

Association between Depressive Symptom Clusters and Food Attentional Bias

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IN, USA**Keywords:** eye-tracking, visual probe, food attentional bias, depressive symptoms**Running title:** Depressive Symptoms and Food Attentional Bias**Address Correspondence and Reprint Requests to:** Misty A. W. Hawkins, Ph.D., Department of Psychology, 116 North Murray, Oklahoma State University, Stillwater, OK 74074. Phone: (405) 744-4593. Fax: (405) 744-8067. E-mail: misty.hawkins@okstate.edu**Funding:** This project was supported by HRSA-10-175 - Equipment to Enhance Training for Health Professionals (D76HP20905) awarded to M.A.C.**Disclosure:** The authors declared no conflict of interest.

Abstract

Background: The mechanisms underlying the depression-obesity relationship are unclear. Food attentional bias (FAB) represents one candidate mechanism that has not been examined. We evaluated the hypothesis that greater depressive symptoms are associated with increased FAB.

Method: Participants were 89 normal weight or overweight adults (mean age=21.2±4.0 years, 53% female, 33% non-white, mean body mass index in $\text{kg}/\text{m}^2 = 21.9 \pm 1.8$ for normal weight; 27.2 ± 1.5 for overweight). Total, somatic, and cognitive-affective depressive symptom scores were computed from the Patient Health Questionnaire-8 (PHQ-8). FAB scores were calculated using reaction times (RT) and eye-tracking (ET) direction and duration measures for a food visual probe task. Age, gender, race/ethnicity, and body fat percent were covariates.

Results: Only PHQ-8 somatic symptoms were positively associated with RT-measured FAB ($\beta = .23, p = .04$). The relationship between somatic symptoms and ET direction ($\beta = .18, p = .17$) and duration ($\beta = .23, p = .08$) FAB indices were of similar magnitude but were not significant. Somatic symptoms accounted for 5% of the variance in RT-measured FAB. PHQ-8 total and cognitive-affective symptoms were unrelated to all FAB indices ($ps \geq .09$).

Conclusions: Only greater somatic symptoms of depression were linked to food attentional bias as measured using reaction time. Well-powered prospective studies should examine whether this bias replicates, particularly for eye-tracking measures, and whether it partially mediates the depression-to-obesity relationship.

1. Introduction

Food attentional bias (FAB) occurs when a person responds more quickly and/or for a longer duration to food-related stimuli, with the underlying assumption that these patterns of responses demonstrate a greater motivational drive and/or cravings for food.¹⁻³ FAB has been linked to negative effects (e.g., increased food cravings and consumption⁴ or weight gain⁵) in normal and overweight individuals. Thus, it is necessary to investigate factors which may serve to develop and/or maintain FAB.

One potential factor that has not yet been explored is depression. Given that negative mood states or depressive symptoms may be reduced by eating,^{6,7} the incentive salience of food⁸ and corresponding attentional biases may be further enhanced among depressed individuals. However, it is important to note that this proposed relationship may not be active among individuals at either end of the weight spectrum. Previous reports suggest that once obesity has developed, food/eating may be imbued with ambivalent/aversive experiences,⁹ leading to attempts to avert attention away from or avoid food stimuli.^{4,10} Likewise, anorexia nervosa is also characterized by attempts to avoid food/eating. A key difference may be that those with anorexia nervosa do avoid eating, resulting in underweight,¹¹ whereas those with obesity do not – despite their attempts to shift attention away from food.

Thus, our hypothesis is that greater depressive symptom severity among normal or overweight individuals is associated with increased FAB, measured by reaction time and eye-tracking. Thus, we tested our hypothesis regarding FAB and depression among those without obesity or underweight.

2. Method

The recruited sample was 120 young adults participating in the research participant pool at a large urban university. We excluded participants who were obese ($BMI \geq 30 \text{ kg/m}^2$; $n=21$) or underweight ($BMI < 18.5 \text{ kg/m}^2$; $n=7$). Per standards in the literature,² we also excluded individuals with RTs times considered out of range ($< 200\text{ms}$, $> 1500\text{ms}$, or $> 3 \text{ SDs}$ above mean; $n = 3$). Our sample was comprised of individuals with normal to overweight BMI ($18.5\text{-}29.9 \text{ kg/m}^2$) and valid RT scores ($n=89$) (Table 1). Of the selected sample, 74 participants had useable eye-tracking (ET) data. ET values were missing for various reasons, including calibration failure, no fixations in areas of interest across trials, or file errors.

Table 1. Characteristics of Participants

	Sample ($N = 89$)	Not Depressed ($n = 73$)	Depressed ($n = 16$)
<i>Demographic Factors</i>			
Age	21.2 ± 4.0	21.5 ± 4.3	20.0 ± 1.7
Female	47 (52.8)	36 (49.3)	11 (68.8)
Race/ethnicity (non-white)	29 (32.6)	22 (30.1)	7 (43.8)
<i>Depressive Symptom Severity (possible range)</i>			
Total Score (PHQ-8) (0-24)	5.5 ± 3.8	4.1 ± 2.3	$12.1 \pm 1.9^*$
Cognitive-Affective Symptoms (0-15)	2.6 ± 2.2	1.9 ± 1.6	$5.5 \pm 2.2^*$
Somatic Symptoms (0-9)	3.0 ± 2.3	2.2 ± 1.5	$6.7 \pm 1.9^*$
Depressed (PHQ-8 ≥ 10)	16 (18.0)	0 (0.0)	16 (100.0)*
<i>Adiposity Indicators</i>			
Body fat percent (BF%)	22.30 ± 7.89	22.0 ± 8.0	23.7 ± 7.7
Body mass index (BMI; kg/m^2)	24.00 ± 3.09	24.0 ± 3.0	23.8 ± 3.5
Overweight	35 (39.3)	42 (57.5)	12 (75.0)
Normal weight	54 (60.7)	31 (42.5)	4 (25.0)
<i>Attentional Biases Scores</i>			
Reaction Time Bias (RT; ms)	-20.32 ± 38.27	-24.4 ± 36.1	$-1.7 \pm 43.5^*$
Eye-tracking Scores (0-1) (>0.50 = food bias; 0.50 = no bias; <0.50 = non-food bias)	Eye-tracking Subsample ^b ($N = 74$ complete cases; $N = 89$ with imputation)		
Direction Bias	0.47 ± 0.20	0.45 ± 0.19	0.55 ± 0.23
Duration Bias	0.49 ± 0.22	0.47 ± 0.21	0.58 ± 0.23

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Note. *Differences $p < .05$ between depressed and not depressed groups. Means and standard deviations are presented for continuous variables. Frequencies and percentages are presented for categorical variables. PHQ-8 = Patient Health Questionnaire-8.

^aThe sample selected for analyses ($N = 89$) excluded participants with BMI < 18.5 or ≥ 30.0 kg/m² ($n = 28$) and RT values out of range ($n = 3$). Excluded participants did not differ from selected participants on any variables except they had higher BMI ($t(116)=5.89$, $p=.001$) and higher body fat percent (BF%; $t(116)=3.56$, $p=.009$), as expected.

^bMissing eye-tracking data were associated with observed variables – age (older) and race-ethnicity (non-white) – suggesting the missingness at random (MAR) mechanism in our data. Thus, we utilized multiple imputation to reduce parameter bias.

2.1 Measures

2.1.1 Depressive symptoms.

Depressive symptoms were assessed using the Patient Health Questionnaire-8 (PHQ-8) total score, cognitive-affective subscale score (Items 1, 2, 6, 7, and 8), and somatic subscale score (Items 3, 4, and 5).^{12,13} Higher scores indicate greater symptoms, and 18% of the sample had a total score ≥ 10 , which is indicative of clinically significant depressive symptoms (Table 1).¹⁴ The PHQ-8 has demonstrated adequate reliability and validity,¹³ and Cronbach's α in this sample was 0.76.

2.1.2 Food attentional bias

FAB was assessed by visual probe task reaction time (RT) and eye-tracking (ET) in order to compute RT bias and ET gaze direction and duration bias. The visual probe task was adapted from the established protocol of Castellanos et al. (2009).¹ Briefly, each food/non-food image pair was presented twice across 60 total trials, with the food image counterbalanced on the left and right and 20 non-critical control images presented once. Per the original protocol, various foods were used (e.g., apples, chocolate, carrots, pretzels) with nature scenes used for controls. RT bias was computed by subtracting mean RT to dots presented in the location of one of the food images from mean RT to dots presented in the location of the non-food images. Positive values indicate faster responses to probes located under food images, indicating that participants were attending to the food images prior to probe appearance.

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Participants' eye movements were tracked after a 9-point calibration using the EyeTrac® Series 6 Desk-mounted Eye Tracking Device (ASL Eyetracking: Bedford, MA). The EyeTrac® recorded subjects' gaze location during the visual probe task. The number and duration of gaze fixations (stable eye position for > 0.1 ms) were calculated for each image pair. Fixation data were used to calculate direction bias and duration bias. Direction bias is a measure of initial orienting of attention toward or away from food cues and computed as a proportion of total paired image presentations in which the participant's first fixation on an image was on the food image. Duration bias is a measure of time spent looking at food versus non-food cues and is calculated as the proportion of time that the participant spent looking at food images divided by the total time spent looking at either food or non-food images. Values over 0.5 indicate a direction or duration bias for food cues.

2.1.3 Covariates

Self-reported age (years), gender (male=0, female=1), and race-ethnicity (0=non-white, 1=white) were covariates. We also adjusted for body fat percent (BF%) measured using a body composition analyzer (Model TBF-300A; Tanita Corporation of America, Inc: Arlington Heights, IL) to control for observed effects of excess adiposity on FAB.¹⁵ State hunger level was measured at the beginning of the session using a visual analog scale (VAS).

2.2 Procedure

To start the 1.5-hour laboratory session, participants consented to all study procedures approved by the local institutional review board. Participants then completed the hunger VAS and the Patient Health Questionnaire-8. The participant then sat in front of a desk-mounted eye-tracking camera and chin rest used to minimize head movement during the practice task, calibration, and experimental visual probe task. Next, participants completed a computerized

survey. For some participants, this survey was administered before the FAB assessment, and the potential moderating influence of the protocol switch was examined. Last, BF% was measured.

2.3 Analyses

Missing Data Handling. Across all of the variables examined here, no more than 17% of data were missing on any variable (eye tracking variables). For the PHQ-8, we imputed within person, within subscale when the participant was missing 1-2 items. Multiple Imputation (MI) analysis was then conducted using PROC MI in SAS 9.3, utilizing a Markov Chain Monte Carlo (MCMC) algorithm known as fully conditional specification (FCS) regression. The imputation model included all the variables used in the current study, as well as additional variables from the larger data set that may add importantly to the imputation (e.g. demographics, weight, height, glasses, practice trials). Thirty separate data sets were imputed with the number of between-imputation iterations set to 200.¹⁶ Analyses were then run on all 30 imputed data sets using PROC MIANALYZE and the statistics automatically pooled. Results were found to be consistent when compared to complete case analyses.

Primary Analyses. We conducted linear regression analyses with three different dependent FAB variables: 1) RT bias score, 2) ET direction bias score, and 3) ET duration score. For each regression model, covariates (age, gender, race/ethnicity, and BF%) were entered in Step 1, and the PHQ-8 total score was entered in Step 2. We then repeated these three analyses entering either the somatic or cognitive-affective PHQ-8 subscale score in Step 2. Covariates were entered on Step 1 to ensure that any effect of depressive symptoms was above and beyond the effects of any covariates. To examine task order as a moderator, PHQ-8 by task order interaction terms were created.

3. Results

Linear regression analyses (Table 2) revealed that greater somatic symptoms were associated with greater RT bias scores, accounting for 5.0% of the variance. Somatic symptoms were not significantly related to the ET bias variables, but the relationships showed a similar pattern. Specifically, non-significant positive standardized regression coefficients of similar magnitude were observed for associations of somatic symptoms with direction and duration bias scores. Somatic symptoms accounted for 3.0-5.0% of the variance in the ET bias variables though the relationships were not significant. The PHQ-8 total score and cognitive-affective symptoms were unrelated to RT, direction, or duration bias scores.

Post hoc removal of the appetite item (#5) of the somatic scale did not eliminate the above associations for somatic symptoms: $\beta = .23, p = .04$ for RT bias; $\beta = .18, p = .16$ for ET Direction, and $\beta = .22, p = .09$ for ET Duration with Item 5 removed. Adding the VAS hunger score as a covariate to the models also failed to eliminate the pattern of results. Examining task order as moderator showed significant interactions for RT bias only: PHQ-8 total score x task order ($p=.049$) and somatic score x task order ($p=.034$). Given the influence of order on RT bias outcomes, we conducted analyses stratified by task order category. Associations of the PHQ-8 total and somatic scores with RT bias were stronger when FAB was assessed after versus before the survey, suggesting that the potential influence of task order should be carefully examined in future studies.

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Table 2. Results of Regression Analyses Examining the Association between Depressive Symptoms and Food Attentional Bias

Variables	Food Attentional Bias ^a											
	RT Bias (<i>N</i> = 89)				ET Direction Bias (<i>N</i> = 89)				ET Duration Bias (<i>N</i> = 89)			
	<i>R</i> ²	ΔR^2	β	<i>p</i>	<i>R</i> ²	ΔR^2	β	<i>p</i>	<i>R</i> ²	ΔR^2	<i>B</i>	<i>p</i>
<i>Demographic Factors & Adiposity (Step 1)</i>												
	.07	--	--	.18	.02	--	--	.70	.02	--	--	.89
Age			.04	.74			.06	.68			-.02	.86
Gender			-.05	.77			.14	.45			.07	.70
Race/ethnicity			-.23	.04**			-.02	.84			-.02	.88
Body fat %			.12	.44			-.13	.48			-.16	.35
<i>Depressive Symptoms (Step 2)</i>												
PHQ-8 Total Score	.10	.03	.19	.09	.04	.02	.12	.37	.05	.03	.16	.24
PHQ-8 Cognitive-Affective Score ^b	.08	.01	.08	.49	.03	.00	.02	.90	.03	.00	.03	.81
PHQ-8 Somatic Score ^b	.12	.05	.23	.04**	.06	.03	.18	.17	.07	.05	.23	.08

Note. RT = reaction time. ET = eye tracking. PHQ-8 = Patient Health Questionnaire.

^aEach food attentional bias variable was examined as the dependent variable in a separate model.

^bThe PHQ-8 total score and subscale scores were each examined as the independent variable in separate models.

**Significant at $p < .05$

4. Discussion

Our objective was to evaluate our hypothesis that greater depressive symptom severity is associated with increased food attentional bias in normal weight/overweight adults. In support of our hypothesis, participants with higher somatic symptoms of depression tended to have faster responses to probes presented under food cues (RT food bias). The observed coefficients for the ET bias measures were not significant but were in the hypothesized direction and accounted for a similar magnitude (3-5%) of the variance in the RT and ET bias measures beyond the covariates. The failure of relationships between somatic scores and ET bias measures to reach significance may be due to inadequate statistical power for the ET bias measures. If replicated in a well-powered sample, these results may suggest that the incentive salience of food may be enhanced among those with greater somatic depressive symptoms, an effect not explained solely by increased appetite or hunger.

Notably, these small-to-medium effects were observed in normal weight and overweight people, suggesting that these basic mechanisms are present prior to obesity development and not just a consequence of existing obesity. Interestingly, post hoc comparisons of obese participants with their normal weight peers replicated the previously documented attentional food avoidance pattern, suggestive of relative disengagement from food stimuli among obese participants¹⁰ – but only among depressed participants. Combined, these findings suggest that depressive symptoms may be a missing moderator of analyses examining food attentional bias in relation to weight status, particularly those studies with longer stimulus presentation times (>200ms) that introduce the potential for attentional shifts.

One possible reason why somatic symptoms of depression, but not cognitive-affective symptoms, were associated with some FAB indices is that somatic symptoms may be more

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strongly associated with dysregulation of factors related to energy homeostasis, such as leptin.^{17,18} Leptin is closely linked to satiety signaling, and its dysregulation can increase food cravings (and corresponding attention to food).¹⁸ Another explanation is that somatic symptoms of depression may reflect the atypical subtype of depression more than the melancholic subtype. Atypical depression is characterized by hyperphagia, hypersomnia, lethargy/fatigue, and increased reactivity to the environment.¹⁹ These features contrast with the reduced appetite, insomnia, agitation, and decreased responsivity to the environment that characterize melancholic depression. Thus, participants endorsing somatic symptoms may exhibit a more atypical presentation and the corresponding increased reactivity to the environment, possibly including food cues. Unfortunately, the use of the PHQ-8 does not allow us to separate atypical and melancholic presentations because the items about appetite and sleep are double-barreled (e.g., “*Have you been bothered by poor appetite or overeating?*”).

Despite the novelty of this study and its use of multiple attentional bias measures, key limitations include an inability to determine the directionality of the observed associations and lower statistical power for the ET bias measures. However, to our knowledge, our sample size of 74 adults is the largest study examining eye-tracking measured FAB in a sample of healthy weight and overweight adults.¹⁵

In conclusion, greater somatic symptoms of depression may be associated with increased FAB, as measured using reaction time, in healthy weight and overweight adults. Given that depression impacts over 16 million U.S. adults,²⁰ this line of research may have widespread implications with replication. Identifying the most obesogenic symptoms of depression and their underlying mechanisms would inform the development of novel interventions to reduce the

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excess obesity risk of adults experiencing depressive symptoms. Specifically, targeting somatic depressive symptoms may serve to reduce maladaptive attentional biases to food.

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Highlights

- Depression may lead to obesity via increased attention to and intake of food.
- Greater somatic depressive symptoms were linked to greater food attentional bias measured using reaction time.
- Food bias may ultimately result in increased calorie intake in depression.

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