

Short Communication

Stress-induced eating in restrained eaters may not be caused by stress or restraint

Michael R. Lowe*, Tanja V.E. Kral

Department of Psychology, Drexel University, Mail Stop 626, 245 N 15th Street, Philadelphia, PA 19102, USA

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Abstract

Restrained eaters tend to increase and unrestrained eaters to decrease their food intake when stressed. This relationship, though robust, does not appear to be caused by restrained eating or by stress per se. For restraint, evidence indicates that none of the common operationalizations of restraint can account for restraint-related effects that have been examined to date. It is therefore unlikely that restraint is responsible for stress-induced eating in restrained eaters. Rather, behavioral and physiological data suggest that restrained eating may be a proxy risk factor for vulnerability to weight gain. For stress, a variety of minimally stressful perturbations (e.g. nonthreatening cognitive loads) have been shown to elicit increased intake in restrained eaters. Thus, the negative affect created by manipulations used to create stress (e.g. scary movies, failure at a task) does not appear to be necessary to provoke overeating. An adequate explanation for stress-induced eating in restrained eaters remains elusive.

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Restrained eating refers to the volitional effort to restrict food intake to control body weight. In the research literature on individual differences in appetite, one of the most robust findings is the opposing responses of restrained and unrestrained eaters to stress. Restrained eaters tend to increase, and unrestrained eaters tend to decrease, their consumption in high relative to low stress conditions (Heatherton and Baumeister, 1991). Most studies on this topic assume that restrained eating is causally connected to restrained eaters' response to stress. Many studies also assume that stress per se is responsible for the elicitation of restrained and unrestrained eaters' divergent eating responses. The purpose of this brief review is to explain why neither of these assumptions may be warranted.

Critique of the restraint model

The restraint model has been used to explain not just stress-induced eating, but to account for the effects of

dieting on a variety of outcomes including salivary responsiveness, distractibility, emotionality, counterregulatory eating and others (Herman and Polivy, 1980). These diverse outcomes will be referred to as 'restraint-related effects' (RREs) in this paper. We have previously demonstrated that restrained eating is a multi-dimensional, not a monolithic, construct (Lowe, 1993). Thus, the assumption that restrained eating causes abnormalities in appetite can be evaluated by decomposing the construct into three types of dieting (i.e. current dieting, frequency of dieting and overeating, and weight suppression) subsumed by this construct.

Interestingly, although the rationale for the three-factor model of dieting was derived from evidence that different types of dieting are associated with different effects on behavior (Lowe, 1993), the three dieting types closely reflect the three explanations Herman and Polivy have offered for RREs over the past 30 years. They (Herman and Polivy, 1975) initially suggested that the effects of restraint on eating behavior were due to a deviation of body weight from a body weight set point; this construct is similar to the three-factor model's weight suppression factor. They then suggested that RREs were due to the breaching of a putative 'diet boundary' in the boundary model of eating regulation (Herman and Polivy, 1984); this construct is similar to

* Corresponding author.

E-mail address: lowe@drexel.edu (M.R. Lowe).

the current dieting factor from the three-factor model. Finally, they suggested that restraint effects were due to ‘unsuccessful dieting’ (Heatherton, Herman, Polivy, King, & McGree, 1988); this construct is similar to the frequency of dieting and overeating factor. Therefore, in evaluating the ability of each of the three factors to account for restraint-related patterns in appetite, we are also evaluating the three explanations Herman and Polivy have offered for these patterns over the years.

Much of the evidence reviewed below on the relationship between the three types of dieting and RREs is based on studies using the forced preload paradigm. Using this paradigm (which involves testing subjects’ food intake after consuming or not consuming a high-calorie preload), restrained eaters have been found to increase, and unrestrained eaters to decrease, their intake in the preload relative to the no preload condition (Herman and Polivy, 1984). The tendency of restrained eaters to increase their consumption after a preload is called ‘counterregulatory eating’. We recognize that conclusions reached about the role of dieting in counterregulatory eating may not hold true for other types of RREs and we address this question at the end of the review.

Factor I: current dieting

We begin by considering whether current dieting might account for RREs. The first eating pattern studied by Herman and Polivy was counterregulation, or the tendency for restrained eaters to eat more with than without a preload and for unrestrained eaters to show the opposite eating pattern. Most restrained eaters are not currently dieting to lose weight (Lowe, 1993). Those who are currently dieting score higher on the Restraint Scale than restrained nondieters (Lowe and Timko, 2004) yet demonstrate behavioral regulation, rather than counterregulation, following a high-calorie preload (Lowe, 1995). Neither overweight restrained eaters nor overweight dieters engage in counterregulatory eating but obese individuals joining a weight loss program (but not yet dieting) do show the counterregulatory eating pattern (McCann, Perri, Nezu, & Lowe, 1992). Among individuals with bulimia nervosa, those who had been dieting frequently in the recent past engaged in binge eating significantly less often than those who dieted infrequently (Lowe, Gleaves, & Murphy-Eberenz, 1998). When dieting is manipulated and documented through weight loss, it is associated with reduced, rather than increased, levels of overeating (Foster, Wadden, Kendall, Stunkard, & Vogt, 1996) and bulimic symptoms (Presnell and Stice, 2003). Thus, although the examples cited represent only some of the RREs documented in past research, this evidence contradicts the idea that ‘dieting to lose weight’ (whether that state is measured through self-report or actual weight loss) accounts for RREs.

Research also suggests that none of the three most frequently used restraint measures (the Restraint Scale and the restraint subscales from the Dutch eating behavior questionnaire and the eating inventory) reflect hypo-caloric dieting. The findings on RMR and TEE in restrained and unrestrained eaters are mixed (as described below), but even if restrained eaters are more energy efficient than unrestrained eaters, there is no evidence that their energy intake relative to their energy needs places them in a state of energy deprivation. The only study that compared restrained and unrestrained eaters (using various restraint measures) on unobtrusive measures of food intake (thereby obviating potential problems with food intake underreporting) found no differences between them in four separate studies (Stice, Fisher, & Lowe, 2004). If restraint scales do not reflect energy intake that is below energy needs—which is usually what dieting refers to—it is unlikely that dieting can account for RREs.

Factor II: frequency of dieting and overeating

The second factor of the three-factor model of dieting essentially recapitulates Heatherton and colleagues’ notion that chronic, unsuccessful dieting is responsible for RREs. This hypothesis has been supported by logical argument more than by empirical evidence (Heatherton et al., 1988).

The evidence for a relationship between weight cycling and binge eating is mixed but even studies that find a relationship between the two could just as well reflect binge eating causing dieting than the reverse. More importantly, the one study that prospectively examined the relation of dieting to binge eating in obese individuals (before they lost weight and 5 years later when they had experienced a full weight cycle by gaining back almost all of the weight they lost) found that binge eating was significantly reduced relative to baseline levels (Foster et al., 1996). Similarly, Wadden, Foster, Sarwer, Anderson, Gladis and Sanderson (2004) found little evidence that strict weight loss dieting causes binge eating problems among obese individuals without any history of binge eating. Lowe and Timko (2004) recently reported that current dieters showed significantly higher levels of past weight cycling than restrained nondieters. This finding demonstrates that weight cycling cannot account for counterregulatory eating because current dieters significantly reduce, rather than increase, their eating following a preload (Lowe, 1995). The findings reviewed in this section indicate that it is unlikely that weight cycling (or frequency of dieting and overeating or unsuccessful dieting) is responsible for RREs.

Factor III: weight suppression

Research also does not support the possibility that RREs are due to restrained eaters’ suppression of their weight

below its highest point ever. Although restrained eaters do report a discrepancy between their current and highest previous weight that is greater than unrestrained eaters', most restrained eaters show relatively low levels of weight suppression (Lowe, 1984). Results from the National Weight Control Registry, which has thoroughly examined the psychological status of a large group of long-term weight suppressors, found that these individuals show no elevation in binge eating or emotional distress relative to appropriate control groups (Wing and Hill, 2001). Other studies have found that weight suppression is associated with the regulation of eating following a preload and with reduced preferences for sweet taste (reviewed in Lowe, 1993). Therefore, available evidence on weight suppression indicates that it probably is not responsible for producing binge eating or counterregulatory eating among restrained eaters. (The only exception to this may be the binge eating that begins after individuals with incipient bulimia nervosa start to binge after losing a great deal of weight—Lowe et al., 1998).

Since, most of the research on the three-factor model of dieting has been based on the forced preload paradigm, it is possible that the preceding critique would not apply if other RREs (e.g. stress-induced eating, heightened salivary responsiveness) were examined. However, available evidence suggests otherwise. For example, while the Restraint Scale has been associated with increased salivary output (Klajner, Herman, Polivy, & Chhabra, 1981), weight loss dieting reduces salivary output to food cues (Durrant, 1981; Rosen, 1981). In addition, weight loss dieting has been shown to produce a reduction in bulimic symptoms (Presnell and Stice, 2003) and dieting to maintain weight has been shown to produce a reduction in bulimic symptoms and negative affect (Stice, Presnell, Groesz & Shaw, 2005). Thus, though theoretically possible, it would not be very parsimonious to suggest that restraint does not account for counterregulatory eating but does account for other RREs.

Is restrained eating a proxy for obesity-proneness?

Based on the foregoing review, it appears that several of the problematic appetitive and behavioral effects documented in research with the Restraint Scale cannot be accounted for by any of the most common operationalizations of restrained eating. It remains possible, however, that dieting in one or more forms uncovers a vulnerability to overeating only in certain predisposed individuals. A similar point has been made in relation to bulimia nervosa—that is, while millions of young women go on diets to lose weight, only a small fraction develop serious bulimic symptomatology as a consequence (Wilson, 2002). This alternative suggestion, however, also appears to be implausible. Restrained eaters, bulimic individuals, and obese individuals would all have to be considered as likely candidates to possess

a vulnerability to overeat when dieting. Yet the evidence for all three groups points to a reduction, rather than to an increase, in overeating when these groups diet (for restrained eaters, see Lowe (1995), for bulimic individuals, see Lowe et al., 1998; for obese individuals, see Foster et al., 1996). Conversely, young, healthy men would be viewed as unlikely to have a vulnerability to developing overeating when dieting, yet many of Keys, Brozek, Henschel, Mickelson, and Taylor (1950) young male conscientious objectors developed binge eating and other forms of eating disturbance when they were put on a semi-starvation diet and lost about 25% of their body weight. Therefore, logic compels us to consider the possibility that restraint is primarily a result of, rather than a cause of, the behavioral patterns with which it has consistently been associated.

There is evidence consistent with the idea that restraint is a proxy of obesity-proneness. Several studies have found that different measures of restraint prospectively predict weight gain rather than weight loss (French, Jeffery, & Wing, 1994; Klesges, Isbell, & Klesges, 1992; Stice et al., 1999). Furthermore, Stice et al. (1999) found that, controlling for temporal stability, bulimic behavior predicted future increases in restraint but restraint did not predict future increases in bulimic behavior. If restraint reflects appetitive responsiveness to the food environment (and therefore, susceptibility to weight gain), then the literature on restrained eating and the older literature on obese individuals' responsiveness to environmental cues may reflect the same phenomenon. This is precisely the conclusion that one of the founders of research on external responsiveness reached more than 20 years ago. Rodin (1981) suggested that restraint 'is only a descriptive term and not a mechanism. Restraint is what some people do if they feel compelled by external cues' (p. 364).

Are restrained eaters physiologically predisposed toward weight gain?

If restrained eating mostly reflects efforts to prevent or reverse weight gain (rather than a drive for thinness, as Polivy and Herman (1987) have long suggested), then it is possible that the predisposition toward weight gain is manifested physiologically, not just appetitively. If restrained eaters have physiological characteristics that predispose them to weight gain, then dietary restraint could be regarded as a response to a positive energy balance (and possibly to weight gain), rather than a cause of overeating or weight gain. A variety of studies that have compared restrained and unrestrained eaters on various metabolic parameters can be used to answer this question.

There is some evidence that the resting metabolic rate of restrained eaters is significantly lower compared to unrestrained eaters (Gingras, Harber, Field, & McCargar,

2000; Platte, Wurmser, Wade, Mercheril, & Pirke, 1996; Poehlman, Viers, & Detzer, 1991). However, other studies failed to find a significant difference in resting energy expenditure (Bathalon, Hays, McCrory, Vinken, Tucker and Greenberg, 2001; Beiseigel and Nickols-Richardson, 2004; Keim and Horn, 2004; Lawson, Williamson, Champagne, DeLany, Brooks and Howat, 1995). These mixed findings also hold true for total energy expenditure (TEE): while Bathalon et al. (2001) found that there was no significant difference in TEE between restrained and unrestrained eaters, Tuschl, Platte, Laessle, Stichler, and Pirke (1990) did show a lower TEE in restrained eaters. This latter difference, however, only emerged after controlling for body composition.

The results of studies on other physiologic parameters have been more consistent. They have identified increased levels of fasting plasma triglycerides (Laessle et al., 1989), elevated free fatty acids (Hibscher and Herman, 1977), reduced levels of leptin (Laessle et al., 2000; Von Prittwitz et al., 1997), reduced fasting insulin levels (Keim and Horn, 2004; Pirke, Tuschl, Spyra, Laessle, Schweiger and Broocks, 1990), and increased levels of cephalic phase insulin release (Teff and Engelman, 1996) in restrained compared to unrestrained eaters. Also, Keim and Horn (2004) found that carbohydrate oxidation after a high-carbohydrate meal was higher in restrained than unrestrained eaters (though no differences were found in fat oxidation) and Verboeket-van de Venne, Westerterp, and ten Hoor (1994) found that 24-h carbohydrate oxidation was higher and 24-h fat oxidation was lower in restrained subjects. These characteristics would favor the storage (rather than the oxidation) of fat in restrained relative to unrestrained eaters, a tendency that would favor greater weight gain over time in restrained eaters.

Tataranni and Ravussin (2002) have identified a low metabolic rate, low rates of fat oxidation (which could be reflected in higher triglyceride levels), insulin sensitivity, a low sympathetic nervous system activity, and low plasma leptin concentrations as metabolic predictors for weight gain.

Salivary responses to food stimuli can be considered a quantitative physiological index of appetite (Wooley and Wooley, 1973), since salivary output has been shown to be associated with subjects' ratings of hunger and food appeal. The findings on salivary output and salivary flow rate in response to food cues are inconsistent in that some studies have found a significant positive relationship between dietary restraint and salivation (Klajner et al., 1981; Legoff and Spigelman, 1987; Tepper, 1992), while others found no such relationship (Bulik, Lawson, & Carter, 1996; Frijters, 1984; Mitchell and Epstein, 1996; Rogers and Hill, 1989). Interestingly, two studies (Durrant, 1981; Rosen, 1981) investigated the effects of actual food restriction rather than restraint status measured by questionnaire. Both of these studies found that actual food restriction was associated with a reduction in

salivation to food cues, which indicates that (a) positive relationships between restraint and salivary output, when found, are not due to energy-restricted dieting, and (b) the inconsistent findings on restraint and salivation may be due to the fact that restrained eating and actual dieting have opposite associations with salivary responding (Lowe, 1993).

Both salivary and urinary cortisol, biological markers of stress, have been shown to be either increased in individuals high in dietary restraint (Anderson, Shapiro, Lundgren, Spataro, & Frye, 2002; McLean, Barr, & Prior, 2001) or not different between restrained and unrestrained individuals (Beiseigel and Nickols-Richardson, 2004). Elevated cortisol levels have been shown to exert a stimulatory effect on energy intake (Dallman, la Fleur, Pecoraro, Gomez, Houshyar and Akana, 2004). In particular, a positive relationship has been found between change in cortisol levels in response to a stressor and intake of high-fat, high-sugar food during stress (Epel, Lapidus, McEwen, & Brownell, 2001). A tendency to overconsume calories when stressed could contribute to a chronic positive energy balance and to weight gain over time. Support for this possibility was found in a recent study that found that increased levels of negative emotionality in childhood prospectively predicted weight gain in adulthood (Pulkki, Elovainio, Kivimaki, Raitakari, and Keltikangas-Jarvinen, *in press*).

Although findings within particular physiological domains (e.g. energy expenditure) are not always consistent, the pattern of results across physiological measures indicate that restrained eaters display physiological differences which would increase their susceptibility to weight gain due to increased energy intake, increased storage of ingested energy, or both. This may explain, at least in part, the aforementioned findings that measures of restraint prospectively predict weight gain, not weight loss (French et al., 1994; Klesges et al., 1992; Stice et al., 1999). Alternatively, it could be hypothesized that these differences may be due to recent or ongoing weight loss. However, even if the lower self-reported intake of restrained eaters (defined by the DEBQ or EI) was assumed to accurately reflect actual intake (rather than underreporting), the degree of caloric restriction does not appear to be sufficient to evoke regulatory physiological adjustments. Furthermore, the fact that restraint measures predict weight gain rather than weight loss and that a study of unobtrusively-measured energy intake found no differences between restrained and unrestrained eaters (Stice et al., 2004) suggest that restrained eaters are more likely to be in positive than negative energy balance. These findings suggest that the physiological differences between restrained and unrestrained eaters most likely reflect restrained eaters' predisposition toward weight gain rather than a regulatory response to weight loss.

Does stress per se cause increased eating in restrained eaters?

The great majority of research on affect-induced eating in restrained eaters has involved the introduction of some kind of negative emotional stimulus (e.g. horror films, apparent failure at an important task, anticipation of public speaking; for a review, see Greeno and Wing, 1994). These manipulations reflect the assumption that negative emotional arousal per se is responsible for increased eating in restrained eaters. In fact, Heatherton, Herman and Polivy (1992) have suggested not only that distress is required but that ‘the sort of distress that leads to disinhibition invariably involves a threat to the dieter’s self-image...’ (p. 802).

In the past several years, however, it has become increasingly evident that negative emotional stress is only one of a variety of nonappetitive stimuli that produce overeating in restrained eaters. For example, Ward and Mann (2000) examined the effect of a cognitive load, consisting of memorizing art slides and doing a simple reaction time task, on food intake in restrained and unrestrained eaters. On a dimension ranging from ‘relaxing’ to ‘stressful,’ the cognitive load manipulation was rated by subjects as more relaxing than stressful. Yet it produced the familiar interaction wherein restrained eaters consumed more, and unrestrained eaters consumed less, in the high versus low cognitive load conditions. The authors concluded that cognitive load, not stress per se, interfered with restrained eaters’ monitoring of their food intake. Other researchers (e.g. Boon, Stroebe, Schut, & Ijntem, 2002; Lattimore & Maxwell, in press) have also come to the conclusion that nonstressful cognitive load manipulations interact with restraint status to produce patterns of food intake just like those produced in past research on stress-induced eating (Herman and Polivy, 1975). Furthermore, a study by Bellisle and Dalix (2001) found that, under conditions of simple distraction (e.g. listening to a detective story), restraint status predicted amount of food consumed.

In sum, although no definitive conclusions can be drawn about what does cause ‘stress-induced’ eating in ‘restrained eaters,’ it appears that neither stress per se nor restrained eating (in any of its forms) is responsible. If restrained eating represents a proxy risk factor for appetitive hyper-responsiveness (Lowe and Timko, 2004), then future research will be needed to determine the nature of the appetitive predisposition that restraint measures reflect. It no longer appears tenable, however, to suggest that stress increases the intake of restrained eaters because of their dieting behavior or that the negative emotional arousal that is usually produced by stressors is a necessary part of this relationship.

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