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What is This?

Predictors of disordered eating in adolescence and young adulthood: A population-based, longitudinal study of females and males in Norway

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Abstract

We investigated longitudinal predictors for disordered eating from early adolescence to young adulthood (12–34 years) across gender and different developmental phases among Norwegian young people. Survey data from a population-based sample were collected at four time points (T) over a 13-year time span. A population-based sample of 5,679 females and males at T1 and T2, 2,745 at T3 and 2,718 at T4 were included in analyses, and linear regression and random intercept models were applied. In adolescence, initial disordered eating and parental overprotectiveness were more strongly related to disordered eating among females, whereas loneliness was a stronger predictor for adolescent males. Initial disordered eating during early adolescence predicted later disordered eating more strongly in late- than mid-adolescence. In young adulthood, no significant gender-specific risk factors were found. The findings provide support for both shared and specific risk factors for the developmental psychopathology of disordered eating.

Keywords

developmental phases, disordered eating, eating problems, gender differences, predictors, risk factors

Eating disorders, particularly anorexia nervosa, have the highest mortality of any mental illness, with an estimated rate of 5.1 deaths per 1,000 person-years (Smink, van Hoeken, & Hoek, 2012). Eating disorders show high co-morbidity with conditions such as major depression, anxiety disorders, and multiple physiological complications (Fairburn & Harrison, 2003), and carry unacceptably high personal, family, and social costs (Simon, Schmidt, & Pilling, 2005).

Disordered eating is usually defined as abnormal eating behaviors or symptoms of eating disorders that are less severe than eating disorders as defined by DSM-IV-TR (American Psychiatric Association, 2000). Despite the lower degree of severity, disordered eating is an important predictor of eating disorders (Stice, 2002), and can also develop into full-blown eating disorders (Shisslak, Crago, & Estes, 1995). Furthermore, disordered eating is associated with a number of psychosocial impairments, including depressive symptoms, lowered self-esteem, substance abuse, suicidal behaviors, and impaired functioning socially and at work (Haedt & Keel, 2010; Mond et al., 2006; Shisslak et al., 1995). These findings show the public health importance of both eating disorders and disordered eating.

Sociocultural, familial, personality and biological risk factors have all been hypothesized to contribute to the development of disordered eating symptoms across development (Rosen, 1996). However, limited attention has impeded a full understanding of the impact of developmental factors on disordered eating (Jacobi, Hayward, de Zwaan, Kraemer, & Agras, 2004; Stice, 2002). To the best of our knowledge, no longitudinal study has previously examined whether risk factors vary across different developmental phases in both males and females. Understanding risk across developmental phase and gender could have important implications for

the developmental psychopathology of disordered eating and the development of targeted prevention programs. Therefore, using data from a large population-based longitudinal study in Norway, we examined gender- and developmental phase-specific predictors of disordered eating from early adolescence through young adulthood.

Development occurs in sequences of phases with systematic changes in the individual's physical, cognitive, emotional, social, and behavioral development. Early patterns of adaptation in early developmental phases tend to promote adaptation in later phases, and early forms of maladaptation tend to lead to later maladaptation (Cicchetti & Rogosch, 2002; Cummings, Davies, & Campbell, 2000). This concept is the main principle in developmental psychopathology, a valuable framework to understand the development of eating problems (Rosen, 1996).

According to the developmental psychopathology perspective, examining changes in symptomatology and risk factors across development improves our understanding of causal processes (Rutter, 1988, 1989), and risk variability depending on the individual's age or development level (Rutter, 1988). Childhood risk

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factors may stem from a different context from that which is seen in adolescence or adulthood (Rosen, 1996). In pre-adolescence, for example, maternal dieting is a strong risk factor for excessive dieting, whereas in adolescence, appearance dissatisfaction is a strong risk factor for excessive dieting and other disordered eating behaviors (Hill, 1993).

Developmental transitions or changes may contribute to risks for physical and mental well-being, and the changes associated with the transition from childhood to early and mid-adolescence may confer a particular vulnerability to risk of psychopathology, including disordered eating (Graber, Brooks-Gunn, Paikoff, & Warren, 1994). For example, the onset of puberty and transition to adolescence leads to biologically pre-determined weight gain and appetite shifts that may result in body dissatisfaction, low self-esteem, and subsequent dieting (Jacobi et al., 2004). The transition to adolescence is also related to more autonomy and independence, which changes adolescent–parent relationships; there is increased perceived parental conflict and decreased parental support (De Goede, Branje, & Meeus, 2009). New academic and social demands placed by the transition to new schools (junior/middle schools) may add responsibilities that stress coping capacities and compromise adjustments, which further exacerbate to engage in disordered eating (Smolak & Levine, 1996; Smolak, Levine, & Gralen, 1993). All these changes may contribute to the development of disordered eating during early/mid-adolescence. Late adolescence marks the transition to young adulthood, which includes leaving home and/or attending college, reduced social support, increased dating and sexual pressures, academic and career pressures, and identity exploration and instability (Smolak & Levine, 1996). These stressors may play a unique role in the development of disordered eating during late adolescence or young adulthood. The meaning of risk factors and predictors shifts across development.

Although some risk factors for disordered eating, such as dieting, body dissatisfaction, negative affect (mostly depressive symptoms), perfectionism, impulsivity, and substance abuse, have been repeatedly documented in longitudinal studies (Jacobi et al., 2004; Stice, 2002), little is known about whether these risk factors or other correlates are limited to specific developmental phases or are active across the life span (Jacobi et al., 2004). In particular, there are no longitudinal studies that have examined whether there are differences in the prospective predictors of disordered eating when comparing mid-adolescence, late adolescence, and young adulthood.

With respect to gender differences, both eating disorders and disordered eating have been consistently found to be more common in females than males (Jacobi et al., 2004), and these gender differences tend to escalate in adolescence, mostly after puberty. This has been attributed to the increase in body fat that accompanies maturation, which inevitably shifts girls further away from the thin ideal endorsed in society, with unhealthy weight-regulating practices being a result (Keel, Fulkerson, & Leon, 1997). The transition to adolescence is also associated with a greater drop in self-esteem and an increase in depression for girls than for boys, which may add further risks (Hankin et al., 1998; Smolak & Levine, 1996; Smolak et al., 1993). Also, the higher level of the hormone estradiol during puberty influences body fat composition and appetite, and may trigger disordered eating behaviors among girls (Klump, Keel, Sisk, & Burt, 2010).

Differences in gender roles and expectations, particularly for thinness and attractive body appearance, are also important factors in explaining why eating disorders are more prevalent in females than in males (Culbert, Racine, & Klump, 2011; Murnen & Smolak, 1997; Smolak & Striegel-Moore, 1996). According to gender socialization theory, the social environment is an important channel for the modeling of gender roles, where women are represented as being dependent and lacking autonomy and competency (Worell & Todd, 1996). These negative aspects of gendered social roles may lead to lowered self-worth, increasing self-concept instability, and increasing reliance on others for approval of physical attractiveness, which may in turn stress adolescent girls and women to attain the ideal of thinness and engage in disordered eating behaviors.

Similarly, self-objectification theory offers an important explanation for gender differences in disordered eating. This theory posits that girls and women are socialized to view and treat their bodies as objects and to be preoccupied with the thin ideal, which in turn leads to body shame or raises concerns about their physical appearance, and consequently, to disordered eating, regardless of their level of body dissatisfaction (Fredrickson & Roberts, 1997; Fredrickson, Roberts, Noll, Quinn, & Twenge, 1998).

However, gender differences on the level of disordered eating may not necessarily imply differences between females and males in how strongly risk factors are associated with disordered eating. There are similarities in the factors associated with disordered eating in adolescent boys and girls (Ricciardelli & McCabe, 2004). These factors include body mass index (BMI), negative affect, self-esteem, perfectionism, drug use, and perceived pressure to lose weight from parents and peers. With the exception of BMI, most of these correlates have only been found in cross-sectional studies (Ricciardelli & McCabe, 2004).

On the other hand, recent longitudinal studies suggest that low self-esteem, perfectionism, body dissatisfaction, and high BMI may be more prominent risk factors for adolescent girls than boys (Beato-Fernandez, Rodriguez-Cano, Belmonte-Llario, & Martinez-Delgado, 2004; Ferreiro, Seoane, & Senra, 2011; McCabe & Ricciardelli, 2006), whereas symptoms of depression have been reported to be more prominent for adolescent boys (McCabe & Ricciardelli, 2006). Nevertheless, most studies did not examine whether the gender differences were statistically different for males and females. Moreover, research in this area has typically used small samples with differing participant characteristics and study methodologies, which may further explain the inconsistent findings across studies.

Examining whether risk factors for disordered eating differ by gender or developmental phase may increase our understanding of the developmental psychopathology of disordered eating and open the possibility of designing targeted preventive interventions for disordered eating on the basis of early identifiable characteristics. For this reason, we aim to: (1) determine the longitudinal relationship of predictors for disordered eating by examining these associations in mid-adolescence, late adolescence, and young adulthood, for both males and females; and (2) examine whether gender and developmental phases moderate the relationships between predictors and disordered eating. This study includes putative biopsychosocial risk factors of disordered eating that are documented in the literature, such as appearance dissatisfaction, BMI, pubertal development, negative affectivity, self-worth, and substance use behaviors (Jacobi et al., 2004; McCabe & Ricciardelli, 2004; Stice, 2002); loneliness (Levine, 2012); self-concept instability (Kansi, Wichstrom, & Bergman, 2003); and parental bonding styles (Tata, Fox, & Cooper, 2001).

Method

Procedure and participants

Data were analyzed from the Norwegian longitudinal study "Young in Norway," which was conducted at four time points: 1992 (T1), 1994 (T2), 1999 (T3), and 2005 (T4). The initial sample at T1 was composed of 12,655 students in grades 7 to 12 (12 to 20 years of age) at 67 representative schools in Norway, with each grade being equally represented. Every school in the country was included in the register from which the schools were selected, and the sample was stratified according to geographical region and school size, which in Norway is closely related to the degree of urbanization. Each school's sampling probability was proportional to the number of students at the school, thus providing an equal probability of selection for each student. The response rate at T1 was 97% ($N = 12,287$).

In 1994, three of the participating schools at T1 were not part of the follow-up study (T2; age 14 to 22), and at another school, a burglary in the school's archives resulted in the loss of the project's identification records. In total, 9,679 students at 63 schools were eligible to complete the T2 questionnaire. Since a considerable proportion of the students had completed their three-year track at the junior or senior high school that they were attending at T1, the subjects who were no longer at the same school at T2 received the questionnaire by mail. For this group, the response rate was 68% ($N = 3,783$), whereas those at their original schools had a response rate of 92% ($N = 4,187$). The overall response rate at T2 was 79%.

At T3, only students who completed the questionnaire in school at T2 ($N = 3,844$) were followed up due to a comparatively lower response rate among those receiving the questionnaire by mail. As such, those who responded by mail at T2 ($N = 3,783$) were not included in the follow-up at T3 and T4. Since the survey was originally planned as a two-wave study, informed consent had to be obtained again at T2. Out of the total number of consenting individuals at T2 ($N = 3,507$, 91.2%), 2,923 (83.8%) responded to the questionnaire that they received by mail at T3 (age 19 to 28), representing an overall response rate of 68%.

In 2005 (T4), all persons who had consented to the follow-up at T2 were again invited to participate (age 25 to 34). In total, 2,890 of 3,507 potential participants, or 82.4%, completed the questionnaire, resulting in an overall response rate of 67%.

In this study, after excluding participants over 17 years of age at T1 ($N = 3,835$), and those with invalid data for various reasons, the actual sample sizes were 5,679 at T1 and T2, 2,745 at T3, and 2,718 at T4 (see Figure 1 for a graphical representation of the selected samples). Moreover, to examine longitudinal associations between predictors and disordered eating during young adulthood (i.e., combined T3 and T4 samples), participants who responded at both time points were included ($N = 2,164$).

Even though a large proportion of the sample that did not respond to the questionnaires at T3 and T4 were planned nonresponders, analyses were conducted to explore the potential impact of variables on attrition. More specifically, we performed a multiple logistic regression to investigate whether variables at T1 predicted dropout at T2, T3, or T4. The results of these analyses revealed that older age, male gender, more occasions of alcohol intoxication over the past year, and higher perceived parental overprotectiveness significantly predicted higher odds for dropout at T2, T3, or T4 ($p < 0.05$). Lower scores for parental care and loneliness also predicted dropout ($p < 0.05$).

Measures

All predictors and outcomes were based on adolescent self-report.

Outcome variable. Disordered eating was assessed by the Eating Attitudes Test-12 (EAT-12), an instrument designed to measure eating problems and concerns related to dieting, bulimia, and food preoccupation and oral control (Garner, Olmsted, Bohr, & Garfinkel, 1982; Lavik, Clausen, & Pedersen, 1991). The response alternatives range from 1 ("never") to 4 ("always"). EAT-12 was constructed from the 26-item version of the EAT by selecting the items with the best psychometric properties (Garner et al., 1982; Lavik et al., 1991). Mean scores were calculated, with high scores reflecting high levels of disordered eating. The scale showed a good internal consistency (Cronbach's α) at each survey point: .73, .77, .77 and .75 at T1, T2, T3, and T4, respectively.

Predictor variables. Appearance satisfaction was assessed by the Body Areas Satisfaction Scale (BASS) (Brown, Cash, & Lewis, 1989), which rates respondents' level of satisfaction with seven items or body areas: face, lower torso, mid-torso, upper torso, muscle tone, weight, and height, with the response options varying from 1 ("very dissatisfied") to 5 ("very satisfied"). A mean score was computed, with high scores indicating a high level of satisfaction, and the scale demonstrated a good internal consistency at each survey point with α = .80, .81, .82, and .82 at T1, T2, T3, and T4, respectively.

General self-worth was measured using the Global Self-Worth Subscale from a revised version of the Harter's Self-Perception Profile for Adolescents (Harter, 1988; Wichstrom, 1995). Five items assess how an adolescent views himself/herself, with the response options ranging from 1 ("corresponds very poorly") to 4 ("corresponds very well"). Higher mean scores reflect high self-worth, and the scale had an acceptable internal consistency on all occasions: .70, .75, .75, and .78 at T1, T2, T3, and T4, respectively.

Negative affectivity, including symptoms of depression and anxiety was measured with a 12-item short version of the Hopkins Symptom Checklist (Derogatis, Lipman, Rickels, Uhlenhuth, & Covi, 1974). Using a response scale ranging from 1 to 4, participants were asked to restrict their ratings to the preceding week. Mean scores were calculated, with high scores indicating high levels of negative affectivity, and the scale revealed a satisfactory internal consistency on all occasions, with α values of .87, .88, .89, and .89 at T1, T2, T3, and T4, respectively.

Loneliness was measured by a five-item version of the UCLA Loneliness Scale (Russell, Peplau, & Cutrona, 1980), with each item having response options ranging from 1 ("never") to 4 ("often"). A higher mean score reflects greater loneliness, and the five-item scale exhibited a somewhat low internal consistency at T1 (α = .65), whereas the α values were acceptable at the remaining three time points, with α = .72, .76, and .78 at T2, T3, and T4, respectively.

Alcohol intoxication was measured by asking participants to indicate how often they had "drunk so much that you felt clearly intoxicated" during the preceding 12 months, and illicit drug use was also assessed by asking about the frequency of illicit drug use over the past 12 months. For both measures, the response scale ranged from 1 ("never") to 6 ("more than 50 times"). High mean scores indicate a high level of alcohol and illicit drug use.
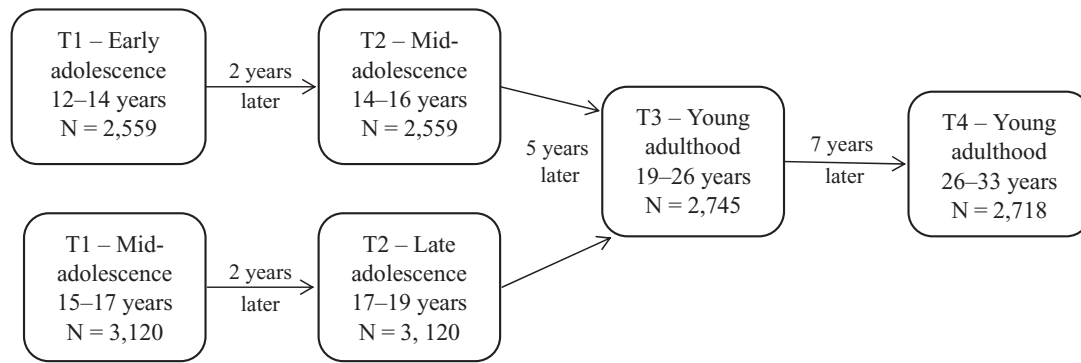
Figure 1. Selected study population at each time point.

Self-concept instability was measured using a revised version of Rosenberg's Stability of Self Scale (Rosenberg, 1986), which has four items, each with a response scale ranging from 1 to 4. High scores indicate an unstable perception of self (Alsaker & Olweus, 1986; Rosenberg, 1986), and the internal consistency was satisfactory at each survey point:.81, .86, .88, and .89 at T1, T2, T3, and T4, respectively.

Pubertal timing was measured by the Pubertal Development Scale (Petersen, Crockett, Richards, & Boxer, 1988), which assesses the development of five pubertal indices and uses a four-point scale ranging from "not begun" to "development completed." Three of the indices are gender neutral, and two are gender-specific; the indices for both boys and girls were pubic hair, growth spurt, and skin changes. In addition, boys rated their development of facial hair and voice change, and girls rated their breast development and whether they had reached menarche. Pubertal timing was only assessed at T1. A higher mean score indicated early pubertal timing, with the scales for both genders demonstrating a good internal consistency: .92 for girls and .90 for boys at T1.

Parental bonding styles were assessed by use of a short version of the Parental Bonding Instrument (Parker, Tupling, & Brown, 1979), which measures the emotional relationship between participants and parents by focusing on two dimensions, parental care and parental overprotection. Each dimension consists of five items and has a response scale ranging from 1 ("very like") to 4 ("very unlike"). High mean scores on the care subscale indicate a parent–child relationship based on emotional warmth, closeness, and empathy, whereas high scores on the overprotection subscale suggest parental obstruction of independent behavior, as well as parental control and parental intrusion (Parker et al., 1979). Adolescents reported on the parental bonding styles only at T1, and the internal consistency coefficients for parental care and overprotection scales were .70 and .71 at T1, respectively.

Body mass index (BMI, kg/m^2) was computed from self-reported measures of height and weight, as self-reported BMI has been demonstrated to be a valid measure of actual BMI (Goodman, Hinden, & Khandelwal, 2000). Age and gender were recorded in all surveys; male was coded as "0" and female as "1."

Statistical analysis

First, we computed descriptive statistics (means, standard deviations) of all variables included in the study. To obtain figures for different developmental phases, we divided the study population into two different cohorts at T1 and T2: a younger cohort, which was defined to be in early adolescence at T1 (age 12 to 14) and mid-adolescence at T2 (age 14 to 16), and an older cohort being in mid-adolescence at T1 (age 15 to 17) and in late adolescence at T2 (age 17 to 19) (see Figure 1). Second, we applied linear regression models to examine the effects of predictors on disordered eating for both the younger and older cohort and both genders separately. To examine longitudinal predictors of disordered eating in mid-adolescence, disordered eating scores at T2 for the younger cohort were regressed on predictors measured at T1. The same analyses were conducted in the older cohort to examine predictors of disordered eating in late adolescence. Third, since the young adulthood period was represented by two time points (T3 and T4), disordered eating scores from both time points were simultaneously included in a regression model as an outcome variable, whereas variables at T2 were included as predictors. A linear random intercept model was used for these regression analyses, taking the dependency of the disordered eating scores at T3 and T4 into account (Rabe-Hesketh & Skrondal, 2008). This model therefore provides regression estimates comparable to usual regression analyses, with an additional estimate of how time point (T3 versus T4) influences disordered eating.

In all regression and linear random intercept models, initial disordered eating scores were included as a covariate, thereby examining how predictors were related to *changes* in disordered eating. This analytical approach helps in establishing temporal precedence between the outcome (i.e., disordered eating) and the predictors. To test for differences in the regression coefficients between: (1) females and males; and (2) mid- and late adolescence, two-way interaction terms were computed and included together with the main effects in regression analyses. Similarly, to examine the possibility for regression coefficients to vary according to a combination of gender and developmental phase, three-way interactions were tested as well. As described by Dawson and Richter (2006), the delta method was used to obtain information on the direction of the three-way interaction terms when such effects were found. This method compares differences among regression coefficients that computed from the three-way interaction analyses – gender*developmental phase*predictor (Dawson & Richter, 2006). We used a similar modeling approach for both main effects and interaction analyses, first assessing the effect of single predictor and then conducting multiple regression among significant predictors ($p < .05$). Multicollinearity was examined using the Variance Inflation Factor (VIF), which in this study was less than three for all variables, indicating few problems with multicollinearity. We used Stata SE/11 for Windows for all analyses.

Results

Descriptive summary

Table 1 presents the means and standard deviations for all study variables in adolescence and young adulthood phases separately for females and males. Females had higher scores for disordered eating, BMI, negative affectivity, and self-concept instability, and lower scores for appearance satisfaction and self-worth than males across all developmental phases. Males had higher scores on illicit drug use and alcohol intoxication compared to females.

Predictors in mid- and late adolescence

We examined how potential risk factors predicted disordered eating prospectively at mid- and late adolescence by means of linear regression analyses for both girls and boys (see Table 2). Measurements at T1 were used as independent variables and disordered eating at T2 as an outcome. In a first step, we conducted one regression analysis for each predictor while controlling for T1 disordered eating and age (see the results in Table 2 denoted "β" models). We then performed multiple regression analyses in which all predictors with initial significant effects ($p < .05$) were simultaneously included (see results in Table 2 denoted "$\beta+$" models). Similarly, we first tested the interaction terms of each predictor in regression models ("β") while controlling for main effects, T1 disordered eating and age. Next, the interaction effects of those significant predictors were examined one by one in multiple regression models ("$\beta+$") (interaction results not shown in Table 2). Findings from both regression models ("β" and "$\beta+$") showed that initial disordered eating was significantly positively associated with disordered eating scores at T2 across developmental phases and genders. Furthermore, the two-way interaction terms revealed that the association between initial disordered eating and later disordered eating was stronger for girls than for boys ($\beta = .31$, $t = 6.59$, $p < .001$ and $\beta+ = .29$, $t = 6.11$, $p < .001$), and stronger in late adolescence than mid-adolescence ($\beta = .31$, $t = 6.64$, $p < .001$ and $\beta+ = .32$, $t = 5.77$, $p < .001$). The three-way interaction term was not significant ($p > .05$).

BMI was positively related to disordered eating in all multiple regression analyses. No significant interaction terms were detected for BMI, indicating that there were no significant differences in the regression coefficients between the four groups. Appearance satisfaction and self-worth were significantly related to lower disordered eating scores in all groups in the initial analyses. However, in multiple regressions, appearance satisfaction in mid-adolescent girls and boys, as well as self-worth in adolescent boys and late adolescent girls, failed to reach statistical significance. Additionally, no significant interaction terms were found for these two variables.

Negative affect predicted a higher disordered eating score among boys and girls, but for girls the significant association in late adolescence disappeared in multiple regression. The interaction analyses in the initial "β" models confirmed boys' regression coefficients for negative affect to be significantly larger than those for girls ($\beta = -.09$, t $= -2.17$, $p < .05$). However, this interaction effect did not retain statistical significance in multiple regressions. Loneliness significantly predicted higher disordered eating scores in mid- and late adolescent boys, but only the association in late adolescence remained significant in multiple regressions. The interaction analyses also showed that loneliness had a stronger significant association with disordered eating in boys than in girls ($\beta = .-11$, $t = -2.85$, $p < .01$ and $\beta+ = -.08$, $t = -2.41$, $p < .05$).

Alcohol intoxication had no significant associations in any group, while illicit drug use significantly predicted a higher disordered eating in mid-adolescent boys and a lower disordered eating in mid-adolescent girls, although these associations failed to reach statistical significance in multiple regression models. Also, a significant three-way interaction term ($\beta = .32$, $t = 3.23$, $p < .01$) and the post-hoc tests exhibited a stronger positive association between illicit drug use and disordered eating among mid-adolescent boys (M slope $= .11$, $z = 2.80$, $p < .01$) compared to all other groups. Again, this significant interaction was not observed in multiple regression analyses.

Self-concept instability significantly predicted higher disordered eating scores in adolescent boys and late adolescent girls but only remained significant in late adolescent girls in multiple regression models. Moreover, a significant three-way interaction term ($\beta = .18$, $t = 2.07$, $p < .05$) and the post-hoc tests showed self-concept instability to have a stronger significant positive association among late adolescent girls (M slope $= .06$, $z = 4.65$, $p < .05$) compared to mid-adolescent girls and late adolescent boys. This interaction term did not retain statistical significance in multiple regression analyses.

Early pubertal development was significantly related to disordered eating among boys in late adolescence but not to the three other groups. Since the pubertal development measurement was gender-specific, we did not assess its interaction effect.

Concerning parental bonding styles, perceived parental overprotectiveness was significantly related to higher disordered eating scores among mid-adolescent boys and adolescent girls but only remained statistically significant in adolescent girls in multiple regression analysis. Its interaction term with gender became significant in multiple regression ($\beta+ = .09$, $t = -2.42$, $p < .05$), indicating a stronger association between parental overprotectiveness and disordered eating in adolescent girls than in boys; no other significant interaction terms were found for parental overprotectiveness. Lastly, a significant relationship between parental care and a lower disordered eating was found among mid-adolescent boys in the "β" model and in three-way interaction analyses ($\beta = -.24$, $t = -2.12$, $p < .05$), although both associations did not retain statistical significance in multiple regression models.

Predictors during young adulthood

To estimate the effect of predictors beyond the adolescent phases, we developed random intercept models with disordered eating scores at T3 and T4 as outcome variables and measures at T2 as predictors. Table 3 shows the summarized results. Similar to the approach used in Table 2, we first conducted random intercept models for each predictor while controlling for T2 disordered eating and age (see results in Table 3 denoted "β") and then selected significant predictors ($p < 0.05$) for multiple random intercept models (see results in Table 3 denoted "$\beta+$"). The same approach was also applied to examine the interaction terms in both regression models ("β" and "$\beta+$" models). However, since both time periods (i.e., T3 and T4) represent young adulthood, we only performed two-way interaction analyses of gender (interaction results not shown in Table 3).

Table 1. Summary measures for variables during adolescence and young adulthood phases, males and females separately.

Variables	T1– Early adolescence Males N = 1,268 M (SD)	T1– Early adolescence Females N = 1,291 M (SD)	T1 – Mid-adolescence Males N = 1,448 M (SD)	T1 – Mid-adolescence Females N = 1,672 M (SD)	T2 – Mid-adolescence Males N = 1,268 M (SD)	T2 – Mid-adolescence Females N = 1,291 M (SD)	T2 – Late adolescence Males N = 1,448 M (SD)	T2 – Late adolescence Females N = 1,672 M (SD)	T3 – Early young adulthood Males N = 1,217 M (SD)	T3 – Early young adulthood Females N = 1,528 M (SD)	T4 – Middle young adulthood Males N = 1,201 M (SD)	T4 – Middle young adulthood Females N = 1,517 M (SD)
Disordered eating	1.52(.36)	1.68(.39)	1.46(.30)	1.75(.37)	1.44(.33)	1.73(.38)	1.41(.31)	1.76(.39)	1.44(.29)	1.71(.38)	1.47(.35)	1.63(.39)
Age	13.49(.55)	13.45(.58)	16.11(.75)	16.12(.76)	15.09(.67)	15.04(.71)	17.76(.72)	17.75(.71)	21.83(1.71)	21.81(1.79)	28.36(1.67)	28.34(1.73)
Body mass index	19.30(2.57)	19.07(2.47)	21.05(2.54)	20.56(2.34)	20.98(5.82)	20.31(5.58)	22.21(4.35)	21.34(4.44)	24.14(4.78)	22.67(4.28)	25.29(3.75)	23.62(4.15)
Appearance satisfaction	3.61(.60)	3.24(.65)	3.68(.55)	3.23(.61)	3.71(.66)	3.20(.66)	3.75(.56)	3.27(.61)	3.71(.63)	3.32(.62)	3.64(.64)	3.38(.59)
Self-worth	2.97(.55)	2.79(.57)	2.99(.54)	2.76(.53)	2.59(.35)	2.50(.33)	2.63(.32)	2.53(.33)	2.64(.13)	2.53(.30)	2.58(.30)	2.51(.28)
Negative affectivity	1.45(.41)	1.63(.48)	1.46(.37)	1.79(.52)	1.44(.41)	1.73(.52)	1.46(.41)	1.79(.52)	1.45(.43)	1.67(.52)	1.41(.43)	1.52(.48)
Loneliness	1.86(.53)	1.93(.58)	1.83(.53)	1.91(.54)	1.83(.57)	1.88(.58)	1.78(.54)	1.85(.54)	1.78(.53)	1.83(.48)	1.76(.51)	1.79(.49)
Alcohol intoxication	1.32(.82)	1.30(.78)	2.54(1.65)	2.53(1.56)	2.04(1.44)	2.05(1.43)	3.25(1.70)	3.05(1.56)	4.07(1.49)	3.53(1.48)	3.90(1.47)	3.12(1.51)
Illicit drug use	1.01(.21)	1.01(.15)	1.03(.34)	1.02(.30)	1.08(.43)	1.07(.41)	1.13(.49)	1.09(.38)	1.11(.56)	1.06(.41)	1.12(.56)	1.05(.37)
Self-concept instability	2.56(.63)	2.76(.59)	2.39(.65)	2.77(.61)	2.29(.71)	2.74(.63)	2.21(.70)	2.69(.68)	2.10(.73)	2.51(.73)	1.95(.70)	2.27(.72)
Pubertal development	2.21(.65)	2.78(.62)	0.76(1.33)	0.94(1.52)	–	–	–	–	–	–	–	–
Parental bonding styles:												
Overprotective	2.25(.52)	2.19(.54)	2.08(.54)	2.01(.58)	–	–	–	–	–	–	–	–
Care	3.12(.51)	3.19(.55)	3.10(.51)	3.15(.57)	–	–	–	–	–	–	–	–

Note. Pubertal development and parental bonding styles were measured at Time 1 only.

Table 2. Linear regression results for early predictors of disordered eating in middle and late adolescence, males and females separately.

	Males				Females			
	Mid-adolescence N = 1,268		Late adolescence N = 1,448		Mid-adolescence N = 1,291		Late adolescence N = 1,672	
Predictors	β (SE)	β+ (SE)	β (SE)	β+ (SE)	β (SE)	β+ (SE)	β (SE)	β+ (SE)
Initial disordered eating	0.42(.04)***	0.37(.03)***	0.54(.04)***	0.45(.02)***	0.51(.01)***	0.46(.03)***	0.62(.03)***	0.60(.02)***
Body mass index (BMI)	0.11(.003)***	0.09(.003)**	0.09(.003)***	0.10(.003)***	0.09(.004)***	0.09(.03)**	0.04(.002)**	0.04(0.002)**
Appearance satisfaction	−0.14(.02)***	−0.02(.02)	−0.11(.01)***	−0.06(.01)**	−0.07(.02)*	0.02(.02)	−0.06(.01)**	−0.05(.01)*
Self-worth	−0.15(.02)***	−0.02(.02)	−0.11(.02)***	−0.02(0.02)	−0.10(.02)***	−0.08(.02)**	−0.06(.02)**	−0.003(.02)
Negative affectivity	0.15(.03)***	0.09(.02)**	0.12(.02)***	0.07(.02)**	0.04(.02)	–	0.05(.02)*	0.02(.02)
Loneliness	0.12(.02)***	0.05(.02)	0.13(.02)***	0.09(.01)**	0.02(.02)	–	0.001(.01)	–
Alcohol intoxication	0.004(.01)	–	0.04(.004)	–	−0.01(.01)	–	0.03(.005)	–
Illicit drug use	0.07(.04)**	0.06(.04)	0.01(.02)	–	−0.05(.06)*	−0.05(.06)	0.02(.03)	–
Self-concept instability	0.11(.02)***	0.05(.01)	0.06(.01)*	0.01(.01)	0.03(.02)	–	0.04(.01)**	0.05(.01)*
Pubertal development	0.04(.02)	–	0.08(.01)*	0.09(.01)**	0.03(.02)	–	0.03(.01)	–
Parental bonding styles:								
Overprotective	0.09(.02)***	−0.05(.02)	0.01(.01)	–	0.07(.02)**	0.06(.02)*	0.04(.01)*	0.03(.01)*
Care	−0.11(.02)***	0.03(.26)	−0.03(.01)	–	−0.02(.02)	–	−0.03(.01)	–

Note. *$p < 0.05$, **$p < 0.01$, ***$p < 0.001$. β = standardized regression coefficients with standard errors in parentheses controlled for initial disordered eating and age. β+ = standardized regression coefficients with standard errors in parentheses from multiple linear regression models with control for other predictors, initial disordered eating, and age.

Table 3. Random intercept model results for predictors at T2 on disordered eating in young adulthood, males and females separately.

	Males N = 923		Females N = 1,241	
Predictors at T2	β (SE)	β+ (SE)	β (SE)	β+ (SE)
Initial disordered eating	0.36(.03)***	0.34(.03)***	0.38(.02)***	0.31(.02)***
Body mass index (BMI)	0.002(.002)	–	0.003(.002)	–
Appearance satisfaction	−0.03(.01)*	−0.01(.01)	−0.07(.01)***	−0.05(.01)***
Self-worth	−0.01(.02)	–	−0.05(.03)	–
Negative affectivity	0.11(.02)***	0.06(.02)**	0.06(.02)**	0.03(.02)
Loneliness	0.06(.01)***	0.02(.02)	0.04(.01)**	0.01(.01)
Alcohol intoxication	0.002(.005)	–	−0.004(.01)	–
Illicit drug use	−0.01(.02)	–	−0.02(.02)	–
Self-concept instability	0.05(.01)***	0.03(.01)**	0.05(.01)***	0.03(.01)**
Pubertal development	0.02(.01)	–	0.002(.01)	–
Parental bonding styles:				
Overprotective	0.002(.01)	–	0.01(.01)	–
Care	−0.04(.02)*	−0.01(.01)	0.001(.01)	–
Survey time points (T3 vs.T4)	0.03(.01)**	0.03(.01)**	−0.09(.01)***	−0.08(.01)***

Note. *$p < 0.05$, **$p < 0.01$, ***$p < 0.001$. β = regression coefficients with standard errors in parenthesis controlled for age and disordered eating at T2. β+ = regression coefficients with standard errors in parentheses from multiple random intercept models with control for other predictors, age, and disordered eating at T2. Pubertal development and parental bonding styles were measured at T1 only.

For both genders, disordered eating and self-concept instability at T2 were significantly positively associated with disordered eating in young adulthood. For these factors, no significant interaction terms with gender were found ($p > .05$). Appearance satisfaction significantly predicted less disordered eating in both genders, but only remained a significant predictor in young adult women in multiple regression. Although its interaction term showed a stronger association with a lower disordered eating in young adult women than young adult men ($\beta = -.04$, $z = -2.21$, $p < .05$), it was no longer statistically significant in a multiple regression analysis.

Negative affectivity significantly predicted higher disordered eating scores in both genders but only remained significant in young adult men in the multiple regression model. Its interaction effect also demonstrated a stronger association in young adult men than young adult women ($\beta = -.24$, $z = -2.12$, $p < .05$) but

disappeared in the multiple regression analysis. Loneliness in both genders and parental care in young adult men showed significant associations to disordered eating in the initial models, but such relationships were not found in multiple regression models. The regression coefficients for the changes of disordered eating scores between the survey time points (T3 and T4) were significantly positive in young adult men and negative in young adult women, thereby revealing an increase in the disordered eating score in young adult men over time, while decreasing over time in young adult women. The interaction term was significant ($\beta = -.12$, $z = -8.07$, $p < .001$ and $\beta+ = -.12$, $z = -7.74$, $p < .001$), indicating a larger decrease in the disordered eating score in young adult women than young adult men over time. No other significant predictors and interaction terms were found in the regression models ("β" and "$\beta+$" models).

Discussion

The main aim of this population-based longitudinal study was to examine whether predictors of disordered eating differed according to gender and developmental phases. The study documented both shared and specific risk factors across gender and developmental phases. More specifically, initial disordered eating and parental overprotectiveness predicted disordered eating more strongly in adolescent girls than in adolescent boys. In contrast, loneliness predicted disordered eating in adolescent boys more strongly than in adolescent girls. Furthermore, initial disordered eating was a stronger predictor in late rather than in mid-adolescence. No other associations between other risk factors and disorder eating differed significantly according to gender or developmental phase in multiple regression models.

The stronger association between initial disordered eating and later disordered eating in adolescent girls than boys could suggest a higher stability of disordered eating in females. Although a few prior longitudinal studies assessing eating problems included female and male participants, none of these studies tested for possible gender difference in the stability of eating problems (Kotler, Cohen, Davies, Pine, & Walsh, 2001; Leon, Fulkerson, Perry, Keel, & Klump, 1999; Neumark-Sztainer, Wall, Larson, Eisenberg, & Loth, 2011). Greater stability of disordered eating in adolescent girls may be due to social messages about the importance of thinness for girls and greater susceptibility to peer and media pressures on girls to be thin in line with gender socialization theory (Murnen & Smolak, 1997; Smolak & Levine, 1996). In addition, the rise in girls' body fat during puberty (Keel et al., 1997), and the role of estradiol for body fat composition and appetite (Klump et al., 2010), may explain the increased stability of disordered eating in adolescent girls.

Perceived parental overprotectiveness was also a stronger risk factor for adolescent girls than boys. This stronger association may be due to the closer attachment that they have with their parents than boys have, especially with their mothers (Gilligan, 1982; Shomaker & Furman, 2009). Adolescent girls often place more value on the quality of their family relationships (Tomori, Zalar, & Plesnicar, 2000). In addition, other studies found that daughters gave their parents higher scores for overprotectiveness than sons did (Murphy, Brewin, & Silka, 1997), with overprotectiveness being significantly associated with disordered eating behaviors in young adult females though not in young adult males (Tata et al., 2001). In general, higher parental protectiveness could result in family conflict and stressful experiences, and disordered eating behaviors could be a

response to these negative affective states and vice versa, particularly for the development of binge eating among females (Striegel-Moore et al., 2005). Hence, the exact mechanism of this association should be verified in future prospective studies.

Loneliness appeared as a male-specific risk factor during adolescence. This finding is in contrast to earlier studies reporting that females are more likely to report disordered eating symptoms in response to loneliness than males are (Levine, 2012). This could be related to the degree of sensitivity towards social relationships, as adolescent girls are found to be more sensitive to the status of their peer relationships and friendships than adolescent boys (Rose & Rudolph, 2006). However, there is generally a scarcity of research on the association between loneliness and eating problems, particularly among males. Further research is recommended to confirm our finding.

At the same time, even though negative affectivity was not a statistically significant predictor in multiple regression models, it consistently predicted disordered eating among adolescent boys and young adult men. These findings may suggest that disordered eating in males might be associated with an increased vulnerability to psychological co-morbidities as compared to females. Similar results have been found in one cross-sectional study that showed that binge eating and bulimic symptoms were associated with psychological problems to a larger degree in adolescent boys than in girls (McCabe & Vincent, 2003).

As for development-specific risk factors, only initial disordered eating exhibited a significantly stronger association in late than in mid-adolescence. This greater stability in late adolescence may be related to a heightened emotional and social distress. Since this phase is marked as the end of adolescence, with graduating high school and starting college, late adolescents may experience reduced social support, different standards of attractiveness, increased dating and sexual pressures, and intensified academic and peer pressures (Smolak & Levine, 1996). These changes and experiences are possibly stronger triggers for disordered eating behaviors in late adolescence than in earlier phases.

There were also risk factors that predicted disordered eating across gender and/or developmental phases in multiple regression analyses (significant main effects). Risk factors that predicted disordered eating in both genders include BMI in adolescence, appearance satisfaction in late adolescence, and self-concept instability in young adulthood. These findings suggest more similarities than differences among the risk factors of disordered eating between males and females in adolescence and young adulthood. The results are consistent with past research among adolescent boys and girls (Leon et al., 1999; Ricciardelli & McCabe, 2004). Our study further provides support for such similarity in young adulthood. Besides, significant main effects include a higher BMI in both mid- and late adolescent girls and boys, appearance satisfaction in late adolescent girls and boys and in young adult women, self-worth in mid-adolescent girls, negative affectivity in all developmental phases in males, and self-concept instability in mid- and late adolescent girls and in young adults of both genders. With regard to a higher BMI, both significant and non-significant results have been reported for adolescent and adult female samples (Stice, 2002) as well as adolescent male samples (Ricciardelli & McCabe, 2004). Regardless of age (Stice, 2002), body dissatisfaction has been mentioned as one of the most consistent and robust risk factors for eating pathology in females but not for adolescent males (Ricciardelli & McCabe, 2004). Prospective studies have reported mixed findings for negative affectivity; both significant (Leon et al., 1999) and non-

significant (Stice, Akutagawa, Gaggar, & Agras, 2000) relationships between negative affect and disordered eating were documented. Individuals with an unstable perception of themselves, particularly in conjunction with low self-worth (Kansi et al., 2003), could be more vulnerable to external information about how others perceive their appearance (Campbell, 1990). This may lead to negative emotions and the subsequent development of disordered eating. In general, some of the divergent findings could be due to the use of different assessment measures for disordered eating and predictors and differences in methodological approaches, follow-up periods, and characteristics of sample populations.

A main strength of the study is its longitudinal nature, which allows for a delineation of the temporal order between predictors and outcome by controlling for initial disordered eating symptoms. To the best of our knowledge, this is the first longitudinal study to explore the prospective effects of predictors of disordered eating at different developmental phases in a large population sample of adolescent and young adult males and females.

Nonetheless, the study also has its limitations. First, we used a general measure for disordered eating, though predictors could vary across different types of disordered eating such as dieting, bulimic symptoms, symptoms of anorexia nervosa, and binge eating. Although EAT-12 has three correlated subscales, these subscales exhibit a rather low reliability, particularly in relation to the Oral Control factor (Engelsen & Hagtvet, 1999). Moreover, since eating disorder not otherwise specified (EDNOS) is the most common type of eating disorder (Wilfley, Bishop, Wilson, & Agras, 2007), it further justifies the use of global disordered eating as an outcome in the study. Second, even though it has been argued that full-syndrome eating disorders may not qualitatively differ from sub-threshold presentations (Stice, 2002; Stice, Killen, Hayward, & Taylor, 1998), it remains to be seen whether the findings from this study will be supported in studies examining the predictors of full-threshold eating disorders. Third, although many predictors displayed some significant effects, their regression coefficients were below 0.2, except for the effects of initial disordered eating, thus suggesting that small effect sizes should be considered when interpreting the findings. Fourth, although we established a factor's status as a risk factor due to precedence (Jacobi et al., 2004), this study cannot determine whether these risk factors were causal. Fifth, the larger proportion of lost follow-up could be a source of bias, especially since the attrition was significantly associated with participants' characteristics and behaviors, such as older age, male gender, higher perceived parental overprotectiveness, and lower scores for parental care and loneliness. These factors may under- or overestimate the frequency and the prevalence of disordered eating and thus potentially bias regression estimates. Sixth, although age demarcations are often used for measuring developmental phases, these can be arbitrary and challenging given the variation in physical, social, and cognitive development. Finally, the larger time lag between measurements during adolescence and young adulthood could attenuate regression estimates of predictors on disordered eating.

Overall, the findings of this study may provide implications for selective prevention and intervention strategies tailored to the concerns of specific gender and developmental phases as well as universal strategies applicable across gender and adolescence and young adulthood. Since a higher BMI was found to be the most consistent predictor for disordered eating across gender and phases of adolescence, actual or perceived overweight could be a natural target for universal prevention programs. Other significant risk factors such as early disordered eating, loneliness, and negative affectivity, which were revealed as specific predictors, could also be targeted in the selective prevention programs and used in identifying or characterizing high-risk participants. Although previous prevention programs have had limited success (Pratt & Woolfenden, 2002), they would be improved by considering these gender- and development-specific risk factors. We also recommend more studies that integrate gender and developmental perspectives into research, which would be useful for elucidating the mechanisms of the developmental psychopathology of disordered eating.

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