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ORIGINAL PAPER

# The Influence of Heritability, Neuroticism, Maternal Warmth and Media Use on Disordered Eating Behaviors: A Prospective Analysis of Twins

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**Abstract** The relative impact of genetic and social influences on disordered eating behaviors (DEB) including binging, purging, excessive dieting and negative self-evaluations about weight remain an issue of debate. The current study sought to examine the relative influence of genetic and social influences on DEB. A 7-year prospective analysis of 580 monozygotic (MZ) and dizygotic (DZ) twins was conducted. Estimates of heritability of DEB were obtained using the DF Analysis Model. Regression equations revealed the relative predictive value of sibling's DEB, neurotic personality, maternal warmth and television and video game exposure on DEB. Heritability estimates for DEB were 0.40 for females and 0.48 for males. Among MZ and DZ twin pairs, female sex, neurotic personality and a genetic variable component, but not maternal warmth or school related problems, predicted DEB. Contrary to the expectations of media effects theory, greater media use was associated with lower DEB among DZ twins and had no influence on MZ twins. These results indicate that DEB is highly heritable and that personality variables may play an important role in the formation of DEB. This suggests that it is important to control for genetic variables when analyzing risk factors for DEB.

**Keywords** Body dissatisfaction · Eating disorders · Genetics · Personality · Mass media

Disordered eating behaviors (DEB) refer to a set of unhealthy behaviors including binge eating, purging, restrictive dieting, and the use of medications such as laxatives or diuretics to lose weight. These are often symptomatic of eating disorders [1] and may reflect concerns with weight and body image [2]. Academics and professionals continue to debate

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the origins of DEB. Most behavioral genetic studies of DEB, eating disorders, or body dissatisfaction find that there are strong genetic components with heritability estimates ranging between 40 and 80% [3–5]. Identifiable social risk factors for DEB tend to account for smaller portions of the explainable variance.

Research has focused on both personality and social risk factors for DEB. Among personality traits, neuroticism has been identified as a potential risk factor for DEB [6, 7]. Among the social risk factors, research has linked maternal influences, including negative feedback, with DEB [8, 9]. Extensive research has also focused on possible contributions of the media—which conveys a "thin ideal" [10]—on DEB. However, this research has provoked debate and controversy [11], partially because meta-analyses of media effects generally find small ($r = 0.08$ [12] to $r = 0.17$ [13]) effects of media consumption on body dissatisfaction. While it is possible that media consumption may increase DEB without increasing body dissatisfaction, this seems unlikely.

To date, relatively little research has attempted to examine hereditary and specific social risk factors for DEB in combination. Recently Ferguson et al. [11] proposed a Catalyst Model to conceptualize the interaction between genetics and the environment and their influence on DEB. Put briefly, the Catalyst Model proposes that genes and specific environmental conditions interact with personality traits, such as neurotic or borderline tendencies, and place individuals at risk for body dissatisfaction and, potentially, DEB. For example, body dissatisfaction and DEB are expected to be prevalent when peer competition is high. Importantly, the Catalyst Model predicts that when other environmental and genetic influences are accounted for, any effect of the media will be transient.

The current study utilizes prospective data to simultaneously investigate genetic, personality, and social risk factors for DEB. It remains possible that, when only social risk factors are considered in prospective analysis, spuriously high correlations due to underlying shared genetic variance may inflate the contribution of individual social risk factors. Controlling for genetics may help us understand which social risk factors are due to unique socialization influences, as opposed to underlying genetic components. The current analysis will consider heredity, as well as neurotic personality, maternal warmth, a history of school problems, and amount of media exposure as risk factors for DEB in a prospective sample of adolescents. To the authors' knowledge this is one of the first studies to do so.

## Methods

### Participants

The current study makes use of data from the National Longitudinal Study of Adolescent Health (Add Health) [14]. The Add Health dataset comprises a multi-wave longitudinal nationally representative sample of American adolescents. Specific details on the general sample and recruitment can be found in the original publication of the Add Health data [14]. Of interest to the current study were the hundreds of monozygotic (MZ) and dizygotic (DZ) twin pairs included in the sample, with a careful selection of only same-sex twin pairs to avoid conflating heritability with gender differences in the DZ twin pairs. A total of 290 MZ and DZ twin pairs ($n = 580$) were included in this study.

The current analysis makes use of data collected when adolescents were first recruited into the Add Health study in 1994–1995, in addition to the wave 3 follow-up, which occurred approximately 7 years later in 2001–2002, and the wave 4 data collected in 2008, approximately 13 years after initial recruitment. The average age of the teenagers was

approximately 16 at time 1 ($SD = 1.75$). 61.4% of the Add Health sample identified as Caucasian, 23.1% identified as African American, 3.6% as Native American, 7.6% as Asian American and 9.4% as other. When summated these percentages are greater than 100% because biracial participants were allowed to self-identify into more than one category. The current twin sample comprises 292 (50.3%) male and 288 (49.7%) female twins. As noted above, all twin pairs selected are same-sex.

## Measures

### *Disordered Eating Behaviors*

The DEB scale is comprised of 13 items related to concern with weight, as well as behaviors symptomatic of disordered or restrictive eating, including fasting, binging, purging, taking laxatives or diuretics, etc. Two items referred specifically to binging: "In the past 7 days, have you been afraid to start eating because you thought you wouldn't be able to stop or control your eating?" and "In the past 7 days, have you eaten so much in a short period that you would have been embarrassed if others had seen you do it?" Coefficient alpha for the total scale was 0.65. Separating out the binging from restrictive eating behaviors did not improve the reliability, and so were retained as a single outcome scale. This variable was assessed during Wave 3.

### *Maternal Warmth*

The maternal warmth scale consisted of five items related to the individual's perception that his or her mother was kind, loving and supportive. Sample items include: "Most of the time, your mother is warm and loving toward you" and "You are satisfied with the way your mother and you communicate with each other." Coefficient alpha for these items was 0.84. This scale was administered during Wave 1: no corresponding scale for fathers was included in the Add Health dataset.

### *School Problems*

The school problems scale consisted of 10 items related to difficulties getting along with teachers and other students, paying attention and feeling happy and safe at school. Sample items include: "During the 1994–1995 school year, how often did you have trouble getting along with your teachers?" and "During the 1994–1995 school year, how often did you have trouble paying attention in school?" Coefficient alpha was 0.76, and this variable was assessed during Wave 1.

### *Media Use*

During Wave 1 data collection, participants reported on the frequency of their television and computer game use. Scholars have argued that the media, including both television and video games, are saturated with thin-ideal images and have proposed that frequency of media use may be one cause of body dissatisfaction and subsequent disordered eating [10]. As such, this variable is included in the current analysis. Frequency of television use and computer game use were summed to generate an overall frequency of media use variable.

*Neuroticism*

The neuroticism scale includes 18 items related to the "Big 5" construct of neuroticism, which indicates an individual's proclivity toward stress, rumination and worry. Example items include: "I have frequent mood swings" and "I worry about things." Coefficient alpha for this scale was 0.86.

Data on neuroticism was only collected during the Wave 4 stage, whereas DEB data was collected during Wave 3. As such, the neuroticism data point is the only retrospective data point in the current analysis. This opens the possibility that the predictive value of neuroticism may be underestimated in the current analysis, although we suspect the risk of this is minimal, given that "Big 5" personality traits exhibit considerable stability over time, particularly during and after the adolescent years [15, 16].

Procedures

Specific procedures related to recruitment and data collection are available in the original Add Health report [14]. Current analyses were conducted with SPSS software. Heredity analyses were conducted using the DF Analysis Model, $K_1 = b_0 + b_1(K_2 - K_m) + b_2[R * (K_2 - K_m)] + e$. In this model the individual's score ($K_1$) is related to their sibling's score on the same variable ($K_2$), the mean for the scale ($K_m$) and the degree of genetic relatedness ($R$) and residual variance ($e$). Entered into a regression equation the ($K_2 - K_m$) refers to the shared environment term, [$R * (K_2 - K_m)$] to heredity and e refers to non-shared environment plus error variance [17]. Main predictive analyses were conducted using hierarchical multiple regressions. Variables were entered from most proximal (biological sex, genetics) to mid proximal (neurotic personality) to most distal (maternal, school and media influences) in accordance with the Catalyst Model [11]. Analyses were conducted separately for MZ and DZ twins. It was expected that the component for genetic variance [$R * (K_2 - K_m)$] would be higher among MZ than DZ twins, given their greater degree of genetic relatedness.

## Results

Heritability of DEB

Results from the DF Analysis Model were analyzed separately for males and females. For females, genetic factors ($h^2$) accounted for 40% of the variance in DEB, with 7% due to shared non-genetic factors ($c^2$). The remaining 53% of the variance was due to non-shared non-genetic factors ($e^2$). For males, results were similar with genetic factors accounting for 48% of the variance, shared non-genetic accounting for 5% of the variance and non-shared, non-genetic factors accounting for the remaining 47%. This data is presented in Fig. 1.

Predictors of DEB

As noted above, hierarchical multiple regression equations were used to predict DEB at Wave 3. MZ and DZ twins were analyzed separately with the expectation that the genetic component [$R * (K_2 - K_m)$] would be a far stronger predictor among MZ twins, relative to DZ twins. This is because this variable reflects the degree of genetic similarity within the twin pairs. In each case, biological sex and [$R * (K_2 - K_m)$] were entered on step 1,

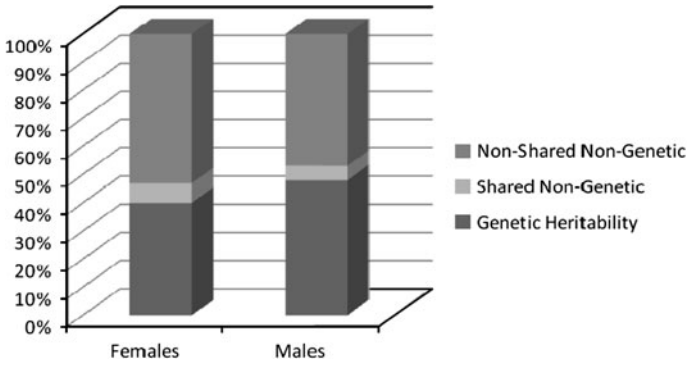


**Fig. 1** Proportions of variance in DEB due to heredity, shared and non-shared non-genetic factors

**Table 1** Standardized regression coefficients for MZ and DZ twins on DEB

| Predictor | MZ | DZ |
|---|---|---|
| Female sex | 0.28 (0.13, 0.42) | 0.25 (0.08, 0.41) |
| Genetic heritability variable | 0.43 (0.29, 0.55) | 0.24 (0.07, 0.40) |
| Neuroticism | 0.13 (−0.03, 0.28) | 0.20 (0.03, 0.36) |
| Maternal warmth | −0.02 | 0.15 |
| School problems | −0.03 | −0.01 |
| Media exposure | −0.02 | −0.27 (−0.10, −0.43) |

Confidence intervals presented are at the 95% for statistically significant variables

neuroticism was entered on step 2 and maternal warmth, school problems and media use data from Wave 1 were entered on step 3. To save space, results presented are for final significant models including all steps.

Among MZ twins, a statistically significant regression model [$R = 0.51$, adjusted $R^2 = 0.25$, $F(6, 148) = 8.88$, $p \leq 0.001$) indicated that the best predictor of DEB was the genetic variable ($\beta = 0.43$), followed by female sex ($\beta = 0.28$) and neuroticism ($\beta = 0.13$). Social variables, including maternal warmth, school problems and media use, were not significant predictors of DEB.

Among DZ twins, a statistically significant regression model [$R = 0.51$, adjusted $R^2 = 0.22$, $F(6, 128) = 7.13$, $p \leq 0.001$) indicated that the best predictor of DEB was female sex ($\beta = 0.25$), followed by the genetic variable ($\beta = 0.24$) and neuroticism ($\beta = 0.20$). Media use among this group was significantly related to *reduced* DEB ($\beta = -0.27$). Maternal warmth and school problems were not significant predictors of DEB (Table 1).

## Discussion

Current analyses were important for several reasons. First, the results provided further confirmation that DEB is highly heritable, with genetic factors explaining 40% of the variance in DEB for women and 48% of the variance for men. These results are largely in line with previous research [3–5]. However, the current results extend this previous work

by considering genetic variables in combination with neuroticism, maternal warmth, school problems and media use.

As expected a genetic heritance estimate variable was a better predictor for DEB among MZ than DZ twins, given this variable reflected greater genetic similarity among the MZ twins. Among MZ twins, DEB were also predicted by female sex and neuroticism. Among DZ twins, neuroticism, female sex, and media exposure were predictive of DEB. Media exposure was *inversely* related to DEB among DZ (but not MZ) twins. This finding is opposite that expected by advocates of media effects theory. However, given that meta-analyses of media effects have generally found low effect sizes [12, 13] it may be that considerable variance in outcomes exists for media effects. It is plausible that any link between media exposure and DEB, however small, is a result of underlying genetic variables. Our results provide evidence that an over-focus on media effects may be counterproductive.

Neuroticism was associated with increased rates of DEB. Although sparse, previous research on neuroticism and DEB is consistent with our results. These results are also consistent with the Catalyst Model that suggests personality plays an intermediary role between genetic predispositions and DEB related outcomes. As such more careful investigation of personality variables as predictors and possible genetic mediator variables for DEB would be worthwhile.

## Policy Implications

While it is popular to blame the media and cultural ideals for DEB, body dissatisfaction, and eating disorders, public policies should be based on an understanding of these factors in combination with personality, genetic, family, and peer factors. The overemphasis on media influence, for instance, has led to the conceptualization of eating disorders as socially constructed illnesses. This has hindered access to proper medical care such as that afforded to physical illnesses and serious mental health illnesses. However, research findings, including those of the current study, have identified a strong influence of genetics, much like that found for other serious psychological disorders (e.g., schizophrenia and depression). A more complete understanding of the role of genetics will encourage policy that places eating disorders in parity with physical disorders and other psychological disorders. In line with research on eating disorders that has uncovered evidence for genetic and biological influences, federal legislation has supported mental health parity, but more work is needed at the state level. Sufficient and equitable treatment for eating disorders is an important social desideratum [18].

Additionally, the fact that the current study simultaneously considers genetic and non-genetic influences on DEBs, and does so using a prospective analysis, contributes to the predictive value of these factors. In conjunction with previous research, these findings should encourage policies and programs that increase public awareness of the risk factors, signs and symptoms of DEB and eating disorders; that develop and implement effective prevention programs for youth; that train health care providers to properly assess and treat DEB and eating disorders; and that fund further research into DEB and eating disorders.

## Limitations

A chief limitation of this study is the availability of predictor variables. For instance, the Catalyst Model suggests that peer competition is likely to be one of the strongest social risk factors for DEB; however an appropriate measure of peer competition was not included in

the Add Health database. Future research ought to consider peer competition as a possible interacting environmental variable along with genetic influences. As noted earlier, neuroticism was measured during Wave 4, not during Wave 1 which would be desirable. Given the stability of personality traits across adolescence and adulthood, we suspect the negative influence of this retrospective measurement is minimal, but there is a possibility that the influence of neuroticism is underestimated in our model.

## Implications

With recent legislative efforts to address the need for further research into the treatment, education, and prevention of eating disorders (e.g., the Federal Response to Eating Disorders Act), studies such as this will inform research on the etiology of eating disorders.

The current analysis sought to examine the relative influence on DEB of genetic and social factors. To our knowledge ours is one of the first analyses of prospective data to consider both genetic and non-genetic factors simultaneously. We hope that our findings will provide helpful information to scholars and practitioners, and that they will stimulate further discourse on this important topic.

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