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Associations between depression and obesity in parents and their late-adolescent offspring: A community-based study

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Abstract

Objective—Major depressive disorder (MDD) and obesity are associated within individuals, but little is known about the association between MDD and obesity within families. We hypothesized that parental MDD would predict increased risk for adolescent obesity and that parental obesity would predict increased risk for adolescent MDD.

Methods—Participants were drawn from the community-based Minnesota Twin Family Study (total *n*=7307, 17-year-old *n*=3774). Parents and their 17-year-old offspring were assessed for MDD using a structured diagnostic interview, and direct assessments of height and weight were conducted (and diagnoses of obesity derived from these measurements).

Results—Parental MDD was associated with offspring obesity (Odds Ratio (OR)=1.74, 95% Confidence interval (CI)=1.24–2.46)). Odds ratios representing the risk associated with maternal and paternal MDD were similar (OR=1.42, CI=1.02–1.92; OR=1.40, CI=.88–2.20, respectively). This parental effect remained significant when adjusting for parental obesity and offspring MDD (OR=1.67, CI=1.18–2.37). Maternal obesity was associated with increased risk for MDD in offspring (OR=1.32, CI=1.06–1.64), but paternal obesity was associated with decreased risk for MDD among offspring (OR=.70, CI=.54–.91). These effects remained significant when adjusting for parental MDD and offspring obesity (OR=1.36, CI=1.07–1.73; OR=.65, CI=.49–.87, respectively). There were no differences in these findings by offspring sex (p-values for all tests of a sex interaction term >.374).

Conclusions—We found general support for hypothesized cross-disorder associations between MDD and obesity in parents and offspring, suggesting that a shared etiology may underlie these associations. Contrary to prediction, paternal obesity was associated with decreased risk for offspring MDD, a finding that requires further investigation.

Keywords

Depression; obesity; parents; offspring; adolescents; comorbidity

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Major depressive disorder (MDD) and obesity are associated (1,2), and people with one of these disorders at some point during their lifetimes are at approximately 1.5 to 2 times increased risk of having the other disorder (3–6). Although each of these disorders runs in families due to both genetic and environmental risk factors (7,8), very little is known about the possible association between MDD and obesity within families. The purpose of this study was to examine the association between MDD in parents and obesity in their late-adolescent offspring, and between obesity in parents and MDD in their late-adolescent offspring. Strengths of this study include its community-based sample, its use of study-assessed height and weight and MDD assessed via structured interviews, and the uniform age of offspring (late adolescence).

There are several reasons that such cross-disorder associations may exist within families. MDD and/or obesity in parents may create an environmental context that predisposes youth to both disorders. For example, parents may model depressive cognitive styles and/or unhealthy eating habits; they may also directly contribute to risk for this comorbidity by providing unhealthy food as an antidote to “feeling bad.” Obese parents may also model poor self-esteem (in themselves or by criticizing their overweight children), which could contribute to MDD symptoms in offspring. In addition, factors that characterize the entire family may contribute to cross-disorder associations between generations. For example, environmental stressors that affect the family (e.g., poverty, perhaps leading to both hopelessness and reduced access to healthy food) may contribute to risk for these disorders among all family members. In addition, shared biological factors, such as dysfunction of the stress response system (hypothalamic-pituitary-adrenal axis; 9) or chronic inflammation (10) may contribute to this form of comorbidity in families.

To date, there has been minimal research on the possibility that such cross-disorder associations may exist within families. Considering the possibility that parental MDD could be associated with obesity in youth, the evidence is mixed. In a questionnaire-based study, parental BMI was found to be associated with adolescent depression (11), but another study did not find an association between maternal depression and overweight status in childhood or early adolescence (12) and a final study (13) reported no association between maternal MDD and obesity in early-adolescent offspring. Considering the possibility that parental obesity could be associated with MDD in youth, the evidence is even more limited. However, supporting the notion that there could be such an association, morbid obesity among people presenting for weight-loss surgery is associated with a history of depression among first-degree relatives (14).

The goal of this study was to examine associations between MDD and obesity in parents and their late-adolescent offspring. Due to the hypothesized importance of early-onset cases of both MDD and obesity, we examined this issue in 17-year-old offspring and their parents (examining lifetime diagnoses of MDD and obesity in parents and their children up to this age). Hypotheses were derived from theory and previous research, with an emphasis on the reasons that cross-disorder associations may exist (discussed above). We expected that MDD in parents would be associated with obesity in offspring and that obesity in parents would be associated with MDD in offspring. We expected that these associations would remain significant once within-individual associations between these disorders were adjusted for

(for example, in an analysis examining links between parental obesity and offspring MDD, adjusting for parental MDD and offspring obesity), and our focus is on the results of this final set of analyses.

Methods

Participants

Participants were drawn from the community-based Minnesota Twin Family Study. Youth were recruited from birth records in the state of Minnesota during specified birth years, and the sample is representative of the population of the state during these years (15). Data were collected between 1990 and 2013. 83% of eligible families participated. The MTFS uses an accelerated longitudinal design with two cohorts: the younger cohort was first assessed at age 11 (n=1512) and followed-up every 3 years thereafter (retention was over 90% at each wave; mean age at age 17 assessment=18.2, SD=.7) and the older cohort was first assessed at age 17 (n=1252; mean age=17.5, SD=.5). In addition, a supplemental "enrichment sample" (ES) of 11-year-olds (n=998), screened such that some participants had higher risk for childhood externalizing disorders, started assessments later and were followed through age 17 (mean age=17.9, SD=.4). Because participants from all 3 cohorts were assessed similarly at age 17 (an age of theoretical interest, as discussed above), we combined the cohorts by using the available data from the age 17 assessment for all participants, thus maximizing power in this community sample.[1] Both mothers and fathers were assessed at the intake visit for each cohort, allowing us to examine cross-generation associations between these disorders in the entire sample. This meant that for the older cohort, parents were assessed at the same time as the youths' data were collected for this study (i.e., intake for both); for the younger and ES cohorts, parents were assessed 6 years earlier (i.e., at intake) than the information provided by the youth that was used in this study (i.e., from their age 17 follow-up assessment).

Participants gave written informed consent (parents for themselves and their minor children) and youth provided written assent. This study was approved by the University of Minnesota IRB.

Approximately 95% of the sample was White, consistent with the demographics of the population of the state of Minnesota at the time these youth were born. Additional information about the study design and participants is provided elsewhere (16).

Measures

MDD—For youth in the older MTFS cohort (first assessed at age 17), MDD was assessed on a lifetime basis using the Structured Clinical Interview for DSM-III-R (17). For youth in the

[1]Because part of the ES consisted of participants who were screened to be at higher risk for childhood externalizing disorders, we did two things to ensure that this sub-sample was not unduly affecting the results. First, were-ran all analyses excluding this sub-sample to ensure that the results applied to the unselected community sample that comprised the vast majority of participants. The pattern of significant results was identical to that of the primary results, with one exception: the association between MDD in mothers and obesity in offspring was non-significant due to reduced power (but the association between MDD in either parent and obesity in offspring remained highly significant, and the odds ratio for maternal MDD remained in the expected direction (OR=1.28, CI=.88–1.88). Second, as detailed in the Statistical Analysis section, we used probability weighting to adjust for the different sampling techniques used in the different cohorts.

younger MTFS cohort and the ES, MDD was assessed at ages 11 and 14 using the Diagnostic Interview Schedule for Children and Adolescents (DICA; 18) and at age 17 using the Structured Clinical Interview for DSM-III-R (17). Maternal reports of youth MDD were obtained using the DICA, and diagnoses reported by either the mother or the child were counted as present (the best-estimate method; 19). Kappa reliabilities of MDD diagnoses using the DICA and the SCID were both kappa=.89 (based on sub-samples of all participants in the MTFS who were assessed using these measures). At the age 11 assessment, lifetime episodes were assessed, while at the age 14 and 17 assessments, episodes that may have occurred since the previous assessment were assessed (and for youth who missed the age 14 assessment but participated at age 17, the age 17 interview inquired about symptoms that may have occurred since age 11). Maternal and paternal MDD were assessed using the SCID; if either parent had a lifetime history of MDD then parental MDD was considered present. Diagnostic interviews were conducted in person by trained interviewers with bachelor's or master's degree in Psychology, reviewed by teams of advanced clinical psychology doctoral students who achieved consensus agreement for each assessed symptom of MDD, and diagnosed using computer algorithms following DSM rules. Definite and probable (exhibiting all symptoms necessary for the diagnosis except one) diagnoses were used.

Obesity—Height and weight were assessed using a Detecto mechanical physician scale with height rod. Body mass index (BMI) was calculated using the standard formula (weight in kilograms divided by height in meters squared). For parents, the standard BMI cutoff of 30 was used to define obesity. If either parent met criteria for obesity, parental obesity was considered to be present. For youth, growth curves from the Center for Disease Control (20,21) were used to determine obesity cutoffs (95^{th} percentile) for each sex based on the average participant age at the assessment that occurred at approximately age 17; this yielded an obesity cutoff of 28.90 for males and 30 for females. Statistical Analyses

SAS version 9.2 was used to conduct generalized estimating equations (22) to account for the nested nature of the data (twins within families). Probability weighting was used to appropriately combine the ES sample (most of which was selected based on increased risk for externalizing disorders) with the rest of the sample (unselected). Details about the computation of the weights are provided elsewhere (23). We conducted two sets of analyses: one examining the association between parental MDD and offspring obesity, and the other examining the association between parental obesity and offspring MDD. For each set, analyses were conducted in stages. First, we assessed the overall association between the parental disorder (present in either parent) and the other disorder in offspring, adjusting for offspring sex and the other disorder in parents (e.g., the association between parental MDD and offspring obesity, adjusting for parental obesity and offspring sex). Second, we conducted this same analysis separately for (a) disorders in mothers and (b) disorders in fathers, again adjusting for offspring sex and the other disorder in the relevant parent. Third, we added an interaction term representing the effect of the youths' sex into each of these three models (i.e., a sex x obesity interaction term) in order to assess whether these associations between parental disorders and offspring disorders differed by offspring sex. When significant cross-generation associations were found, we repeated that analysis

adjusting for the "opposite" disorder in offspring in order to provide a strict test of the specific association between the two disorders. For example, if the association between parental MDD and offspring obesity was significant, we adjusted for offspring MDD (as well as parental obesity and offspring sex, as before) in this set of analyses. Finally, we examined potential interaction effects between maternal and paternal MDD in the prediction of offspring obesity, and between maternal and paternal obesity in the prediction of offspring MDD.

For all analyses, a cutoff of $p<.05$ was used to determine statistical significance, and odds ratios provided an indication of effect sizes. Because some full sample analyses were followed by analyses based on smaller subsamples, results that were significant at a trend level ($p<.10$) are also noted so that effects that were still strong but not significant in the subsample would be evident.

Because there were more missing data among fathers from non-intact families (biological parents not married at youth age 17), described below, we then re-ran analyses using only the sub-sample of intact families (75%) in order to examine whether the pattern of results from the primary analyses remained significant when only parents who were both biologically related to and reared their children were included. Intact families were those where the parents were living together at the time of the age-17 offspring assessment.

Finally, in order to ensure that the differential timing of parental assessments across the cohorts (at youth age 11 for the younger cohorts and youth age 17 for the older cohort) did not affect the results, we re-ran all analyses including a cohort-by-independent variable interaction term (as well as a term representing the main effect of cohort). In all cases the interaction term was non-significant (lowest p-value=.25), indicating that the timing of the parental assessment did not affect the association between the independent and dependent variable.

Results

Missing data

All participants were included in all analyses for which they provided data (e.g., a participant who provided MDD data but not obesity data was still included in analyses involving MDD).

Among 17-year-olds, 12% were missing data on MDD by age 17 and 20% were missing data on obesity at 17. Missing data on MDD by 17 was not associated with parental MDD, parental obesity, or youth obesity ($p>.05$ for all). Missing data on obesity at 17 was associated with increased rates of MDD by 17 but negatively associated with parental MDD and parental obesity (so offspring of parents with MDD and/or obesity were more likely to contribute obesity data at age 17). Families in which data were present for 1 twin but not the other were rare: 0.01% for obesity and 0.05% for MDD.

Among mothers, 0.7% were missing MDD data and 2.3% were missing obesity data. Missing MDD data was not associated with the presence of obesity, and missing obesity data was not associated with the presence of MDD.

Among fathers, 10.3% were missing MDD data and 17.7% were missing obesity data. Missing MDD data was not associated with the presence of obesity, but missing obesity was associated with the presence of MDD. Most of the additional missing obesity data was due to fathers, primarily those from non-intact families, participating by phone (19% of fathers from non-intact families, compared to 3% from intact families).

To summarize, there was little evidence of important missing data effects, though youth and fathers with histories of MDD were more likely to have missing obesity data, and fathers from non-intact families were more likely to have missing obesity data (due to participating by phone). We examined the significance of this latter pattern (see below).

Descriptive analyses

Prevalence rates of MDD and obesity among youth (drawn from raw, unweighted data) are presented in Table 1, by the presence/absence of disorders among parents. There was a general trend for offspring of parents with one disorder to be at increased risk for the other disorder. Cross-disorder associations between parents and offspring

Results of analyses examining associations between parental MDD and offspring obesity and between parental obesity and offspring MDD are presented in Table 2. All analyses used weighted data.

Parental MDD and offspring obesity—Parental MDD was positively associated with offspring obesity (OR=1.58, CI=1.10–2.28). Odds ratios for the risk associated with maternal (1.37) and paternal (1.39) MDD were similar though non-significant. The effect for overall parental MDD remained significant after adjusting for parental obesity and offspring MDD (OR=1.57, CI=1.01–2.45, p=.047). There were no differences by offspring sex, and no significant interaction effect between maternal and paternal MDD.

Parental obesity and offspring MDD—Results for parental obesity were more complex, with maternal obesity being associated with increased risk for MDD in offspring (OR=1.34, CI=1.07–1.69) and paternal obesity being associated with decreased risk for MDD among offspring (OR=.68, CI=.52–.90). These effects remained significant after adjusting for parental MDD and offspring obesity (maternal obesity-offspring MDD: OR=1.41, CI=1.09–1.81, p=.08; paternal obesity-offspring MDD: OR=.62, CI=.46–.85, p=.003). There were no differences by offspring sex, and no significant interaction effect between maternal and paternal obesity.

Analyses using intact families only

Due to concerns about higher rates of missing obesity data among fathers from non-intact families, we re-ran analyses including only families in which the biological parents were (still) married at youth age 17. Among these families, rates of missing data were low: 1.4% of mothers and 5.9% of fathers had missing obesity data, while 0.5% of mothers and 3.6%

of fathers had missing MDD data. The results were broadly similar to those of the primary analyses. Specifically, the parental MDD - offspring obesity OR dropped from 1.58 in the full sample to 1.49 (CI=.92–2.42, p=.104) in the intact families. In addition, the ORs for the maternal (OR=1.12, CI=.67–1.87, p=.662) and paternal (OR=1.98, CI=1.04–3.76, p=.038) effects generated values with confidence intervals that overlapped those for mothers and fathers when using the full sample (see Table 2).

Conclusions

The results of this study indicate that even once within-person comorbidity is adjusted for, there are significant cross-disorder associations between MDD and obesity in parents and offspring. The pattern of findings was largely as expected, with parental MDD consistently associated with increased risk for obesity among offspring and maternal obesity being associated with increased risk for MDD among offspring. However, paternal obesity was associated with *decreased* risk for MDD among offspring. Importantly, these associations generally remained significant when the "opposite" disorder among parents and youth was adjusted for, making it clear that these links were not simply due to within-person comorbidity between these disorders. No differences in these patterns were found by offspring sex.

There are many possible explanations for the increase in risk for obesity among offspring of MDD parents and the increase in risk for MDD among offspring of obese mothers. Although investigating mediators and moderators was beyond the scope of this investigation, we suggest that future research focus on three areas. First, it seems possible that environmental stressors that affect the whole family, perhaps poverty and/or the effects of living in an impoverished neighborhood, could contribute to this comorbidity. Second, shared biological risk factors (such as HPA axis disruption or inflammation), which may be genetically influenced, could represent a fruitful avenue for future research. Finally, psychological and/or parenting factors could account for these associations. For example, parents with MDD and/or mothers with obesity may utilize parenting styles, or model coping mechanisms, that contribute to risk for the other disorder among offspring.

The one inconsistent finding to emerge from our results, that offspring of obese fathers tend to have a reduced risk for MDD, requires replication and further exploration. One possibility is that this is a coincidental byproduct of the fact that fathers who are obese have different rates of rearing their children compared to fathers who are non-obese (and parental separation/divorce is associated with increased risk for depression). Indeed, in our sample, fathers from intact families (i.e., biological parents still married) were more likely to be obese than fathers from non-intact families, and youth from non-intact families were more likely to have histories of depression. Once we adjusted for intact versus non-intact status of the family, there was no significant association between paternal obesity and youth MDD. Also related to this issue was the fact that fathers from non-intact families frequently participated by phone (instead of in person), resulting in more missing data for paternal height and weight (and therefore obesity) among fathers from non-intact families. We followed-up on this finding by repeating all analyses using only participants from intact families, and although most results mirrored those using the entire sample, the association

between paternal obesity and a reduced risk for offspring MDD was non-significant. Therefore, among families in which fathers rear their children until age 17, there appears to be no significant association between paternal obesity and risk for offspring MDD. We encourage future research to examine associations among paternal obesity, father-child involvement, and risk for MDD among youth.

These findings are consistent with the findings of Eley et al. (11), but Mamun et al. (12) and Richardson et al. (13) did not find associations between depression in parents and overweight (12) or obesity (13) in offspring. This could be because in the Mamun et al. (12) study, only early-adolescent overweight was examined (to age 14), or because maternal depression was operationalized by endorsing 3 out of 7 depression symptoms on the Delusions-Symptoms-States Inventory (no reference provided) at the time the children were 5 years old. Similarly, the Richardson et al. (13) study only examined the association between maternal depression (assessed via questionnaire at 4 assessments) and early-adolescent obesity. Integrating these results, it is possible that this association between maternal depression and offspring obesity only emerges in mid- or late adolescence. It is also possible that some episodes of maternal depression were missed in these other studies because they were not ongoing at the exact times that the assessment(s) occurred.

Strengths of this study included the community-based sample (thereby avoiding the biases inherent in clinical samples), the uniform late-adolescent age of the offspring (thereby detecting early-onset cases of MDD and obesity but avoiding the possible attenuation of effects if these associations differ by age), and the direct clinical assessments of MDD and obesity in both parents and youth. This study has limitations, however. The sample was overwhelmingly White; the results may not be generalizable to families from other racial or ethnic backgrounds. In addition, these effects may vary by development (e.g., later-onset cases of MDD and/or obesity may be related to different influences), and these findings are limited to those offspring cases that emerge by late adolescence. For the younger and ES cohorts, parents' assessments occurred 6 years prior to the youths' assessments that were used in this study (parent data was from the intake assessment at youth age 11, while youth data was from the age 17 follow-up assessment). It is possible that some parents' weights may have changed, or they may have developed MDD, during that 6-year period. However, there were no significant differences in the rates of MDD or obesity among parents in the younger and ES cohorts compared to the older cohort and no differential associations between the independent and dependent variables in these analyses by cohort; therefore, we do not believe that this methodological difference affected the results.

In conclusion, we found support for cross-disorder associations between MDD and obesity in parents and late-adolescent offspring, with either disorder in parents broadly being associated with increased risk for the other disorder in offspring. This pattern is suggestive of a shared etiology between these conditions. One finding was inconsistent with this pattern: obesity in fathers was associated with decreased risk for MDD in youth, a finding that may relate to differences in family structure or levels of paternal involvement in child-rearing; this result awaits replication and further study. More broadly, future research into potential environmental, psychological, and/or biological explanations for these cross-

disorder associations is warranted and could inform the development of family-based intervention and prevention programs for these disorders.

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Glossary

MDD	major depressive disorder
OR	odds ratio
CI	confidence interval

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Table 1

Cross-disorder associations between obesity and MDD among parents and youth

	Obesity in youth (n=259, 9%)		Major Depressive Disorder in Youth (n=604, 18%)	
	With parental disorder	**Without parental disorder**	**With parental disorder**	**Without parental disorder**
Parental MDD (n=1550, 44%)	126 (10.1%)	105 (6.6%)	344 (25.2%)	226 (12.8%)
Maternal MDD (n=1168, 32%)	106 (11.0%)	153 (7.5%)	289 (27.5%)	312 (13.9%)
Paternal MDD (n=612, 18%)	47 (9.9%)	174 (7.7%)	117 (22.7%)	403 (16.1%)
Parental obesity (n=1804, 55%)	203 (13.5%)	30 (2.6%)	282 (17.5%)	220 (16.7%)
Maternal obesity (n=1150, 31%)	160 (16.8%)	96 (4.8%)	221 (21.7%)	368 (16.4%)
Paternal obesity (n=1038, 33%)	125 (14.4%)	72 (4.3%)	128 (13.6%)	322 (17.6%)

Raw prevalence rates presented in this table (these rates are not adjusted for the fact that there are two offspring per parent). N's presented are the number of each type of participant with each disorder (e.g., 259 youth had obesity, 1168 mothers had MDD). Percentages presented are the percentage of that type of participant who had that disorder (e.g., 9% of youth overall had obesity, while 10.1% of youth who had a parent with MDD had obesity and 6.6% of youth who did not have a parent with MDD had obesity). N's and percentages for "Parental" variables were only calculated if enough data on both parents was present (each disorder was considered present in the parent(s) if at least 1 parent had it, and was considered absent in the parents if both parents contributed data and did not have it).

Table 2

Associations between MDD and obesity in parents and late-adolescent offspring

	Youth Obesity		Youth Major Depressive Disorder	
	Main Effect OR (95% CI)	**Interaction by Sex (estimate (SE))**	**Main Effect OR (95% CI)**	**Interaction by Sex (estimate (SE))**
Parental MDD	1.58 * (1.10–2.28) p=.013	.31 (.37) p=.408		
Maternal MDD	1.37 § (.97–1.94) p=.077	.03 (.35) p=.925		
Paternal MDD	1.39 (.86–2.27) p=.183	−.11 (.49) p=.823		
Parental obesity			1.01 (.80–1.29) p=.908	.23 (.25) p=.372
Maternal obesity			1.34 * (1.07–1.69) p=.011	.07 (.24) p=.759
Paternal obesity			.68 ** (.52–.90) p=.007	−.03 (.30) p=.924

§ p<.10;

* p<.05;

** p<.01

Odds ratios (for main effects) and parameter estimates (for interaction effects) are derived from generalized estimating equations, which model the associations between variables while adjusting for the nested nature of the data (twins nested in families). Weights were applied to adjust for the fact that most youth were from an unselected community sample but some were from a selected sample (the Enrichment Sample). All analyses adjust for the other disorder in parents. All significant results remained significant when adjusting for the other disorder in the child.