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Mediation of the bidirectional relations between obesity and depression among women

Jeffrey R. Vittengl

Department of Psychology, Truman State University, Kirksville, MO, USA

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ABSTRACT

Past research established that obesity increases risk for development of depression, and depression increases risk for development of obesity. The current study tested physical impairment (difficulty with instrumental activities of daily living), social dysfunction (low social support and high social strain), and emotional eating (using food to cope with stress) as mediators of the bidirectional, longitudinal relations between depression and obesity. A national sample of mid-life adults in the United States (N = 7108) was assessed at three time points over 18 years. Depression predicted increases in obesity, and obesity predicted increases in depression, for women but not for men. Among women, path analyses revealed that physical impairment, social dysfunction, and emotional eating mediated development of obesity from depression, and that physical impairment and emotional eating mediated development of depression from obesity. These results suggest that prevention or treatment of obesity-linked depression and depression-linked obesity in women may need to address multiple connections between these disorders.

1. Introduction

The association between depression and obesity is well-established, but the reasons for this connection are incompletely understood (Luppino et al., 2010; Preiss et al., 2013). The current study tested mediators of the development of obesity from depression, and depression from obesity, among mid-life adults. Tested mediators included physical impairment, social dysfunction, and emotional eating. These results may inform efforts to prevent or treat depression-linked obesity and obesity-linked depression.

Both cross-sectional (Onyike et al., 2003; Pereira-Miranda et al., 2017) and longitudinal (Luppino et al., 2010; Marmorstein et al., 2014) research has shown associations between obesity and depression. For example, obese persons had 55% higher odds of developing depression, and depressed persons had 58% higher odds of becoming obese, in meta-analyses of longitudinal studies (Luppino et al., 2010). However, connections between obesity and depression appear stronger among women but reduced or absent among men (Jorm et al., 2003; Onyike et al., 2003; Pereira-Miranda et al., 2017).

Researchers have proposed several mechanisms accounting for observed connections between obesity and depression. Depression and obesity may be intertwined via several somatic processes including immuno-inflammatory and hypothalamic-pituitary-adrenal axis dysregulation (Penninx et al., 2013). At another level of analysis, a number of behavioral and psychosocial processes have been implicated, including physical impairment, social dysfunction, and emotional eating (Preiss et al., 2013).

Related to physical impairment, poorer self-reported health may partly account for correlations of obesity with depression (Siegel et al., 2000) and subjective well-being (Böckerman et al., 2014). Further, low physical activity is associated with both obesity (Kilpeläinen et al., 2011) and depression (Hiles et al., 2017) and may partly mediate their relations (Simon et al., 2008). Thus, perhaps especially as mid-life adults age, physical impairment has the potential to mediate bidirectional relations between obesity and depression.

Social dysfunction, including facets such as weight-based discrimination (Jackson et al., 2015), low social participation (de Wit et al. 2010), and poor social support (Jorm et al., 2003), may partly explain connections between obesity and depression. For example, social dysfunction associated with depression (e.g., perhaps due to low behavioral activation; Vittengl et al., 2015) or obesity (e.g., perhaps due to social stigma; Robinson et al., 2017) might eventually produce the other disorder.

Finally, coping with stress or negative emotion by eating more or favorite foods ("emotional eating") may increase both depression (e.g., if eating for short-term mood improvement supplants behaviors that reduce sources of stress) and obesity (e.g., if non-emotional caloric intake is not reduced commensurately). Indeed, both cross-sectional and longitudinal studies suggest that emotional eating may partly mediate relations between depression and obesity (Konttinen et al., 2010;

E-mail address: vittengl@truman.edu.

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Lazarevich et al., 2016; van Strien et al., 2016).

In this context, the current study tested connections between obesity and depression in a national sample of midlife adults assessed at three time points over a period of 18 years. The hypotheses were (1) obesity and depression are linked bidirectionally across time (i.e., earlier obesity predicts later increases in depression, and earlier depression predicts later increases in obesity); and (2) physical impairment, social dysfunction, and emotional eating each partly mediate the bidirectional relations between obesity and depression (i.e., physical impairment, social dysfunction, and emotional eating account for the relations between depression and obesity across time). Because the obesity-depression link has been stronger among women than men in past research, data were analyzed separately by gender.

The purpose of the current study was to extend past research by testing mediation in a more comprehensive framework. First, the current study's use of three time points helped disentangle the three-term mediation process (cause→mediator→outcome). Past research often included only one or two time points, which although informative, confounded in time parts of the mediation process. Second, the current analyses were bidirectional, examining development of depression from obesity and obesity from depression simultaneously. Prior studies usually assessed only one direction of development in a system that is likely bidirectional (Atlantis et al., 2009; Pan et al., 2012). Third, prior studies focused on a single mediator, whereas the current analyses tested three potential mediators simultaneously (physical impairment, social dysfunction, emotional eating), reflecting increasing evidence of multiple connections between depression and obesity.

2. Methods

2.1. Participants and procedure

Data were drawn from the Midlife Development in the United States Survey, a national study of health and well-being conducted in three waves in years 1995–1996 (n = 7108), 2004–2006 (n = 4963), and 2013–2014 (n = 3294; Ryff et al., 2016). Participants were recruited via national random digit dialing (n = 3487), a sample of their siblings (n = 950), random digit dialing in metropolitan areas (n = 757), and a national database of twin pairs (n = 1914) and provided informed consent. The data collection was approved by Health Sciences and the Education and Social/Behavioral Sciences Institutional Review Boards of the University of Wisconsin–Madison (Radler, 2014). Wave 1 participants were English-speaking, non-institutionalized adults, residing in the coterminous United States, ages 25–74 years. Characteristics of the wave 1 sample are shown in Table 1. Waves 2 and 3 attempted to reassess all living participants from the prior wave. Participants completed telephone interviews and mail-in questionnaires.

2.2. Measures

2.2.1. Obesity

Obesity level was scored as a composite of body mass index (BMI) and waist circumference at survey waves 1 and 3. Participants self-reported their current weight and height, which were used to compute BMI (kg/m^2). Participants also received a tape measure and standardized instructions to measure and record their waist circumferences. Following standard guidelines, BMI level was coded as 0 (BMI < 30), 1 (BMI 30–34.9), 2, (BMI 35–39.9), or 3 (BMI ≥ 40; Obesity Education Initiative, 1998). Waist circumferences > 35 inches for women or > 40 inches for men also mark obesity (World Health Organization, 2008). Parallel to BMI, waist circumference was coded as 0 (women < 35, men < 40 inches), 1 (women 35–39.9, men 40–44.9 inches), 2 (women 40–44.9, men 45–49.9 inches), or 3 (women ≥ 45, men ≥ 50 inches). Agreement between BMI and waist circumference levels was substantial (at wave 1, intraclass correlation = 0.74), and their sum produced a reliable obesity scale (at wave 1, alpha = 0.86).

Table 1
Description of study sample at survey Wave 1.

Variable	*N*	*M* or %	*SD*
Age in years	7049	46.38	13.00
Female gender	7027	51.7%	
White race	6176	90.7%	
Household income	6110	10.99	9.39
Education level	7095	6.77	2.49

Note. Income in $10,000 units adjusted for inflation to year 2015 U.S. dollars. Education rated on a 1–12 scale (e.g., *1 = no school/some grade school,* 5 = *graduated from high school,* 9 = *bachelor's degree,* 12 = *doctoral degree*).

2.2.2. Depression

Criteria for major depressive disorder (APA, 1987) were assessed with the Composite International Interview short-form (Kessler et al., 1994,1998) at survey waves 1 and 3. Assessment focused on the past year. Positive screens for two weeks of depressed mood and/or anhedonia were followed by assessment of six additional symptoms (e.g., appetite changes, fatigue, thoughts of death), yielding a 0–7 scale. The hierarchical nature of the depression scale precluded estimation of internal consistency, but reliability of the Composite International Interview short-form has been good in past research (Kessler et al., 1998).

2.2.3. Physical impairment

Participants rated how much their health limited performance of seven behaviors (e.g., lifting or carrying groceries; bending, kneeling, or stooping; walking several blocks) on a 4-point scale from *not at all* to *a lot*. Higher average scores reflect greater physical impairment (Lawton & Brody, 1969). Alpha reliability for the scale was high (0.94) at survey wave 2.

2.2.4. Social dysfunction

On 4-point scales from *never* to *often* ("make too many demands on you," "criticize you," "let you down," "get on your nerves;") and *not at all* to *a lot* ("really care about you," "rely on them for help," "understand the way you feel," "open up to them if you need to talk"; reverse-keyed) participants rated the quality of their social relationships with friends and family separately (16 total items; Schuster et al., 1990). Higher average scores mark poorer social functioning. Alpha reliability for the scale was moderately high at survey wave 2 (0.85).

2.2.5. Emotional eating

Among items describing coping behaviors in response to stress, participants rated two items ("I eat more than I usually do", "I eat more of my favorite foods to make myself feel better") reflecting use of food on 4-point scale ranging from *not at all* to *a lot* (Tsenkova et al., 2013). The average of these two items produced a scale with high alpha reliability (0.90) at survey wave 2.

2.3. Analyses

Bidirectional relations between obesity and depression were estimated with path analyses implemented via PROC CALIS in SAS software version 9.3 (SAS Institute, Inc., Cary, NC). Path models used full information maximum likelihood estimation to include cases with missing data. Statistical significance of paths was evaluated using an a priori alpha level of 0.05, two-tailed. In addition, the overall fit of models was judged as good with goodness of fit index ≥ 0.95, comparative fit index ≥ 0.95, and root mean square error of approximation ≤ 0.05.

Path models included depression and obesity measured at survey waves 1 and 3, plus potential mediators measured at wave 2, as depicted in Figs. 1 and 2. Measuring potential causes (time 1), mediators (time 2), and outcomes (time 3) at separate times helped disentangle

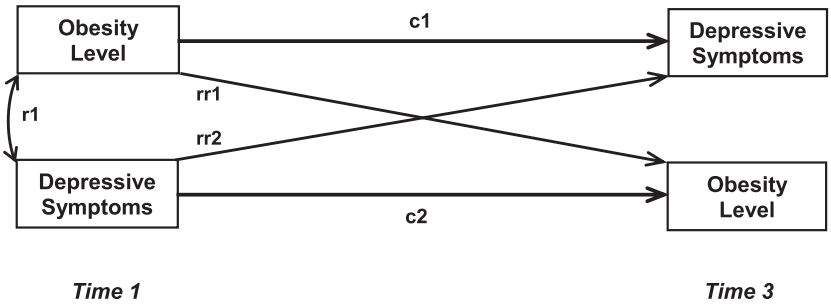

Fig. 1. Path model of direct (unmediated) relations between depression and obesity.

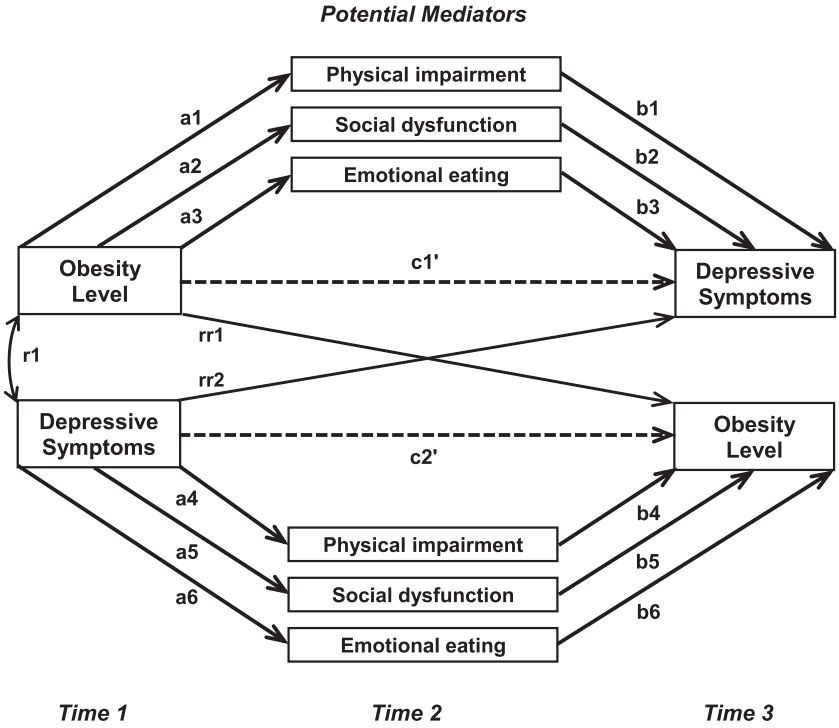

Fig. 2. Path model of mediated relations between depression and obesity (paths c1′ and c2′). Covariance among mediators also modeled but not depicted.

the hypothesized processes. Each model estimated all paths simultaneously, adjusting each path for other included paths. The models included concurrent relations between depression and obesity at time 1 but not at time 3, because preliminary analyses indicated that the time 3 relations were not significant, *ps* ≥ 0.10, when controlling the time 1 relations.

Because longitudinal relations between obesity and depression were significant among women but not among men (Fig. 1), mediation was tested only for women (Fig. 2). Path coefficients were used to test mediation following established procedures (Baron & Kenny, 1986; MacKinnon, 2008). The joint statistical significance of paths **a** and **b** (e.g., in Fig. 2, path **a1** is time 1 obesity → time 2 physical impairment, and path **b1** is time 2 physical impairment → time 3 depression) signaled mediation (e.g., that physical impairment at least partly mediated the relation between obesity and depression). The product of paths **a** × **b** estimated the amount of mediation. The statistical significance of **a** × **b** has often been tested using the Sobel approximation of the standard error (Baron and Kenney, 1986) and more recently with the partial posterior method that may be more powerful (Falk and Biesanz, 2016). The current analyses tested **a** × **b** using both methods.

Before submitting variables to path analyses, variance attributable to family (some participants were siblings) was partitioned with a series of multilevel linear models implemented via PROC HPMIXED in SAS software version 9.3. Each variable to be included in path analyses was predicted from the random effect of family, and residuals (i.e., variance not attributable to family) were retained for path analyses. Appendix 1 shows variance estimates. The statistical significance of these models was not of interest. Instead, removing family variance helped satisfy the assumption of independent observations for the path analyses.

3. Results

Descriptive statistics for the variables included in the mediation analyses appear in Table 2. Bidirectional relations between depression and obesity, as shown in Fig. 1, were estimated separately for women and men. In support of the first hypothesis, obesity at time 1 predicted significantly increased depression at time 3 (path **c1**; see Table 3 for path coefficients), and depression at time 1 predicted significantly increased obesity at time 3 (path **c2**) among women. For men, however, the longitudinal relations between depression and obesity were not

Table 2
Descriptive statistics for variables included in mediation analyses.

Variable	*N*	*M*	*SD*	Range
Obesity: Time 1	6125	0.69	1.31	0–6
Depression: Time 1	7108	0.79	1.93	0–7
Physical impairment: Time 2	4020	1.79	0.88	1–4
Social dysfunction: Time 2	4023	1.77	0.40	1–4
Emotional eating: Time 2	3997	1.87	0.93	1–4
Obesity: Time 3	2658	1.19	1.59	0–6
Depression: Time 3	3294	0.60	1.71	0–7

Table 3
Path coefficients for bidirectional relations between obesity and depression.

Path	Description	Women		Men	
		B	*SE*	*B*	*SE*
c1	Time 1 Obesity → Time 3 Depression	0.059**	0.023	0.018	0.026
c2	Time 1 Depression → Time 3 Obesity	0.065**	0.020	−0.015	0.021
r1	Time 1 Depression ↔ Time 1 Obesity	0.059**	0.018	0.022	0.018
rr1	Time 1 Obesity → Time 3 Obesity	0.663**	0.014	0.676**	0.015
rr2	Time 1 Depression → Time 3 Depression	0.312**	0.021	0.200**	0.025

Note. See Fig. 1 for path diagram. Coefficients are standardized. Model fit was good for women and men, respectively, judged by the goodness of fit index (0.999, 1.000), comparative fit index (0.998, 1.000), and root mean square error of approximation (0.022, 0.000).
* $p < 0.05$, ** $p < 0.01$, two-tailed.

significant. Consequently, mediators of the relations between depression and obesity were tested for women only.[1]

Physical impairment, social dysfunction, and emotional eating were tested simultaneously as potential mediators of the bidirectional relations between depression and obesity among women. Fig. 2 depicts the path model with the addition of the potential mediators. When both paths **a** and **b** were statistically significant for a potential mediator, there was evidence of mediation (see Table 4). Tests of the products of paths **a** × **b** for each mediator variable supported identical conclusions (see Table 5).

In support of the second hypothesis, physical impairment, social dysfunction, and emotional eating each significantly mediated the relation between earlier obesity and later depression among women ($p < 0.01$; see Tables 4 and 5). The remaining relation between earlier obesity and later depression (path **c1′**) was quite small (beta = −0.009) and non-significant ($p = 0.72$), suggesting that these three mediators collectively accounted for most or all of the development of depression from obesity.

Also in support of the second hypothesis, physical impairment and emotional eating, $p < 0.01$, but not social dysfunction, significantly mediated the relation between earlier depression and later obesity among women (see Tables 4 and 5). The remaining association between earlier depression and later obesity (path **c2′**) was reduced (from beta = 0.065 to 0.037) and non-significant ($p = 0.06$) but close to the $p < 0.05$ threshold. Thus, physical impairment and emotional eating accounted for much, but perhaps not all, of the development of obesity from depression.[2]

Table 4
Path coefficients for mediation of relations between obesity and depression among women.

Path	Description	*B*	*SE*
Mediation of Obesity's "Effect" on Depression			
a1	Time 1 Obesity → Time 2 Physical impairment	0.337**	0.019
a2	Time 1 Obesity → Time 2 Social dysfunction	0.082**	0.021
a3	Time 1 Obesity → Time 2 Emotional eating	0.264**	0.020
b1	Time 2 Physical impairment → Time 3 Depression	0.111**	0.025
b2	Time 2 Social dysfunction → Time 3 Depression	0.122**	0.024
b3	Time 2 Emotional eating → Time 3 Depression	0.066**	0.024
c1′	Time 1 Obesity → Time 3 Depression	−0.009	0.025
Mediation of Depression's "Effect" on Obesity			
a4	Time 1 Depression → Time 2 Physical impairment	0.115**	0.020
a5	Time 1 Depression → Time 2 Social dysfunction	0.160**	0.021
a6	Time 1 Depression → Time 2 Emotional eating	0.086**	0.020
b4	Time 2 Physical impairment → Time 3 Obesity	0.074**	0.021
b5	Time 2 Social dysfunction → Time 3 Obesity	0.010	0.020
b6	Time 2 Emotional eating → Time 3 Obesity	0.156**	0.020
c2′	Time 1 Depression → Time 3 Obesity	0.037	0.020
Concurrent and Retest Correlations			
r1	Time 1 Depression ↔ Time 1 Obesity	0.058**	0.018
rr1	Time 1 Obesity → Time 3 Obesity	0.597**	0.017
rr2	Time 1 Depression → Time 3 Depression	0.274**	0.021

Note. See Fig. 2 for path diagram. Coefficients are standardized. Covariance among mediators also modeled: physical impairment and social dysfunction (0.079, $SE = 0.019$); physical impairment and emotional eating (0.023, $SE = 0.019$); social dysfunction and emotional eating (0.137, $SE = 0.020$). Model fit was good, judged by the goodness of fit index (1.000), comparative fit index (1.000), and root mean square error of approximation (0.000).
* $p < 0.05$, ** $p < 0.01$, two-tailed.

Table 5
Tests of the products of paths **a** and **b** from the mediation model.

Variable	Path **a** × **b**	*SE*
Mediation of Obesity's "Effect" on Depression		
Physical impairment	0.038**	0.009
Social dysfunction	0.010**	0.003
Emotional eating	0.017**	0.007
Mediation of Depression's "Effect" on Obesity		
Physical impairment	0.009**	0.003
Social dysfunction	0.002	0.003
Emotional eating	0.013**	0.004

Note. See Table 3 for path **a** and **b** coefficients.
* $p < 0.05$, ** $p < 0.01$, two-tailed, by the Sobel test (Baron and Kenney, 1986) and by the partial posterior method (Falk and Biesanz, 2016).

4. Discussion

The current analyses supported the hypotheses that the longitudinal relations between depression and obesity are bidirectional and manifold. Among women but not men, depression predicted increased obesity, and obesity predicted increased depression, over a period of 18 years. Measured at the mid-point of this period, three mediators helped explain relations between obesity and depression among women.

In particular, obese women who later developed depression were those with difficulty completing instrumental activities of daily living such as walking moderate distances and climbing stairs (physical impairment), less social support and more social strain (social dysfunction), and who coped with negative emotions by eating more, especially favorite foods (emotional eating). At the same time, depressed women who later developed obesity were those with more physical impairment and emotional eating. Social dysfunction, however, did not significantly mediate development of obesity from depression.

These mediators may be important targets for prevention and treatment of obesity-linked depression and depression-linked obesity. Further, multiple mediators suggest that the best interventions might be multifaceted. For example, mindfulness-based cognitive-behavioral

[1] When participants endorsed depressed mood and/or anhedonia lasting two weeks or more, 56.1% also reported an appetite decrease, 23.5% reported no appetite change, and 20.4% an appetite increase. Appetite change (coded −1 = decrease, 0 = no change, or 1 = increase) did not significantly moderate relations of the overall depressive symptom score at time 1 with obesity at time 3 (path c2, Fig. 1) or obesity at time 1 with the overall depression score at time 3 (path c1, Fig. 1) for women or men, *ps* > 0.50. Consequently, appetite changes were not included in the primary analyses.

[2] Analyses controlling ethnicity (white versus non-white) and socioeconomic status (the average of standardized ratings of level of education and household income) yielded substantively equivalent results. See Online Supplement 1.

therapy (Hofmann et al., 2010; Rogers et al., 2017; O'Reilly et al., 2014), behavioral activation therapy (Mazzucchelli et al., 2009; Pagoto et al., 2008; Busch et al., 2013), and interpersonal psychotherapy (Cuijpers et al., 2011; Tanofsky-Kraff et al., 2010, 2017) are psychosocial interventions that reduce both depression and obesity or problematic eating behaviors. Matching mediators identified in the current analyses, mindfulness, behavioral activation, and interpersonal psychotherapies may achieve good outcomes in part by acting on dysregulated emotion and behavior (including emotional eating), physical activity/impairment, and social dysfunction, respectively. Thus, combining or sequencing these interventions might be particularly useful in breaking the obesity-depression link among women.

Finding no statistically significant associations between obesity and depression among men replicated past research (Onyike et al., 2003; Pereira-Miranda et al., 2017). Why obesity and depression are more strongly associated among women than among men is unclear and an important topic for future research. Explanations suggested by past research include increased social pressure for thinness, greater sensitivity to social cues, and changes in estrogen levels among women (Pereira-Miranda et al., 2017). Based on the current results, additional hypotheses include that women experience more (or greater consequences for) social dysfunction, physical impairment, and emotional eating in response to initial depression or obesity.

Contributions of the current analyses include use of a national sample of adults to test multiple mediators of obesity-depression relations in a bidirectional model with three time points. Three time points separated potential causes, mediators, and outcomes (e.g., obesity→physical impairment→depression) to reduce the potential for spurious relations with fewer time points (e.g., cross-sectional correlations among obesity, impairment, and depression). Moreover, reflecting emerging complexity in the literature from prior studies of single mediators, the current analyses showed that physical impairment, social dysfunction, emotional eating each partly-independently accounted for connections between depression and obesity.

Nonetheless, important limitations temper the conclusions. For example, the specific mechanisms by which identified mediators may bring about changes in depression and obesity were not identifiable. In addition, the long time frame (18 years) may have precluded detection of connections between depression and obesity operating at shorter intervals. Finally, biological or somatic processes, which may reflect and/or contribute to the behavioral and psychosocial mediators identified here, could not be tested directly with the available survey and questionnaire data. To complement the current findings, future research might profitably test possible biological mechanisms suggested in previous research (Penninx et al., 2013).

The current analyses extended the literature on the manifold connections between obesity and depression among women (Atlantis et al., 2009; Preiss et al., 2013). Both conditions are leading public health problems producing great personal and economic costs (GBD 2015 Risk Factors Collaborators, 2016). Better understanding of the bidirectional development of obesity and depression via mediators such as physical impairment, emotional eating, and social dysfunction may speed development of more effective prevention and treatment interventions.

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Disclosure of interests

The author is a paid reviewer for UpToDate.

Appendix 1. Variance components estimated in multilevel models

Variable	Family variance	Residual variance
Obesity: Time 1	0.447	1.273
Depression: Time 1	0.516	3.205
Physical impairment: Time 2	0.198	0.576
Social dysfunction: Time 2	0.037	0.121
Emotional eating: Time 2	0.104	0.767
Obesity: Time 3	0.775	1.768
Depression: Time 3	0.287	2.625

Note. Family modeled as a random effect.

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