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Relationship between depressive mood and eating disorders in a non-clinical young female sample: A one-year longitudinal analysis of cross-lagged and simultaneous effects

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ABSTRACT

Although it is generally agreed that eating disorders (EDs) and depressive mood (DM) are related, the main ambiguity arises from difficulties in determining their cause–effect relationships. The aim of this study was to examine the longitudinal reciprocal causation between EDs and DM among female students. Several models (cross-lagged effects and simultaneous effects) were tested in order to disentangle the prospective relationships between DM (measured by the Beck Depression Inventory—Short Form) and EDs (measured by the Eating Attitudes Test—26) using structural equation modeling with latent variables on one-year longitudinal data. A total of 567 female students were interviewed at the beginning of the first university year (T1); 373 of them were re-interviewed 6 months later (T2), and 359 were re-interviewed after a further 6 months (T3). The results support (1) the prospective reciprocal effects model and (2) the simultaneous reciprocal effects model. The implications of the findings in terms of theoretical improvements and effective treatments are discussed.

1. Introduction

Eating disorders (EDs) are probably one of the most common psychiatric problems faced by adolescent girls and young adult women (Fairburn & Harrison, 2003; Goss & Fox, 2012; Machado, Machado, Gonçalves, & Hoek, 2007), and are generally associated with psychological distress, and depression (Fennig & Hadas, 2010; Mischoulon et al., 2011). It is now widely accepted that the cultural ideal of thinness may be a source of eating disorders as well as depression[1] among women (McCarthy, 1990; Polivy & Herman, 2002). Thin-ideal internalization, which occurs at a very early age (Hayes & Tantleff-Dunn, 2010), places women in an ongoing and obsessive self-evaluation of body image, leading to body dissatisfaction and psychological distress (Fitzsimmons-Craft, 2011; Stice, Hayward, Cameron, Killen, & Taylor, 2000; Thompson & Stice, 2001). Gunnard et al. (2012) found that a high self-standard in physical appearance was a principal feature of EDs. Ferreiro, Seoane, and Senra (2011) found that body dissatisfaction was a significant predictor of both depressive symptoms and disordered eating. In a 6-month prospective study with a sample of female freshmen, Bradford and Petrie (2008) showed that thin-ideal internalization predicted subsequent body dissatisfaction, while negative body image predicted subsequent depressive mood. Although it is generally agreed that EDs and depressive mood (DM) are related, the main ambiguity arises from difficulties in determining their cause–effect relationships (Fox & Power, 2009; Levy & Dixon, 1985; O'Brien & Vincent, 2003). The aim of this study was to examine the longitudinal reciprocal causation between EDs and DM among young adult women. Clarification of the directionality of causal effects among these constructs may lead to some important theoretical improvements in their understanding, and hence to more effective treatments aimed at reducing emotional distress.

1.1. EDs and DM: which disorder predates the other?[2]

Some authors argue that EDs are an affective disorder variant to which adolescent girls and young adult women are vulnerable (Hinz & Williamson, 1987). These authors argue that EDs are the consequences of negative emotions (Heatherton & Baumeister, 1991). For example, in an 8-month longitudinal study among female adolescents, Wertheim, Koerner and Paxton (2001) found that a higher intensity of baseline depressive symptoms predicted eating problems. Analyzing

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[1] Throughout this article, *depression and depressive mood are used interchangeably as a* dimensional, not a categorical, construct (Flett, Vredenburg, & Krames, 1997; Hankin, Fraley, Lahey, & Waldman, 2005). Thus, we are interested in individual differences in depressive mood rather than a clinical category such as a major depressive disorder.

[2] We limited our review to prospective studies of non-clinical populations because clinical referral affects the stability and the relationships between EDs and DM.

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8-year longitudinal data from a sample of adolescents, the study performed by Johnson, Cohen, Kotler, Kasen, and Brook (2002) revealed that depressive disorders during early adolescence were associated with an elevated risk of the onset of EDs. Another 4-year prospective study carried out by Dobmeyer & Stein (2003) showed that depressed mood was related to an increased severity of eating disorder symptoms in a sample of female undergraduates. In an 8-month longitudinal study with a sample of female students during their first year at university, Gilbert and Meyer (2005) showed that depression predicted an increase in bulimic attitudes. In a 16-month longitudinal study with a sample of adolescents, McCabe and Ricciardelli (2006) showed that changes in levels of depression were the strongest predictors of extreme weight change behaviors (e.g., drive for thinness, bulimia) among girls (mean age: 13.33 years). Analyzing 6-year longitudinal data from a sample of female adolescents, Measelle, Stice, and Hogansen (2006) found a unidirectional relationship between depression and EDs, wherein initial levels of depression predicted future growth in EDs. More recently, analyzing 5-year prospective data from a sample of women experiencing the transition from adolescence to adulthood, Ferriter, Eberhart, and Hammen (2010) found that higher levels of baseline depressive mood significantly predicted increased risk of EDs. A deficit in emotion regulation (Haynos & Fruzzetti, 2011) as well as an emotion-processing deficit (Bydlowski et al., 2005) have been suggested as potential explanations. Thus, as a distraction from adverse emotions, EDs may constitute a maladaptive coping strategy (Heatherton & Baumeister, 1991).

On the other hand, some authors argue that depressive mood is a consequence of EDs. Analyzing 4-year longitudinal data from a sample of female students, Stice et al. (2000) found that EDs predicted the onset of depression. Similarly, analyzing data from adolescent girls assessed at ages 11, 14 and 17, Marmorstein, von Ranson, Lacono, and Malone (2008) found that EDs predicted later depressive symptoms. One explanation is that EDs may cause feelings of shame, guilt, or dysphoria (Stice, 1998), increasing the risk of depression. Another explanation is that caloric deprivation, which leads to an elevation in corticotropin-releasing hormone, promotes depressive mood in women with EDs (O'Brien & Vincent, 2003).

However, there is also evidence that EDs and depressive mood have reciprocal relationships. Analyzing 8-year longitudinal data from a community sample of female adolescents, Presnell, Stice, Seidel, and Madeley (2009) showed that depression predicted future increases in EDs, and that EDs likewise predicted future increases in depression. In a 6-month prospective study with a sample of female freshmen, Bradford and Petrie (2008) found a reciprocal relationship between EDs and DM. These findings replicated those of the prospective data examined by Stice, Burton, and Shaw (2004) showing that bulimic pathology and depression were reciprocally related. More recently, results from a prospective study among adolescents and young adult women conducted by Skinner, Haines, Austin, and Field (2012) showed that depressive mood at baseline was strongly predictive of the onset of EDs, while EDs predicted the development of high levels of depressed mood during 2 years of follow-up. The serotonin connection was suggested here as a potential explanation (Steiger, 2004). In fact, the serotonin transporter (5-HT) has been associated with both EDs (Bailer & Kaye, 2011) and DM (Meltzer, 1990). Analyzing prospective data from adolescent girls, Mata and Gotlib (2011) found that the serotonin transporter gene (5-HTTLPR) moderated the relationship between change in depressive mood and bulimic pathology.

1.2. Purpose of this study

To summarize, there is some evidence that EDs promote depressed mood, and it is theoretically and empirically plausible that DM promotes EDs, or that these two constructs are reciprocally related. The aim of the present study was to contribute to this debate by testing several models (i.e. cross-lagged effects and simultaneous effects) in order to disentangle the prospective relationships between depressive mood and disordered eating using structural equation modeling with latent variables on one-year longitudinal data collected from female students. In sum, this study explored competing hypotheses regarding the temporal relations between DM and EDs (Figs. 1 and 2).

2. Method

2.1. Participants and procedure

This research used data from a longitudinal study on mental health among French freshmen students in a large public university. Participants were prospectively recruited at the beginning of their first year of university. At T1, they filled out our questionnaire at the University Medical Care Center, which approved our research protocol, during their mandatory medical check-up. The study was approved by the Ethical Committee of this institution and signed informed consent was obtained from all the participants. At T2 (six-month follow-up) and at T3 (one-year follow-up), students were telephoned and a questionnaire was sent to them by post or e-mail according to their choice. A total of 567 female students were interviewed at the beginning of the university year (T1); 373 of them (66%) were re-interviewed 6 months later, at the end of the university year (T2), and 359 (of 373) were re-interviewed after a further 6 months, at the beginning of the following university year (T3). Thus, only data based on the 359 female students who filled out the questionnaire at T1, T2 and T3 were included. The mean age was 18.7 ($SD = 1.3$) years at T1. Among these 359 participants, 47 were students in Sciences (13%), 104 in Medicine (29%), 140 in Art and Humanities (39%) and 68 in Law and Economy (19%). Concerning their living conditions, 143 participants have declared to live alone (40%), 143 lived at their parents' home (40%) and 73 lived with roommates or in couple (20%).

Analyses of variance were conducted to determine differences between dropouts and freshmen who completed the questionnaires at T1 and T3. They did not differ significantly for socio-demographic variables, body mass index, eating disorders and depressive mood, suggesting that bias was not introduced due to attrition.

2.2. Measures

2.2.1. Depressive mood

Depressive mood was measured using the Beck Depression Inventory—Short-Form (BDI-SF; Beck & Beck, 1972) which is a 13-item, abbreviated version of the 21-item, self-report BDI screening test for depression (Beck et al., 1961). The BDI-SF is suitable for use in non-clinical populations (aged 13 and over) where brief and quantifiable screening tests are required. It is scored as an index of severity of depressed mood. Individual items assessed on the BDI-SF include mood, pessimism, sense of failure, lack of satisfaction, guilty feelings, self-hate, self-punitive wishes, social withdrawal, indecisiveness, body image, work, inhibition, fatigability and loss of appetite. Each of the 13 items is scored from 0 to 3. The BDI-SF appears to have a level of internal consistency comparable to that of the long form (Beck, Steer, & Garbin, 1988). In the present sample, Cronbach alphas of the BDI were .82 at T1, .88 at T2, and .84 at T3.

At T1, T2 and T3, the mean scores for the BDI-SF were 4.60 ($SD = 4.28$), 6.12 ($SD = 6.00$), and 4.83 ($SD = 4.61$), respectively.

2.2.2. Eating disorders

Disordered eating was assessed using the Eating Attitudes Test (EAT-26) (Garner et al., 1982), which forms three subscales: dieting, bulimia, and oral control. Participants responded to each item with one of six choices: *never* (0), *rarely* (0), *sometimes* (0), *often* (1), *very often* (2), or *always* (3). Although the EAT-26 does not provide a diagnosis of an eating disorder, higher scores indicate greater disturbances in eating attitudes and behaviors (i.e. eating disorder risk). In the present sample, Cronbach alphas of the EAT-26 were .88 at T1, .83 at T2, and .87 at T3.