



## Sex differences in the association between dietary restraint, insulin resistance and obesity



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### ABSTRACT

**Background & aims:** Restrained food consumption may alter metabolic function and contribute to eventual weight gain; however, sex differences in these relationships have not been assessed. The objective of this study was to examine the relationship between restrained eating and insulin resistance and the influence of body mass index and sex on this relationship in a large community sample of both men and women. We hypothesized that restrained eating would be related to insulin resistance and this relationship would be influenced by sex and body mass index.

**Methods:** In this cross-sectional, observational study, we studied 487 individuals from the community (men  $N = 222$ , women  $N = 265$ ), who ranged from lean (body mass index 18.5–24.9 kg/m<sup>2</sup>,  $N = 173$ ), overweight (body mass index 25–29.9 kg/m<sup>2</sup>,  $N = 159$ ) to obese (body mass index >30 kg/m<sup>2</sup>,  $N = 155$ ) weight categories. We assessed restrained eating using the Dutch Eating Behavior Questionnaire and obtained fasting morning plasma insulin and glucose on all subjects.

**Results:** In men, but not in women, restrained eating was related to homeostatic model assessment of insulin resistance (HOMA-IR) ( $p < 0.0001$ ). Furthermore, HOMA-IR was significantly higher in men who were high- versus low-restrained eaters ( $p = 0.0006$ ).

**Conclusions:** This study is the first to report sex differences with regard to the relationship between restrained eating and insulin resistance. Our results suggest that high restrained eating is associated with insulin resistance in men but not in women.

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### 1. Introduction

Obesity affects two-thirds of the United States population (Flegal, Carroll, Ogden, & Curtin, 2010). Many individuals attempt to control their weight by consciously limiting or restraining food intake. Unfortunately, restrained food consumption may alter metabolic function (Gingras, Harber, Field, & McCargar, 2000; Keim & Horn, 2004; Laessle, Tuschl, Kotthaus, & Pirke, 1989a, 1989b; Pirke et al., 1990; Schur, Cummings, Callahan, & Foster-Schubert, 2008; Teff & Engelman, 1996) and, in several studies, has been found to lead to eventual weight gain (Klesges, Isbell, & Klesges, 1992; Stice, Presnell, Shaw, & Rohde, 2005). Some studies attribute this discrepancy to decreased energy expenditure in restrained eaters (Tuschl, Platte, Laessle, Stichler, & Pirke, 1990). Other

studies have examined metabolic function in the setting of restrained eating (Gingras et al., 2000; Keim & Horn, 2004; Laessle et al., 1989a, 1989b; Pirke et al., 1990; Schur et al., 2008; Teff & Engelman, 1996). Most, but not all, of these studies have found that lean women who are restrained eaters have lower fasting insulin (Pirke et al., 1990), decreased insulin resistance, increased postprandial insulin and glucose, increased insulin sensitivity (Martins, Morgan, & Robertson, 2009), and increased cephalic phase insulin response (Teff & Engelman, 1996) as compared to their non-restrained counterparts. Women who are overweight and obese (OW/OB) restrained eaters are relatively more insulin sensitive as compared to non-restrained OW/OB women (Keim & Horn, 2004). These studies give valuable insight into metabolic pathophysiology that may contribute to weight gain in some restrained eaters but notably have limitations. A majority of these studies investigate restrained eating in women, rather than including both sexes, have a relatively small sample size, and include subjects who are either lean or OW/OB but do not assess differences across the weight spectrum. Additionally, none of these studies directly compare lean to obese restrained- and unrestrained-eaters in relation to insulin resistance and no study has examined sex differences in these relationships. As differences exist between men and women with

**Abbreviations:** BMI, body mass index; DEBQ, Dutch Eating Behavior Questionnaire; FPG, fasting plasma glucose; HOMA-IR, homeostatic model assessment of insulin resistance; OW, overweight; OB, obese; SAS, statistical analysis software.

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respect to insulin resistance (Geer & Shen, 2009), we contend that men and women differ with regard to the relationship between restrained eating and insulin resistance and that these metabolic discrepancies may partially account for differential patterns of weight gain in men and women. In this study, we examine the association of restrained eating and insulin resistance and the influence of body mass index (BMI) on this relationship in a large community sample of both men and women. We hypothesized that restrained eating would be related to insulin resistance and this relationship would be influenced by sex and BMI.

## 2. Materials and methods

### 2.1. Subjects: anthropometric and biochemical evaluation

Healthy men and women, between ages 18 and 50 years, were recruited from the community via local advertisements (see Supplemental methods for subject characteristics). The study was approved by the Yale Human Investigation Committee. All subjects provided signed informed consent. Subjects presented to the laboratory at 8:00 am after an overnight fast; blood samples for fasting plasma glucose (FPG) and fasting insulin were obtained; weight and height were measured.

### 2.2. Assessments and questionnaires

Demographic, physical and mental health assessments were obtained on all subjects. To assess restrained eating we used the Dutch Eating and Behavior Questionnaire (DEBQ) which is a valid measure of eating restraint, emotional eating, and external eating (Vanstrien, Frijters, Bergers, & Defares, 1986; Wardle, 1987). In particular, the DEBQ's Restraint (DEBQ-R) Scale has been shown to be an effective tool for examining dietary restraint (Laessle et al., 1989a, 1989b; van Strien, Herman, Engels, Larsen, & van Leeuwe, 2007).

### 2.3. Statistical analyses

Statistical analysis software (SAS) was used to perform demographic statistics, correlations, and general linear model statistical analyses (see Supplemental Materials for statistical details).

## 3. Results

### 3.1. Group demographics, behavioral measures, and metabolic parameters

We studied 487 individuals from the community (men  $N = 222$ , women  $N = 265$ ), who were lean (BMI 18.5–24.9 kg/m<sup>2</sup>,  $N = 173$ ), overweight (BMI 25–29.9 kg/m<sup>2</sup>,  $N = 159$ ) and obese (BMI > 30 kg/m<sup>2</sup>,  $N = 155$ ). There were no statistically significant differences between men and women in this sample with regard to age, education, race, BMI, fasting insulin, and HOMA-IR. There was a statistically significant difference between the sexes with regard to FPG and reported restrained eating; men showed significantly higher levels of FPG compared to women, and women reported higher levels of restrained eating compared to men (Supplemental Table 1).

### 3.2. Metabolic parameters as a function of restrained eating, BMI, and gender

In the whole group, restrained eating was correlated with HOMA-IR ( $r = 0.15$ ,  $p = 0.001$ ) (Fig. 1a), insulin ( $r = 0.147$ ,  $p = 0.0012$ ), and BMI ( $r = 0.27$ ,  $p < 0.0001$ ) (Fig. 1b), while it was not correlated with glucose ( $r = 0.02$ ,  $p = 0.66$ ). Overall regression models for HOMA-IR indicated a significant effect of restrained eating ( $t = -2.18$ ,  $p = 0.03$ ), and significant interactions between restrained eating and BMI ( $t = 2.43$ ,  $p = 0.02$ ), restrained eating and sex ( $t = 2.25$ ,  $p = 0.03$ ), BMI and sex ( $t = 2.08$ ,  $p = 0.04$ ), and restrained eating, BMI, and sex ( $t = -2.54$ ,  $p = 0.01$ ) on HOMA-IR (Supplemental Table 2). On the

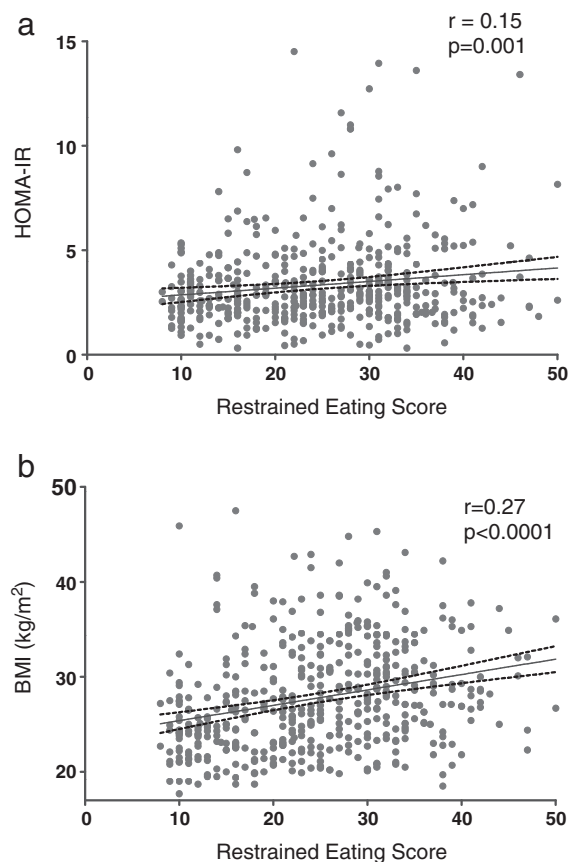


Fig. 1. Scatter plots showing the associations between restrained eating and a) HOMA-IR, and b) BMI.

other hand, overall regression models for fasting insulin indicated that significance level was marginally missed with regard to the effect of restrained eating on insulin ( $t = -1.96$ ,  $p = 0.051$ ) and the effect of restrained eating and sex ( $t = 1.93$ ,  $p = 0.054$ ) on insulin. There was no significant interaction between sex and BMI ( $t = 1.77$ ,  $p = 0.08$ ) but there was a significant interaction between restrained eating and BMI ( $t = 2.13$ ,  $p = 0.03$ ) and restrained eating, BMI, and sex ( $t = -2.17$ ,  $p = 0.03$ ).

As sex was significantly interacting with restrained eating and BMI for HOMA-IR, follow-up models to explore these associations were conducted separately in men and women. In men, we found a significant relationship of restrained eating with HOMA-IR ( $t = -2.19$ ,  $p = 0.03$ ), restrained eating with insulin levels ( $t = -1.99$ ,  $p = 0.05$ ), and significant interactions between restrained eating and BMI ( $t = 2.44$ ,  $p = 0.02$ ) on HOMA-IR and between restrained eating and BMI on insulin ( $t = 2.16$ ,  $p = 0.03$ ). Meanwhile, in women, there were no significant effects of restrained eating on HOMA-IR or insulin and no significant interactions between restrained eating, BMI and HOMA-IR or insulin (Supplemental Table 2). As HOMA-IR levels and restrained eating scores were correlated in men ( $r = 0.29$ ,  $p < 0.0001$ ) (Fig. 2a) but not in women ( $r = 0.03$ ,  $p = 0.59$ ) (Fig. 2b), we divided the group into low- and high-restrained eaters (median split) and found that HOMA-IR levels were statistically different in men who were low- versus high-restrained eaters ( $p = 0.0006$ ) (Fig. 2c). An assessment of the significant interactions between restrained eating and BMI in men revealed that while in low-restrained eaters, HOMA-IR levels showed a significant graded increase different between each of the BMI categories, men who were high-restrained eaters showed no differences in HOMA-IR levels comparing the lean and overweight men, but a statistically significant difference between lean/overweight as compared to obese high-restrained men (Fig. 2d). There were no associations that

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