI z-score), and current z-BMI. z-BMIs were calculated using Cole's lambda-mu-sigma (LMS) approach, in accordance with LMS parameters publicly available from the Center for Disease Control (2010). A web-based user interface is available at <https://niuxin.shinyapps.io/devws/>, making its computation easier and its adoption by researchers simpler.

Discussion: By using z-BMIs in place of weights, DWS is more sensitive to the developmentally-relevant factors of age, height, and sex. Preliminary findings suggest that DWS is more strongly related to measures of eating pathology and biological reactions to weight loss than traditionally-computed WS, although more research is needed to test this hypothesis.

Keywords

Weight suppression; Eating disorders; Bulimia nervosa; Developmental history; Adolescence

1. Introduction

Weight suppression (WS), the discrepancy between individuals' highest past weight at adult height and their current weight, has demonstrated associations with numerous measures of eating pathology, weight change, hormone levels, eating disorder (ED) prognosis, and ED treatment outcome.

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Declaration of competing interest

None.

Among individuals with bulimia nervosa (BN), WS has been associated with: greater future weight gain (Piers, Espel-Huynh, & Lowe, 2019); poorer short-term treatment outcomes in CBT (Butryn, Lowe, Safer, & Agras, 2006); heightened fear of weight gain and losing control of eating (Zanetti, Santonastaso, Sgaravatti, Degortes, & Favaro, 2013); longer duration of illness (Keel, Bodell, Haedt-Matt, Williams, & Appelbaum, 2017); increased likelihood of developing a bulimic syndrome over a 10-year period (Keel & Heatherton, 2010); and greater frequency and intensity of binge eating and exercise (Butryn, Juarascio, & Lowe, 2011; Keel & Heatherton, 2010), among other outcomes (for a review, see Lowe, Piers, & Benson, 2018). Among individuals with anorexia nervosa (AN), higher WS has been associated with: greater future weight gain (Berner, Shaw, Witt, & Lowe, 2013; Piers et al., 2019); higher caloric requirements to reach ideal body weight (Haynos, Snipes, Guarda, Mayer, & Attia, 2016); lower likelihood of a return of menses with weight gain (Berner, Feig, Witt, & Lowe, 2017); greater endorsement of bulimic symptoms during treatment (Wildes & Marcus, 2012); and greater probability of elevated psychopathology at 1-year follow-up (Swenne, Parting, & Salonen Ros, 2017). Furthermore, a recent study by Stice and colleagues demonstrated that WS is influential in the etiology of several EDs: elevated WS predicted future onset of AN, BN, and purging disorder over a 3-year period (Stice, Rohde, Shaw, & Desjardins, 2020). Taken together, these findings suggest that the weight discrepancy measured by WS is integral to both the etiology and maintenance, of EDs.

Traditional WS (TWS) is simply calculated as the difference between one's highest past weight at adult height, and his or her current weight. However, there is reason to believe that this calculation is suboptimal (Gorrell, Reilly, Schaumberg, Anderson, & Donahue, 2019; Lowe et al., 2018; Schaumberg et al., 2016). For example, a female who reports a highest past weight of 150 lbs at 12 years of age will have a higher weight relative to her 12-year-old peers, compared to a female who reached the same highest weight at 16, relative to her 16-year-old peers. The assumption underlying WS has been that greater weight loss from one's highest past weight would invoke stronger regulatory responses to promote weight regain, such as binge eating. However, for those individuals who reach their highest past weight before adulthood (i.e., before 21-years), the absolute level of highest past weight is not as intrinsically meaningful as the level of weight elevation relative to the median weight of same-sexed peers of the same age and height. Because EDs usually develop during late adolescence or early adulthood, highest previous weight will often have been reached during adolescence. Therefore, relative to older populations, the substitution of developmentally-calculated WS for TWS may have a particularly large impact when investigating correlates of disordered eating in youth. In addition, because boys often show continued increases in height during adolescence (The Nemours Foundation, 2015), developmentally-calculated WS may also be preferable for this population.

Only one study to date (Accurso, Lebow, Murray, Kass, & Le Grange, 2016), to our knowledge, has published findings using a developmentally-calculated WS. In a sample of youth with BN, WS was negatively correlated with current zBMI, and positively correlated with dietary restraint over the past month. Several interaction effects also emerged within older adolescents (i.e., ages 16 and older): WS moderated the effect of current zBMI on binge eating and compensatory behaviors. For older youth high in WS, higher zBMI was positively associated with binge eating frequency, and the negative relation between zBMI

and compensatory behaviors became weaker. These findings provide preliminary support for the use a developmentally-calculated WS, particularly within older youth with an ED.

Therefore, in this paper we propose a new construct, termed “developmental weight suppression” (DWS). DWS is calculated as the difference between one’s highest *premorbid* and current z-BMIs (i.e., BMI z-scores). By utilizing z-BMIs in place of raw weights, DWS takes into account an individual’s developmental status at highest *premorbid* weight in relation to age, height, and sex, and should therefore come closer to accurately capturing the construct that WS was originally developed to assess (Lowe, 1993). In addition, past research (Shaw et al., 2012) has shown that individuals with BN often reach their highest past weight *after* they develop their ED. Because a highest past weight reached post-morbidly could be impacted by the disorder itself, we opted to calculate the DWS measure using highest *premorbid* weight - not highest weight reached at adult height. We also provide a web-based user interface (UI) for ease of DWS calculation, and the promotion of its use by researchers.

2. Calculation of developmental weight suppression

DWS is calculated as the difference between highest *premorbid* z-BMI and current z-BMI. Cole’s lambda-mu-sigma (LMS) approach (Cole & Green, 1992), per guidelines set forth by the Center for Disease and Control (CDC, 2009), is used to calculate z-BMI (<https://www.cdc.gov/nccdphp/dnpa/growthcharts/>; https://www.cdc.gov/growthcharts/percentile_data_files.htm). Age- and sex-dependent lambda (L, the power of transformation to achieve normality for smoothed BMI percentiles), mu (M, the median of the smoothed percentiles), and sigma (S, the variation of the smoothed percentiles) values are publicly available in a BMI-for-age growth chart on the CDC website (<https://www.cdc.gov/growthcharts/zscore.htm>).

The steps used to calculate DWS are enumerated below. Variables need to calculate DWS include: highest *premorbid* weight, and height and age at which this weight was reached; and current weight, height, and age. Although the UI linked below automatically performs all calculations, the aggregate R-script that performs the calculations is available in the Appendix. Datasets to pilot the script can be found at: github.com/simarsingh25/dws. For brevity, the CDC growth chart used to derive LMS parameters is henceforth referred to as the “reference database,” while the database of calculated DWS values is henceforth referred to as the “target database.”

2.1. Step 1: Calculate current and highest *premorbid* BMIs for participants in target database

Using the BMI formula, calculate current and highest *premorbid* BMIs:

$$BMI = \frac{weight(lbs)}{[height(in)]^2} \times 703$$

2.2. Step 2: Calculate current and premorbid ages, in months, for participants in target database

Because LMS values in the reference database correspond to age in half-months, age in the target database must also be reported in months. This is done for current age and age at which highest premorbid BMI was reached. Knowing an individual's birth date is ideal, because it allows for current age to be calculated to the exact half-month; however, if birth date is unknown, it is also acceptable to simply convert age in years, to age in months.

2.3. Step 3: Recode ages that exceed 240-months, to 240-months

Growth curves plateau after the age of 20 (CDC, 2009); therefore, the reference database stops at 240-months. Any current age or highest premorbid age that exceeds 240-months, must be recoded to 240-months.

2.4. Step 4: Determine lambda, mu, and sigma constants based on age and sex

Two merges are performed: a current status merge, and a highest premorbid status merge. Merges are matched on sex (1 = Male; 2 = Female), and age. Because age in the reference database is reported in half-months, the merge is performed matching reference and target ages to the closest half-month. If target age happens to fall perfectly between two half-month values (i.e., age of 200 months in the target database is equidistant from 199.5 months to 200.5 months in the reference database), the merge rounds up to the greater age. Rounding up compared to rounding down produces very small differences in calculated z-BMI: the final values are identical to the 3rd decimal place.

2.5. Step 5: Calculate z-BMIs

Current and highest premorbid z-BMIs are calculated using Cole's z-BMI formula:

$$zBMI = \left[\left(\frac{BMI}{\mu} \right)^\lambda - 1 \right] \div (\lambda \times \sigma)$$

2.6. Step 6: Calculate DWS

DWS is calculated by subtracting current z-BMI from highest premorbid zBMI. Per the traditional WS calculation, all negative values are recoded to zero. Negative values imply that one's current z-BMI exceeds their highest past z-BMI – which likely means the individual does not realize that their current weight exceeds their self-reported highest past weight.

3. Web-based user interface

To make the derivation of DWS values easier, we created a user-friendly web interface, available at the following webpage: <https://niuxin.shinyapps.io/devws/>. On the UI, there are detailed, step-by-step instructions for preparing a correctly formatted comma-separated values (CSV) file, uploading the file, and designating an output filepath. Once submitted, the program will generate a new CSV file with current and highest premorbid zBMIs, and DWS

values. Our simple tool substantially reduces the amount of time spent calculating DWS, and encourages investigators to use this new measure in their research.

4. Pilot data on DWS validity

The incremental validity of DWS (i.e., its ability to show stronger relationships than TWS to relevant variables) was piloted in a sample of patients with BN, recruited as part of a recently completed study of the behavioral and biological correlates of weight suppression. The study was approved by the Drexel University Institutional Review Board, and all participants provided informed written consent prior to study participation.

4.1. Participants

Participants were women between the ages of 18–45, with a BMI between 18.5 and 30.0 kg/m², who met full-threshold ($n = 81$) or subthreshold ($n = 10$) diagnoses for BN, per the DSM-5 (American Psychiatric Association, 2013). Individuals were recruited via geographically targeted online and paper advertisements in clinic, community, and campus settings in the greater Philadelphia and New York areas. They needed to be in treatment or treatment-seeking. Sample demographics are provided in Table 1.

4.2. Methods

All participants completed the Dieting and Weight History Questionnaire (DWHQ; Witt, Katterman, & Lowe, 2013) and the Eating Disorder Examination (EDE, v.16, Fairburn, Cooper, & O'Connor, 2008); at intake. The DWHQ is a 16-item, self-report questionnaire used to assess an individual's weight history (i.e., current weight, highest premorbid and postmorbid weights, lowest postmorbid weight, ages and heights at these weights), as well as dieting and overeating histories. The reliability of self-reported historical weights has been substantiated by several studies (Dahl & Reynolds, 2013; Casey et al., 1991; de Fine Olivarius et al., 1997), including one study that demonstrated a correlation of 0.85 between objectively measured weights and recalled weights 20 years later (Tamakoshi et al., 2003), thereby supporting the use of weight recall in the calculation of WS.

The EDE is a clinician-administered interview used to assess severity of ED symptoms over the past 28-days. It consists of four subscales (restraint, eating concerns, weight concerns, and shape concerns) and an averaged global score. The EDE also collects information on the frequency of binge episodes and compensatory behaviors over the past one and three months. Of the frequency data, only objective binge (OBEs) and vomiting episodes over the past three months were analyzed in this report. EDE interviewers demonstrated high inter-rater reliability, with κ 's ranging from 0.95 to 1.00.

Pearson bivariate correlations were run between TWS and DWS to determine their inter-relationship. As a preliminary test of the ability of DWS to show enhanced relationships with relevant ED measures, both TWS and DWS were independently correlated with EDE subscales and frequency items. Fischer z-tests were then used to determine whether the strength of the correlations observed between EDE scores and DWS were statistically superior to those observed between EDE scores and TWS. A Fischer r-to-z transformation calculator available on VassarStats (Lowry, 2020) was used to determine the difference

between correlation coefficients. DWS was entered first into the calculator, then TWS; therefore, a positive and significant z -value indicated that DWS was more strongly associated with the outcome variable, while a negative and significant z -value indicated that TWS was more strongly associated. All analyses were conducted in SPSS v.25 (IBM Corp. Released, 2017).

4.3. Results

TWS and DWS were significantly, though modestly, correlated ($r = 0.40, p < 0.001$). Correlations between TWS, DWS, and measures of ED psychopathology are reported in Table 2. DWS was significantly correlated (all p 's < 0.05) with several indices of ED-psychopathology (all EDE subscales, OBE's, vomiting episodes); however, associations with EDE subscales were not in the expected direction (i.e., correlations were negative). TWS was not correlated with any psychological measures (p 's > 0.05). Fisher's r -to- z transformations revealed the correlations between outcome measures and DWS were significantly stronger than those between outcome measures and TWS, for the following variables: EDE global score, EDE shape concerns, OBEs, and vomiting episodes (all p 's < 0.05 ; see Table 1).

4.4. Discussion

The moderate TWS-DWS correlation observed suggests that, in this sample of women with BN, a large majority of variance (84%) was not shared across the DWS and TWS constructs. Indeed, the pattern of correlations across both WS measures and ED-related psychopathology may be partially explained by the low shared variance: TWS showed no associations with bulimic psychopathology, while DWS demonstrated statistically significant associations across all indices of disordered eating. Taken together, this suggests that DWS may represent a more valid operationalization of the construct that weight suppression was originally developed to measure (Lowe, 1993). That is, if highest past weight is a proxy of an individual's potential to gain excessive weight, and if weight loss from that proxy captures the degree of the body's compensatory response to the weight loss, then quantifying past highest weight in relation to the age and height at which it was reached appears to better capture the strength and clinical impact of such responses.

The lack of association between TWS and bulimic psychopathology in this report and several other studies (Carter, McIntosh, Joyce, & Bulik, 2008; Dawkins, Watson, Egan, & Kane, 2013; Zunker et al., 2011) raises the possibility that nonfindings may have been partly due to the way in which WS was assessed (i.e., TWS). Consistent with past research using TWS, preliminary findings suggest that DWS is positively related to bulimic behaviors (i.e., bingeing, purging). Interestingly however, DWS appears to be *negatively* related to ED-related cognitive symptoms (i.e., weight/shape/eating concerns, dietary restraint); this is in contrast to previously demonstrated *positive* relations between TWS and ED cognitions. Although the negative correlations are novel, this may suggest that the farther an individual with BN is from their highest premorbid z -BMI, the less severe their disordered cognitions; alternatively, the closer they get to their highest premorbid z -BMI, the greater their ED cognitions. However further research is needed to determine the replicability of these novel findings.

This also warrants consideration of DWS differential predictive validity across populations. Because DWS's relevance may diminish as age increases, it may be a particularly useful indicator of psychopathology among youth and young adults with EDs. Additionally, although the current analyses were conducted in a sample of women with BN, past research has demonstrated an association between TWS and course of illness in AN (Berner et al., 2013, 2017; Haynos et al., 2016; Swenne et al., 2017). Thus, future research should aim to clarify the relation between DWS and AN psychopathology, and determine whether it is a superior proxy of illness compared to TWS.

Several shortcomings in the DWS tool warrant elaboration. While the reliability of self-reported historical weights has been supported by several studies (Casey et al., 1991; Dahl & Reynolds, 2013; de Fine Olivarius et al., 1997; Tamakoshi et al., 2003), the chance of error in historical recall remains. Furthermore, "premorbid," per the DWHQ, is based on self-report (i.e., "Before experiencing this first sign [of an ED], what was the highest weight you reached?"), and for individuals with multiple relapses, defined as prior to onset of first episode. Because the DWHQ is not a clinician-administered interview, error in patient interpretation and recall remains a possibility. Ideally, weight, height, and age histories are verified by a clinician or trained assessor using growth charts, to ensure optimal accuracy. Furthermore, it is possible that one's highest premorbid weight may occur at a different time than when one's weight is highest relative to their peers. For example, if an individual weighed 150 lbs at age 13, weighed 155 lbs at age 16, and developed an ED at age 17, their highest premorbid z-BMI would be at age 13, despite reaching a highest premorbid weight at age 16. Thus, it may be useful for assessors to gather additional information on when in a participant's life they were the most overweight, in order to increase the likelihood of establishing true highest past z-BMI.

Importantly, the current version of DWS UI relies on CDC growth charts derived from data gathered on individuals in the United States; in its present form, it will not always be extendable to international populations. However, the code provided in the Appendix can be used internationally, so long as lambda, mu, and sigma parameters corresponding to a particular region's demographic are available. Development of algorithms to particular racial and ethnic groups, and to particular countries, is an important consideration for future versions of the UI.

5. Conclusion

Given the ambiguities associated with the traditional measure of WS (Gorrell et al., 2019; Lowe et al., 2018; Schaumberg et al., 2016), DWS offers a promising solution. As previously discussed, DWS is more sensitive to the sex, age, and height at which individuals reach their highest premorbid weight. This is especially useful when studying ED populations, since many individuals develop their disorder in adolescence, when developmentally-appropriate increases in weight and height are still occurring. Indeed, preliminary work suggests that DWS may be a superior measure for predicting various ED outcomes.

Nevertheless, TWS has proven to be a consistently predictive of future weight gain and also related to various ED outcomes in past literature (cf., Lowe et al., 2018). Thus, we are not suggesting that the traditional measure of WS is no longer relevant; rather, we are proposing the implementation of a new, developmental measure (which, of course, requires measurement of highest premorbid weight and age at which it was reached), alongside the traditional measure. If DWS is eventually shown to consistently out-predict TWS, then it may indeed replace it.

In sum, DWS may serve as a more sensitive, clinically relevant construct with the potential to more fully unearth the impact of weight suppression on various ED characteristics and outcomes. Although our tool allows for simple and widespread use of the DWS measure, we suggest that investigators employ both measures of WS in their future research in order to further validate the use of DWS and compare it with the traditional WS measure.

Ethics statement

This study piloted a new construct on data collected as part of a multisite study (NIH Grant ID: MH095982). All subjects gave their informed consent for inclusion before they participated in the study. The study was conducted in accordance with the Declaration of Helsinki, and the protocol was approved by Drexel University Institutional Review Board (IRB ID: 1209001548R005).

Appendix

Developmental weight suppression, v.01 (09.10.2020)

This code is intended to accompany the manuscript “A new, developmentally sensitive measure of weight suppression,” (2021) by authors Singh, S., Apple, D.E., Zhang., F, Niu., X., and Lowe., M.R.

Step 1: Ensure CDC data is saved in your working directory. Import CDC data. Note that, because growth curves plateau after 20, the CDC rows for LMS values accompanying 240.5 months should be deleted from the saved reference dataset.

```
ref <- read.csv(file="CDC_reference.csv", header = TRUE)
```

Step 2: Split CDC data into male and female reference data sets. Male = 1, Female = 2.

```
refM <- ref[which(ref$cdc_sex==1), ] # Male reference dataset
```

```
refF <- ref [which(ref$cdc_sex==2), ] # Female reference dataset
```

Step 3: Import your target data. Make sure sex is coded appropriately: Male = 1, Female = 2. Here, target.csv is the dataset you are calculating developmental weight suppression (DWS) for.

```
upload_file <- read.csv("target.csv", header = TRUE)
```

Step 4: Remove any cases which have missing data from the target dataset.


```

data <- upload_file[complete.cases(upload_file), ]

n = dim(data)[1]

data$sex <- as.numeric (data$sex)

```

Step 5: Recode individuals who are >20 years old, to 20 years old, in the target dataset.

```

data$premorbid_age[data$premorbid_age>20]=20.0

data$current_age[data$current_age>20]=20.0

```

Step 6: Split your target data into sex datasets

```

ind1 <- which(data$sex==1)

dataM <- data[ind1, ] # Target data-subset of males

n1 <- dim(dataM) [1]

ind2 <- which(data$sex==2)

dataF <- data[ind2, ] # Target data-subset of females

n2 <- dim(dataF) [1]

```

Step 7: Calculate zBMIs for males. Note that target dataset age may fall equidistant from 2 CDC ages. In this case, we want to select the LMS values corresponding to the higher age, as this yields a more conservative zBMI estimate. Otherwise, we want to select LMS values that correspond with the CDC age closest to the target age.

```

if (dim(dataM)[1] > 0 ) {

  # Step 7a: Calculate premorbid zBMI for males.

  premorbid_zBMI <- NULL

  for (i in 1:n1M

    if(minCabs(refM$cdc_age_mos - dataM[i, ]$premorbid_age*12)) == 0.5){ # If
the # target dataset age is equidistant from 2 CDC ages.

      p <- which.min(abs(refM$cdc_age_mos - dataM[i, ]$premorbid_age*12))

      m <- p+1

      premorbid_temp <- ((dataM$highest_premorbid_BMI[i] / refMQn, ]
$mu )'refM[m, ]$lambda - 1)/(refM[m, ]$lambda * refM[m, ]$sigma)

```

```

    } else {

      p <- which.min(abs(refH$cdc_age_mos - dataM[i, ]$premorbid_age*12)) #
      Otherwise, # match LMS values to closet age.

      premorbid_temp <- ((dataM$highest_premorbid_BMI[i] / refM[m, ]
      $mu )^refM[m, ]$lambda - 1)/(refM[m, ]$lambda * refM[m, ]$sigma)

    }

    premorbid_zBMI <- c(premorbid_zBMI, premorbid_temp)

  }

  # Step 7b: Calculate current zBMI for males.

  current_zBMI=NULL

  for (i in 1:n1){

    if(min(abs(refM$cdc_age_mos - dataM[i, ]$current_age*12)) == 0.5H # If
    the target # dataset age is equidistant from 2 CDC ages.

      p <- which.min(abs(refM$cdc_age_mos - dataM[i, ]$current_age*12))

      m <- p+1

      current_temp <- ((dataM$current_BMI[i] / refM[m, ]$mu )^refM[m, ]
      $lambda - 1)/ (refM[m, ]$lambda * refM[m, ]$sigma)

    } else {

      p <- which.min(abs(refM$cdc_age_mos - dataM[i, ]$current age+12)) #
      Otherwise, # match LMS values to closet age.

      current_temp <- ((dataM$current_BMI[i] / refM[p, ]
      $mu )^refM[p, ]$lambda - 1)/ (refM[p, ]$lambda * refM[p, ]$sigma)

    }

    current_zBMI <- c(current_zBMI, current_temp)

  }

  # Step 7c: Combine and label current and premorbid zBMIs into a male zBMI
  data-subset.

```

```

dataM <- cbind(dataM, premorbid_zBMI, current_zBMI)

names(dataM$premorbid_zBMI) <- "premorbid_zBMI"

names(dataM$current_zBMI) <- "current_zBMI"

}

```

Step 8: Calculate zBMIs for females. Note that target dataset age may fall equidistant from 2 CDC ages. In this case, we want to select the LMS values corresponding to the higher age, as this yields a more conservative zBMI estimate. Otherwise, we want to select LMS values that correspond with the CDC age closest to the target age.

```

if( dim(dataF)[1] > 0 ) {

  # Step 8a: Calculate premorbid zBMI for females.

  premorbid_zBMI=NULL

  for ( i in 1:n2){

    if(min(abs(refF$cdc_age_mos - dataF[i, ]$premorbid_age*12)) == 0.5){ # If
the # target dataset age is equidistant from 2 CDC ages.

      p <- which.min(abs(refF$cdc_age_mos - dataF[i, ]$premorbid_age*12))

      m <- p+1

      premorbid_temp <- ((dataF$highest_premorbid_BMI[i] / refF[m, ]
$mu )^refF[m, ]$lambda - 1)/(refF[m, ]$lambda *refF[m, ]$sigma)

    } else {

      p <- which.min(abs(refF$cdc_age_mos - dataF[i, ]$premorbid_age*12)) #
Otherwise, # match LMS values to closet age.

      premorbid_temp <- ((dataF$highest_premorbid_BMI[i] / refF[p, ]
$mu )^refF[p, ]$lambda - 1)/(refF[p, ]$lambda * refF[p, ]$sigma)

    }

    premorbid_zBMI <- c(premorbid_zBMI, premorbid_temp)

  }

  # Step 8b: Calculate current zBMI for females.

```

```

current_zBMI=NULL

for (i in 1:n2){

  if(min(abs(refF$cdc_age_mos - dataF[i, ]$current_age*12)) == 0.5){ # If
the target # dataset age is equidistant from 2 CDC ages.

    p <- which.min(abs(refF$cdc_age_mos - dataF[i, ]$current_age*12))

    m <- p+1

    current_temp <- ((dataF$current_BMI[i] / refF[m, ]
$mu )^refF[m, ]$lambda - 1)/ (refF[m, ]$lambda * refF[m, ]$sigma)

  } else {

    p <- which.min(abs(refF$cdc_age_mos - dataF[i, ]$current_age*12)) #
Otherwise, # match LMS values to closet age.

    current_temp <- ((dataF$current_BMI[i] / refF[p, ]$mu )^refF[p, ]
$lambda 1)/ (refF[p, ]$lambda * refF[p, ]$sigma)

  }

  current_zBMI <- c(current_zBMI, current_temp)

}

# Step 8c: Combine and label current and premorbid zBMIs into a female
zBMI data-subset.

dataF <- cbind(dataF, premorbid_zBMI, current_zBMI)

names(dataF$premorbid_zBMI) <- "premorbid_zBMI"

names(dataF$current_zBMI) <- "current_zBMI"

}

```

Step 9: Combine male and female zBMI data-subsets into a final dataset.

```

if ( dim(dataM) [1] == 0){          # If no males, then append females to final dataset
  output_file <- dataF } else
  if(dim(dataF) [1] == 0){        # If no females, then append males to final dataset
    output_file <- dataF } else {

```

```

output_file <- rbind(dataM, dataF) # If males and females, append all to final dataset
}

```

Step 10: Calculate DWS, and recode negatives to 0.

```

output_file$DWS <- (output_file$premorbid_zBMI - output_file$current_zBMI)

output_file$DWS[output_file$DWS<0]=0

```

References

- Accurso EC, Lebow J, Murray SB, Kass AE, & 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), 1–6. [PubMed: 26855778]
- Association AP (2013). *Diagnostic and statistical manual of mental disorders (DSM-5®)* (5 ed.). American Psychiatric Publishing.
- Berner L, Feig EH, Witt AA, & Lowe MR (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.
- Berner L, Shaw J, Witt A, & Lowe MR (2013). Interaction of weight suppression and body mass index in the prediction of symptomatology and outcome in anorexia nervosa. *Journal of Abnormal Psychology*, 122, 694–708. [PubMed: 24016010]
- Butryn ML, Juarascio A, & Lowe MR (2011). The relation of weight suppression and BMI to bulimic symptoms. *International Journal of Eating Disorders*, 44(7), 612–617.
- Butryn ML, Lowe MR, Safer DL, & Agras WS (2006). Weight suppression is a robust predictor of outcome in the cognitive-behavioral treatment of bulimia nervosa. *Journal of Abnormal Psychology*, 115(1), 62–67. [PubMed: 16492096]
- Carter FA, McIntosh VV, Joyce PR, & Bulik CM (2008). Weight suppression predicts weight gain over treatment but not treatment completion or outcome in bulimia nervosa. *Journal of Abnormal Psychology*, 117(4), 936. [PubMed: 19025238]
- Casey VA, Dwyer JT, Berkey CS, Coleman KA, Gardner J, & Valadian I (1991). Long-term memory of body weight and past weight satisfaction: A longitudinal follow-up study. *American Journal of Clinical Nutrition*, 53(6), 1493–1498.
- Centers for Disease Control and Prevention. (2009, 8 4). Percentile Data Files with LMS Values. U.S. Department of Health and Human Services. https://nam03.safelinks.protection.outlook.com/?url=https%3A%2F%2Fwww.cdc.gov%2Fgrowthcharts%2Fpercentile_data_files.htm&data=04%7C01%7Cr.pio%40elsevier.com%7Cf874fd0c46e441a18ccc08d8fb63f0bb%7C9274ee3f94254109a27f9fb15c10675d%7C0%7C0%7C637535754094618379%7CUnknown%7CTWFpbGZsb3d8eyJWIjoiMC4wLjAwMDAiLCJQIjoiV2luMzIiLCJBTiI6IklhaWwiLCJXVCi6Mn0%3D%7C3000&sdata=e79odPcpYlaMXnCJaKmj0PKjYnNxnJuWs%2F4q69Hec%3D&reserved=0
- Cole TJ, & Green PJ (1992). Smoothing reference centile curves: the LMS method and penalized likelihood. *Statistics in Medicine*, 11(10), 1305–1319. [PubMed: 1518992]
- Dahl AK, & Reynolds CA (2013). Accuracy of recalled body weight—a study with 20-years of follow-up. *Obesity*, 21(6), 1293–1298. [PubMed: 23913738]
- Dawkins H, Watson HJ, Egan SJ, & Kane RT (2013). Weight suppression in bulimia nervosa: Relationship with cognitive behavioral therapy outcome. *International Journal of Eating Disorders*, 46(6), 586–593.
- Fairburn CG, Cooper Z, & O'Connor M (2008). *Eating disorder examination edition 16.0D*. In *Cognitive behavior therapy and eating disorders* (pp. 265–306). New York, NY: Guilford Press.

- de Fine Olivarius N, Andreasen AH, & Løken J (1997). Accuracy of 1-, 5- and 10-year body weight recall given in a standard questionnaire. *International Journal of Obesity and Related Metabolic Disorders*, 21(1).
- Gorrell S, Reilly EE, Schaumberg K, Anderson LM, & Donahue JM (2019). Weight suppression and its relation to eating disorder and weight outcomes: A narrative review. *Eating Disorders*, 27(1), 52–81. 10.1080/10640266.2018.1499297 [PubMed: 30040543]
- Haynos AF, Snipes C, Guarda A, Mayer LE, & Attia E (2016). Comparison of standardized versus individualized caloric prescriptions in the nutritional rehabilitation of inpatients with anorexia nervosa. *International Journal of Eating Disorders*, 49(1), 50–58.
- IBM Corp Released. (2017). IBM SPSS statistics for windows, version 25.0. Armonk, NY: IBM Corp.
- Keel PK, Bodell LP, Haedt-Matt AA, Williams DL, & 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.
- Keel PK, & Heatherton TF (2010). Weight suppression predicts maintenance and onset of bulimic syndromes at 10-year follow-up. *Journal of Abnormal Psychology*, 119(2), 268–275. [PubMed: 20455599]
- Lowe MR (1993). The effects of dieting on eating behavior: A three-factor model. *Psychological Bulletin*, 114(1), 100. [PubMed: 8346324]
- Lowe MR, Piers AD, & Benson L (2018). Weight suppression in eating disorders: A research and conceptual update. *Current Psychiatry Reports*, 10.1007/s11920-018-0955-2
- Lowry R (n.d.). VassarStats: Website for Statistical Computation. Retrieved September 10, 2020, from <http://vassarstats.net/rdiff.html>.
- Piers AD, Espel-Huynh HA, & Lowe MR (2019). The independent and interacting effects of weight suppression and admission body mass index on treatment weight change in patients with anorexia nervosa or bulimia nervosa.
- Schaumberg K, Anderson LM, Reilly EE, Gorrell S, Anderson DA, & Earleywine M (2016). Considering alternative calculations of weight suppression. *Eating Behaviors*, 20, 57–63. [PubMed: 26643591]
- Shaw JA, Herzog DB, Clark VL, Berner LA, Eddy KT, Franko DL, & Lowe MR (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(4), 512–523.
- Stice E, Rohde P, Shaw H, & Desjardins C (2020). Weight suppression increases odds for future onset of anorexia nervosa, bulimia nervosa, and purging disorder, but not binge eating disorder. *American Journal of Clinical Nutrition*, 112(4), 941–947.
- Swenne I, Parting T, & Salonen Ros H (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. 10.1186/s12888-017-1486-9 [PubMed: 28915806]
- Tamakoshi K, Yatsuya H, Kondo T, Hirano T, Hori Y, Yoshida T, et al. (2003). The accuracy of long-term recall of past body weight in Japanese adult men. *International Journal of Obesity*, 27(2), 247–252. [PubMed: 12587006]
- The Nemours Foundation. (2015, June 14). Nemours: Understanding Puberty. Retrieved September 10, 2021, from <https://www.nemours.org/patientfamily/khlibrary/articles/understanding-puberty.html>.
- Wildes JE, & Marcus MD (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. 10.1016/j.brat.2012.02.006 [PubMed: 22398152]
- Witt AA, Katterman SN, & Lowe MR (2013). Assessing the three types of dieting in the three-factor model of dieting. The dieting and weight history questionnaire. *Appetite*, 63, 24–30. [PubMed: 23220357]
- Zanetti T, Santonastaso P, Sgaravatti E, Degortes D, & Favaro A (2013). Clinical and temperamental correlates of body image disturbance in eating disorders. *European Eating Disorders Review*, 21(1), 32–37. [PubMed: 22807118]

Zunker C, Crosby RD, Mitchell JE, Wonderlich SA, Peterson CB, & Crow SJ (2011). Weight suppression as a predictor variable in treatment trials of bulimia nervosa and binge eating disorder. *International Journal of Eating Disorders*, 44(8), 727–730.

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Table 1

Participant demographics and descriptives.

	<i>M (SD)</i>
Age (yrs)	25.2 (5.6)
Length of illness (yrs)	9.2 (6.4)
Current weight (lbs)	139.3 (25.0)
TWS (lbs)	17.3 (16.0)
DWS	0.8 (0.6)
	<i>n (%)</i>
% individuals with a highest premorbid weight before 21 yrs	85 (93.4)
<i>Race</i>	
White	55 (60.4)
African American	9 (9.9)
Asian	16 (17.6)
Other	11 (12.1)
<i>Ethnicity</i>	
Hispanic/Latina	14 (15.4)
<i>Diagnosis</i>	
Threshold BN	81 (89.0)
Subthreshold BN	10 (11.0)

Note. TWS = traditional weight suppression; DWS = developmental weight suppression; BN = bulimia nervosa.

Table 2

Correlations between traditional and DWS, and outcome measures.

	<u>TWS</u>	<u>DWS</u>	<u>Fischer's r-to-z test</u>
	<i>r</i>	<i>r</i>	<i>z</i>
DWS	0.40 ^{**}	–	–
EDE global score	<0.01	–0.31 ^{**}	2.03 [*]
EDE shape concerns	0.02	–0.30 ^{**}	1.95 [*]
EDE weight concerns	–0.02	–0.27 ^{**}	1.63
EDE eating concerns	0.01	–0.21 [*]	1.29
EDE restraint	<0.01	–0.21 [*]	1.33
EDE OBEs, past 3mo.	0.04	0.33 ^{**}	1.98 [*]
EDE vomiting, past 3mo.	–0.01	0.33 ^{**}	2.13 [*]

Note. A positive z-score indicates that r_{DWS} is greater than r_{TWS} .

* $p < 0.05$;

** $p < 0.01$.