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Weight suppression is a risk factor for eating disorders: Implications for etiology, maintenance, and treatment

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The study by Stice et al. (1), published in this issue of the Journal, is the first to examine weight suppression (WS) as a risk factor for the development of eating disorders (EDs). Stice's group has produced a long line of similar studies examining various psychological and behavioral risk factors for the development of EDs (2); this is the first to examine weight suppression, defined as the difference between a person's highest past weight (at their adult height) and their current weight. The authors found that, among >1000 young women, WS was a risk factor for the development of threshold and subthreshold anorexia nervosa, bulimia nervosa, purging disorder, and any eating disorder [but not binge eating disorder (BED)]. The methodological rigor, large sample size, and prospective nature of the study boost confidence in its findings.

Nearly all research on WS and eating disorders examines the maintenance of the disorder or relations between WS and characteristics or symptoms of those who already have EDs (3–5). Two previous studies in a large sample of college students found that those with higher WS were more likely to develop a bulimic syndrome over 10 and 20 y (6, 7) and, among those with this syndrome at baseline, were more likely to still be suffering from it 10 y later (7). The current study has the added strengths of confirming ED diagnoses via interviews and examining the role of WS in the development of several EDs or of any ED. This means that solid evidence now exists that the single variable of WS is relevant to both the etiology and maintenance (4, 5) of all EDs except for binge eating disorder. Further, we have proposed that WS has a mechanism of action that could account for its impact on the perpetuation of EDs once they develop: higher WS consistently predicts greater future weight gain among those with EDs (4). Such weight gain is anathema to those with EDs, likely causing them to redouble efforts to lose weight or prevent further weight gain. All this evidence indicates that existing psychological models of EDs are incomplete because they do not incorporate the influence of past and current weight on ED psychopathology. Further, aside from weight restoration in those with anorexia nervosa, it suggests that current treatments are suboptimal because they do not explicitly address level of WS in the assessment or treatment of individual ED cases (6, 8).

Stice et al.'s study has several additional implications. First, restrained eating was controlled for in the analyses, and WS was found to be unrelated to measures of dietary restraint, BMI, thin-ideal internalization, body dissatisfaction, and negative affect. This means that WS, a major source of ED psychopathology according to this study and many others (4), is not being accounted for in existing theories of ED etiology and maintenance (Cognitive Behavior Therapy-Enhanced, Dialectical Behavior Therapy, Integrative Cognitive-Affective Therapy, etc.). Psychological influences on EDs (e.g., internalization of the thin ideal; cognitive rules regarding food intake) may themselves impact ED symptoms but also sometimes lead to actual weight loss, which represents an additional source of ED psychopathology (4, 9). By the same token, weight loss speeds weight regain (4), further fueling internalization of the thin ideal, cognitive eating rules, etc. By focusing only on the psychological side of this self-perpetuating cycle, current therapies are working with one hand tied behind their backs.

Second, the Stice et al. study makes clear that there are 2 types of food restriction (WS and restrained eating) that are independent risk factors for the development of EDs. Further, Stice and colleagues have shown elsewhere that restrained eaters, unlike weight suppressors, do not restrict their intake below their energy needs. Thus, although all models of EDs posit that "dieting" is central to ED psychopathology, and that reducing dieting is a critical part of treatment, none of them do so in a way that accounts for multiple forms of food restriction (10, 11). This means, by definition, that strategies for addressing food restriction and its potential effects on weight have not been sufficiently incorporated into ED treatments.

Third, the baseline level of WS (∼4.5 kg) almost doubled for those who developed an ED at the 3-y follow-up, with the exception of those who developed BED (whose WS went from 4.8 to 5.6 kg). This indicates that *1*) weight loss may have little to do with the development of BED and *2*) the role of actual weight loss in 2 disorders involving binge eating [BED and bulimia nervosa (BN)] may be quite different. The development of BED may involve a hyperactive appetitive drive in an environment with

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Abbreviations used: BED, binge eating disorder; BN, bulimia nervosa; ED, eating disorder; WS, weight suppression.

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omnipresent palatable foods; significant weight loss, if it occurs at all, may be a consequence of binge-based weight gain. The lower level of WS in those with BED may also help explain why treatment outcomes are better in BED than in BN (9).

Fourth, when highest past weight was used in place of WS as a predictor, it also emerged as a risk factor for EDs, although not as robustly as WS. This finding is an important reminder that reaching an elevated premorbid BMI could itself represent a risk for developing an ED (12). This could occur because those with higher BMIs at a given point in time are most likely to subsequently experience the largest increase in their weight (13). If such individuals are highly sensitive about their weight, they may engage in radical efforts to lose weight, fueling their ED risk.

In sum, it would be desirable in future research to *1*) consider the role of both restrained eating and WS when investigating factors contributing to the etiology or maintenance of EDs and *2*) start investigating how individual differences in WS might be used to modify treatment to better tailor interventions to the weight histories of individual patients (8).

MRL is a paid advisor to the Renfrew Center for Eating Disorders.

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