

A Comparison of Early Family Life Events Amongst Monozygotic Twin Women with Lifetime Anorexia Nervosa, Bulimia Nervosa, or Major Depression

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ABSTRACT

Objectives: To investigate the differential profile of early family life events associated with lifetime anorexia nervosa (AN), bulimia nervosa (BN), and major depression (MD).

Method: Only data from the monozygotic twins ($n = 622$) were examined from a community sample of female twins who had participated in three waves of data collection. Eating disorder and MD diagnoses were ascertained from the Eating Disorder Examination at Wave 3 and interview at Wave 2 respectively. Early family events were ascertained from self-report measures at Waves 1 and 3. Two case control designs were used, including a comparison of women: (1) who had lifetime AN, BN, MD, and controls, and (2) twin pairs discordant for either AN, BN, or MD (where the unaffected cotwin formed the control group).

Results: Across the two types of designs, compared to controls, both AN and BN were associated with more comments from the family about weight and shape when growing up. AN was uniquely associated with higher levels of paternal protection while BN was associated with higher levels of parental expectations.

Conclusion: While some overlap among early life events was indicated, especially related to parental conflict and criticism, there was evidence to support some degree of nonoverlap among life events associated with AN, BN, and MD. © 2007 by Wiley Periodicals, Inc.

Keywords: anorexia nervosa; bulimia nervosa; major depression; monozygotic; twin; family; life events

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Introduction

Case control designs that compare retrospectively recalled putative risk factors between people affected and unaffected by a psychiatric disorder are considered to offer valuable information about so called “retrospective correlates.”¹ These correlates represent variables that may be usefully examined in longitudinal designs. This case control design has been adopted previously in the area of eating disorders and family life events in three ways,² where there has been comparison of: (1) affected and matched, unaffected controls; (2) dis-

cordant siblings; and (3) discordant identical or monozygotic (MZ) twins.

Studies using matched unaffected controls show, compared to women with anorexia nervosa (AN), women with bulimia nervosa (BN) reported more parental criticism and critical comments from family about weight, shape, or eating.³ Examination of matched but unrelated individuals’ reports on parental treatment compared to their perceptions of the treatment of their siblings suggested that women with BN were more likely to rate their fathers as showing less affection and more control toward them than their sibling,⁴ with greater maternal affection and lower maternal control associated with higher harm avoidance scores, and decreased paternal control associated with higher novelty seeking scores.⁵ The importance of paternal factors has been further supported in a prospective study of the development of eating disorders and problems with eating and weight that showed maladaptive paternal behavior (i.e., abusive interactions and low levels of affection) to be uniquely associated with increased risk after the effects of other childhood adversities, including maladaptive maternal

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behavior, were controlled.⁶ Women with AN have reported more critical comments about weight, shape, or eating than psychiatric controls, as well as less parental contact and higher levels of parental underinvolvement and expectations.³ Discordant sibling designs have also shown that women with AN reported higher levels of parental expectations when growing up compared to their healthy sisters.⁷

The most powerful case control design is where MZ twins discordant for the eating disorder are compared, as the reason for such differences is likely to be due to the environment rather than due to genes, thus providing valuable information about putative risk factors that are more easily manipulated than genetic risk factors. Previous studies showed that MZ twins with lifetime BN reported lower levels of maternal care when growing up compared to their unaffected cotwin,⁸ but no such differences were noted for MZ twins with lifetime AN compared to their unaffected cotwin.⁹ Bulik and colleagues¹⁰ compared MZ twins discordant for BN and found that affected twins reported higher levels of discord in their families when growing up, but also recalled their parents as being warmer toward them. To date, no discordant MZ design has used a psychiatric control group.

We are still not in a position to draw strong conclusions from these case control designs, but across the studies there is an indication that critical comments related to eating and low levels of expressed parental affection are potential risk factors for disordered eating, with an independent contribution of low levels of paternal affection. With the caveat that most case control studies have been conducted with BN populations, it appears that family discord and family criticism could be important risk factors for BN, while high parental expectations may be more important for AN. The current paper seeks to add to and clarify these previous findings by examining and comparing MZ twins with respect to retrospectively recalled family life events. Two case control designs were employed, both using MZ twins. First, an affected and matched, unaffected control design was utilized with all MZ twins treated as individuals, where four nonoverlapping groups were compared: women with lifetime AN, BN, major depression (MD), and unaffected controls. Second, MZ twin pairs who were discordant for either lifetime AN, BN, or MD or who were concordant for being unaffected were compared with respect to differences in life events. The findings between these two designs are examined in order to see if there are life events that are uniquely associated with each disorder.

Method

Participants

Data for this study comes from three waves collected from a cohort of 8536 twins (4268 pairs) twins born between 1964 and 1971, who were enrolled by their parents with the Australian Twin Registry.¹¹ As shown in Figure 1, the first wave of data was collected between 1989 and 1992, when twins were aged 18–25 years, using a self-report Health and Lifestyle Questionnaire (HLQ), primarily designed to measure drinking behavior. While education below university level and being a DZ rather than an MZ twin predicted reduced likelihood of participating in the self-report questionnaire, there was no association between BMI and questionnaire nonresponse.¹¹ Over 1996–2000, when the twins were aged 24–36 years, a diagnostic telephone interview was carried out in a second wave of data collection. Informed consent was obtained from participants prior to administering the interviews, as approved by the Queensland Institute of Medical Research institutional review board.

Between 2001 and 2003, 2320 female twins (1140 complete pairs) who had participated in either Wave 1 or

FIGURE 1. Sample size and waves of assessment of female–female MZ twins from the Australian Twin registry (ATR). *Note:* Wave 3 sample has no overlap with previously reported discordant MZ data from the ATR.^{8,9}

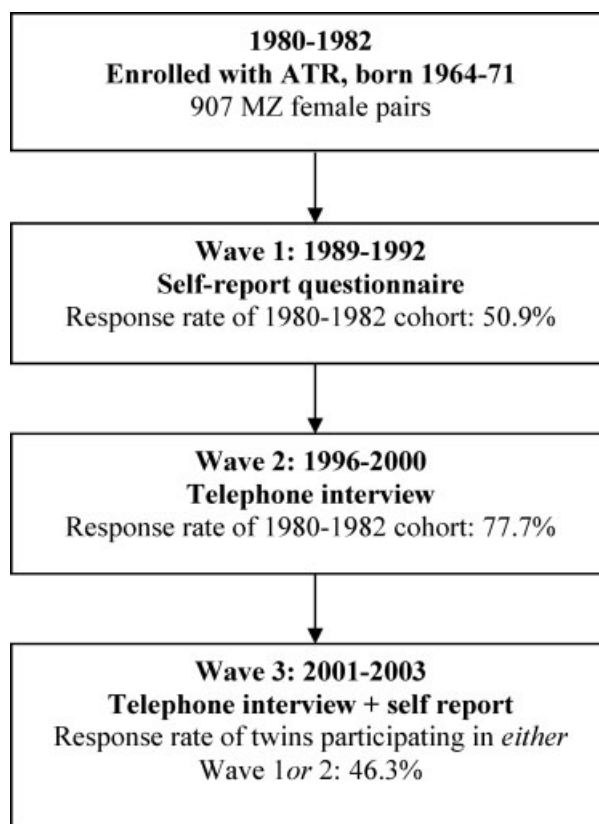


TABLE 1. Description of the self-report variables examined as retrospective correlates

Variable	Description and Cronbach's alpha
<i>Wave 1 – Life Events when growing up</i>	
Maternal care	Parental Bonding Inventory ^{14,15} : 3 care items and 4 protectiveness items each for mother and father (respective $\alpha = .69, .65, .69, .58$)
Paternal care	
Maternal over-protectiveness	Frost Multidimensional Perfectionism Scale ¹⁶ : respectively 5 items ($\alpha = .86$), and 4 items ($\alpha = .89$)
Paternal over-protectiveness	
<i>Wave 3 – Life events in first 16 years</i>	
Parental expectations	Revised Moos Family Environment Scale, conflict subscale ¹⁷ : 9 items ($\alpha = .73$)
Parental criticism	
Parental conflict	Risk Factor Interview ¹⁸ : 2 items, 4-point Likert scale: "Members of my family made comments about my weight or shape" and "Members of my family made comments about how much I ate".
Comments about weight	
Comments about amount eaten	

Wave 2 were approached to participate in Wave 3 that consisted of a self-report questionnaire and a telephone interview. Data (self-report, interview, or both) were obtained from 46.3% of those approached (mean age 35 years (SD = 2.11), ranging from 28 to 40 years), where the number of eating problems at Wave 1 did not predict participation at Wave 3.¹² Wave 3 included 1002 completed interviews and 1016 completed self-report questionnaires, where a total of 1056 women responded (962 completed both the interview and the questionnaire, 54 completed the questionnaire only, and 40 completed the interview only). The Flinders University Clinical Research Ethics Committee approved the study and written informed consent was obtained.

Only MZ women were included in the analyses of the current study, including 226 complete MZ pairs and 170 women from incomplete MZ pairs, where only one twin participated. Zygosity was determined on the basis of responses to standard questions about physical similarity and confusion of twins by parents, teachers, and strangers, methods that give better than 95% agreement with genotyping.¹³

Measures

Family Life Events. Self-report measures from Waves 1 and 3 relating to the family environment in the first 16 years of life were completed by the twins and are described in Table 1. The mean item score for each measure was calculated.

Depression Status. The diagnostic interview at Wave 2 included the Semi-Structured Assessment for the Genetics of Alcoholism¹⁹ and was adapted for telephone use with an Australian sample and updated for Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (DSM-IV) diagnostic criteria. The interview also included assessments of sociodemographic factors, childhood family environment, and experience of childhood sexual abuse.

Eating Disorder Status. A telephone interview was conducted at Wave 3 consisting of the Eating Disorder Examination (EDE), 14th edition,²⁰ revised with the insertion of lifetime questions, so that lifetime eating disorder diagnoses could be ascertained. All diagnostic questions addressed a three-month and a lifetime time frame. Thus the interview included questions relating to behavioral features of DSM-IV eating disorders as well as dietary restraint, eating concern, shape concern, and weight concern over the last 28 days. The prevalence of eating disorders in the whole group has been reported more fully elsewhere, as has the eating disorder assessment procedure and ascertainment of diagnostic groupings.^{12,21} A total of 19 women (1.9%) met full DSM-IV criteria for lifetime AN, 16 (1.6%) further met the criteria for AN with the exception of amenorrhea, and a further 8 women (0.8%) also met AN criteria but their amenorrhea status was less clear (e.g., some were taking the oral contraceptive pill at the time of low weight). A total of 35 women met full DSM-IV criteria for BN, where 6 had also had AN, leaving 29 women (2.9% of the total sample), 7 of these with a nonpurging BN. Most of the women did not have a current eating disorder, with only two women with lifetime AN continuing to have an eating disorder (one BN and one purging disorder), and two women with lifetime BN continuing to meet criteria for BN.

All interviewers were postgraduate Clinical Psychology trainees ($n = 10$) who had been trained in use of the EDE. Each of the interviews was taped and corrective feedback was provided until the interviewer had acquired the skills required to complete the interview independently. Monthly group meetings were held to discuss the interview process ensured interview fidelity. Two previous indicators of diagnostic reliability have been published previously, where both showed good reliability.²²

Statistical Analyses

The first case control design investigated whether family life events differed significantly across the three psychiatric diagnoses and control group. Subjects ($n = 622$) from MZ twin pairs were treated as individuals in the

TABLE 2. Comparison of family life events in the first 16 years for monozygotic twins

Variable	AN (<i>n</i> = 23)* [M (SE)]	BN (<i>n</i> = 20)** [M (SE)]	MD (<i>n</i> = 186) [M (SE)]	Control (<i>n</i> = 393) [M (SE)]	<i>F</i> (<i>p</i>) ES whole sample	<i>F</i> (<i>p</i>) ES eating disorder onset before age 16
Comments about weight or shape	2.71 (0.19) ¹	2.76 (0.20) ¹	2.37 (0.07) ^a	2.16 (0.05) ^{2b}	6.55 (<.001) 0.22	6.63 (<.001) 0.17
Comments on how much I ate	2.62 (0.17) ¹	2.66 (0.17) ^{1a}	2.27 (0.06) ^{1a}	1.94 (0.04) ^{2b}	15.38 (<.001) 0.34	15.44 (<.001) 0.25
Parental expectations	2.10 (0.11)	2.32 (0.12) ^a	2.19 (0.04) ^{1a}	2.04 (0.03) ^{2b}	4.24 (.006) 0.18	8.18 (<.001) 0.20
Parental criticism	1.87 (0.12) ^a	2.07 (0.12) ^{1b}	2.01 (0.05) ^{1b}	1.71 (0.03) ^{2a}	12.32 (<.001) 0.31	19.10 (<.001) 0.30
Parental conflict	2.15 (0.08)	2.29 (0.08)	2.21 (0.03) ^{1a}	2.08 (0.03) ^{2b}	6.20 (<.001) 0.26	6.07 (<.001) 0.20
Maternal care	3.43 (0.14)	3.26 (0.16)	3.46 (0.05)	3.57 (0.04)	2.351 (.07) 0.16	2.54 (.06) 0.11
Paternal care	3.14 (0.16)	3.19 (0.17)	3.18 (0.06)	3.36 (0.04)	2.77 (.04) 0.17	2.19 (.09) 0.10
Maternal protection	2.10 (0.14)	1.99 (0.15)	1.99 (0.05)	1.93 (0.04)	0.62 (.60) 0.08	0.29 (.83) 0.04
Paternal protection	2.28 (0.14) ¹	1.77 (0.14)	1.96 (0.05)	1.89 (0.04) ²	3.23 (.02) 0.19	1.52 (.21) 0.08

Notes: AN, anorexia nervosa; BN, bulimia nervosa; MD, major depression; M, mean; SE, standard error; ES, effect size.

*9 women (39.1%) also had lifetime MD.

**11 women (55%) also had lifetime MD.

Means in the same row with different numerical superscripts were significantly different in the whole sample (Bonferroni adjustment, $p < .05$) and means with different alphabetical superscripts were significantly different in the subgroup who reported developing their eating disorder before age 16 (Bonferroni adjustment, $p < .05$).

analyses, and divided into four groups: (1) twins with lifetime AN ($n = 23$), (2) twins with a lifetime BN diagnosis but no lifetime AN ($n = 20$), (3) twins with a lifetime MD diagnosis with no AN or BN ($n = 186$), and (4) twins with no lifetime AN, BN, or MD ($n = 393$). In order to correct for correlated-observations, linear mixed-effects modeling in SPSS (fixed-effects models with nonresidual errors) was used to compare the variables across the four groups, an analytic approach that not only adjusts for correlated observations, but is asymptotically efficient with unbalanced data. Between group effect sizes (ES) were calculated using Cohen's d , where $d = 2t/(\sqrt{df})$. ES of 0.2 are considered small, 0.5 are considered to be medium, and 0.8 are considered to be large. In order to inform firmer causal conclusions, the analyses were repeated for the subgroup of twins who reported developing their eating disorder after age 16 (the age up to which the environmental variables were assessed), which only included 13 women with lifetime AN and 16 women with lifetime BN.

The second case control design investigated differences in family life events using pairs discordant for one of the three psychiatric diagnoses, in addition to twin pairs concordant for not being affected by AN, BN, or MD. This included female twin pairs discordant for AN ($n = 14$), BN ($n = 14$), and MD ($n = 64$), and 100 concordant unaffected pairs (controls). Two types of analyses were used to examine the data. First, paired t -tests were used to investigate any differences between the family environment variables reported by each twin in the discordant pair, in order to identify variables that differed between pairs of twins growing up in the same family. Given the small numbers of discordant AN and BN pairs and the decreased ability to find significant differences compared to the larger MD sample, between group ESs were also calculated. Results were therefore considered to be worthy of note if they were (i) significant at $p < .05$, and/or

(ii) had a large ES. Second, linear mixed-effects modeling was used to compare reports from the second twin of each pair, i.e., the twin unaffected by any of the psychiatric diagnoses, thereby providing a more "objective" report of the family environment compared to the results from the first case control design, as reporting was not influenced by the experience of the disorder.

Results

Descriptives

Given that adolescence is one of the risk periods for developing an eating disorder, the age of developing the eating disorder was examined in order to better understand the relationship between the timing of the life events and the development of the eating disorder. Of the twins who formed part of the first case control design, the women with AN first developed this disorder at a mean age of 17.43 years ($SD = 3.26$), ranging from 14 to 24 years. Ten of the 23 women (43.5%) were under the age of 16 when they developed the disorder. The women with BN first developed the disorder at a mean age of 20.50 years ($SD = 5.74$), with an age range of 10–29 years. Four of the 20 women (20.0%) were less than 16 years when they first developed the disorder.

Of the twins who formed part of the second case control design, the women with AN developed this disorder at a mean age of 17.00 years ($SD = 3.11$), age range from 14 to 24 years, with 6 of the 14 (42.9%) aged less than 16 years. Women with BN developed this disorder at a mean age of 20.68 years ($SD = 6.07$), age range from 10 to 29 years, where 2 women (12.5%) developed the disorder before the age of 16 years.

First Case Control Design

The results of the comparison among singleton MZ twins is summarized in **Table 2**. Both the women with AN and BN recalled more comments about weight or shape when they were growing up than controls, and all three groups (AN, BN, and MD) recalled significantly more comments about the amount that they ate than controls. Women with MD reported significantly higher perceived parental expectations than do controls, while women with BN and MD reported significantly higher levels of parental criticism than do controls. Only the MD group reported significantly higher levels of parental conflict than controls, but it should be noted that the mean level of conflict reported by the women with BN was higher than the MD group, but because of the larger SE, the mean only approached significance ($p = .07$). Finally, no significant differences were reported with respect to the care or protectiveness variables, with the exception of the AN group who reported higher levels of perceived paternal protectiveness when growing up than the controls. While the analyses examining the subgroup who reported their eating disorder developing after age 16 had decreased discriminatory power, it was of interest to note that women with BN reported higher levels of parental expectations than controls, and women with AN reported significantly lower levels of parental criticism than women with either BN or MD.

Second Case Control Design

The results of the comparison of family functioning within MZ twin pairs discordant for the psychiatric disorder of interest are summarized in **Table 3**. Twins with AN reported significantly higher levels of paternal protectiveness when growing up compared to their cotwin controls, and large effect sizes were obtained for comments about weight and shape and comments about amount eaten, where the affected twin reported higher levels of these comments. Compared to their unaffected cotwin, women diagnosed with BN reported large effect sizes with respect to parental conflict and paternal care. Finally, women diagnosed with MD reported more parental criticism and comments about how much they ate than their unaffected cotwin.

Comparisons across unaffected twins in each of the four groups showed that cotwins of BN probands reported more comments about weight and higher levels of parental expectations than cotwins of MD probands or controls. Both cotwins of AN and BN probands reported more comments about the amount eaten and parental criticism than cot-

TABLE 3. Comparison of family functioning for monozygotic twin pairs discordant for AN, BN, MD, and concordant unaffected twin pairs (C)

Variable	Discordant AN ($n = 14$ pairs) [#]			Discordant BN ($n = 14$ pairs) ^{###}			Discordant MD ($n = 65$ pairs) ^{###}			Concordant unaffected twins ($n = 100$ pairs)			Between group comparisons
	A [M (SE)]	U [M (SE)]	ES [d (φ)]	A [M (SE)]	U [M (SE)]	ES [d (φ)]	A [M (SE)]	U [M (SE)]	ES [d (φ)]	A [M (SE)]	U [M (SE)]	ES [d (φ)]	
Comments: weight	2.93 (0.24)	2.54 (0.25)	0.92 (.14)	3.00 (0.24)	2.92 (0.25) ¹	0.17 (.78)	2.23 (0.11)	2.12 (0.12) ²	0.20 (.45)	2.08 (0.09)	2.03 (0.09) ²	0.11 (.61)	Unaffected twin BN > MD, C AN, BN > MD, C BN > MD > C AN, BN > MD > C AN, MD > C AN < C
Comments: amount	2.71 (0.21)	2.39 (0.20) ¹	0.85 (.17)	2.71 (0.21)	2.39 (0.20) ¹	0.66 (.27)	2.18 (0.10)	1.93 (0.09) ²	0.57 (.03)	1.84 (0.08)	1.80 (0.07) ²	0.05 (.80)	
Parental expectations	2.16 (0.15)	2.19 (0.15)	0.07 (.90)	2.52 (0.15)	2.54 (0.15) ¹	0.12 (.84)	2.10 (0.07)	2.12 (0.07) ²	0.05 (.86)	1.99 (0.06)	1.92 (0.05) ³	0.25 (.23)	
Parental criticism	1.98 (0.17)	2.04 (0.15) ¹	0.19 (.75)	2.20 (0.17)	2.12 (0.15) ¹	0.05 (.94)	1.91 (0.08)	1.74 (0.07) ²	0.68 (.01)	1.67 (0.06)	1.56 (0.06) ³	0.32 (.13)	
Parental conflict	2.20 (0.13)	2.26 (0.13) ¹	0.04 (.94)	2.28 (0.13)	2.07 (0.13)	1.06 (.11)	2.20 (0.06)	2.16 (0.06) ¹	0.37 (.17)	2.00 (0.05)	1.97 (0.05) ²	0.53 (.22)	
Maternal care	3.42 (0.17)	3.36 (0.14)	0.30 (.63)	3.28 (0.19)	3.30 (0.16)	0.04 (.96)	3.58 (0.08)	3.59 (0.07)	0.07 (.82)	3.64 (0.07)	3.64 (0.06)	0.06 (.80)	
Paternal care	3.08 (0.20)	3.05 (0.18) ¹	0.22 (.73)	3.42 (0.21)	3.37 (0.21)	0.86 (.26)	3.08 (0.10)	3.25 (0.09)	0.32 (.28)	3.44 (0.08)	3.47 (0.07)	0.13 (.57)	
Maternal protection	2.21 (0.17)	2.11 (0.16)	0.22 (.72)	2.00 (0.19)	2.08 (0.19)	0.16 (.81)	1.88 (0.08)	1.96 (0.08)	0.25 (.39)	1.81 (0.07)	1.82 (0.06)	0.02 (.92)	
Paternal protection	2.32 (0.18)	1.87 (0.15)	1.63 (.03)	1.66 (0.18)	1.75 (0.18)	0.37 (.61)	1.98 (0.08)	1.96 (0.08)	0 (1.00)	1.81 (0.07)	1.79 (0.06)	0.14 (.53)	

Notes: A, affected; U, unaffected; ES d, effect size (Cohen's d) for within twin pair comparisons.

Within twin comparison with a significant p value (<.05) or large ES are in bold.

Means of unaffected twins in the same row with different numerical superscripts were significantly different (an absence of a superscript indicates that the value does not differ significantly from any of the other groups) using least significant difference post-hoc comparisons (equivalent to no adjustment).

[#] Two pairs were removed as they were concordant for lifetime AN.

^{###} Pairs who had lifetime AN were removed.

^{###} Pairs who had lifetime AN or BN removed.

wins of MD probands or controls. Cotwins of both AN and MD probands reported more parental conflict than do controls, and cotwins of AN probands reported less paternal care than do controls.

Conclusion

The current study used two matched case control designs to investigate whether any early family life events were unique to three different psychopathologies, including AN, BN, and MD. The strength of the current study was the use of two case control designs that yielded results from three different perspectives. First, in common with studies using unrelated but matched unaffected controls, we compared the way in which the family environment was perceived by controls and by women affected by lifetime AN, BN, or MD. Second, we used the discordant MZ design, where twins are genetically identical and are raised in the same family, to examine differences in environmental experience. Interestingly, our results showed no family environment differences between twins in the unaffected (control) pairs, thus suggesting that differences do not routinely exist in the absence of psychopathology, thus providing confirmation that measurement error and random developmental processes are not likely to be competing explanations for the observed environmental differences.²³ Third, we compared families where twins were discordant for a lifetime psychiatric diagnosis with respect to the unaffected cotwin report, in order to make some conclusions about the “objective” environment, where reports were not influenced by the experience of having had an eating disorder.²⁴

The first finding of note was that it was only paternal, rather than maternal, care and control (overprotection) that was associated with eating disorders, in contrast to a finding from Wade and colleagues⁸ that women with lifetime BN reported lower maternal care than their affected MZ twin. There was no association of these variables with MD. While lower levels of paternal care were weakly linked with both BN and AN, there was a strong finding that increased paternal control was uniquely associated with AN. This accords with previous findings where a combined paternal care and protection measure was found to be higher for women with AN compared to matched controls,³ and that paternal factors were uniquely associated with increased risk of disordered eating above and beyond maternal factors.⁶ Interestingly this contrasts somewhat with research from other

areas that indicates *both* maternal and paternal variables are of importance in children's normative functioning and abnormal development,²⁵ indicating that fathers may play a unique role in the development of eating disordered behavior in their daughters compared to other psychopathologies. This suggestion should be interpreted cautiously in the context of low internal reliability of our PBI measure.

Second, comments about amounts eaten or appearance made by family members when the children were growing up was identified as a retrospective correlate for both AN and BN, consistent with a previous study.³ While this former variable was also a retrospective correlate for MD, comments about appearance were only of relevance to the eating disorder groups. Clinicians are familiar with the obsession with the “number on the scales” evidenced by their eating disorder clients, and this may somewhat reflect an early developmental focus on weight and appearance compared to other families. It is also not unknown for families to continue with unhelpful weight- and shape-related comments even after the eating disorder has been recognized. In therapy it may be useful to assess the degree to which such comments are currently made by the family in order to consider whether it might be useful to help the client develop appropriate coping skills to deal with such pressures.

Third, and contrary to two previous studies^{3,7} that used a single item to assess parental expectations¹⁸ which found that women with AN reported higher expectations from parents when growing up, it was the women with BN that reported higher parental expectations and not the women with AN. The women with lifetime MD also reported higher levels of parental expectations than do controls, but significantly lower expectations than the women with BN when examining nonaffected twins' reports of the environment. Clearly further clarification is required as to the impact of parental expectations in the development of eating disorders.

The fourth finding of note from the current study was that parental conflict and criticism were associated with all the psychopathologies, consistent with the conclusions of a recent review of risk factors for unipolar and bipolar depression showing support for a relationship between the development of psychopathology and both childhood emotional abuse from parents and negative parental inference about the causes and consequences of negative events in their child's life.²⁶ Such influences in early life can prospectively predict negative changes to children's attributional styles.²⁷ Hence

some degree of focus on general attributional style in eating disorder therapy may represent an important way in which the impact of early life events can be moderated. Of special interest here is that the unaffected AN twins reported problems with respect to parental conflict and criticism that were not reported by their affected cotwin, in contrast to twins with lifetime BN. This may reflect several issues, including a tendency for the twin affected by AN to understate discord in the family, or that while extra parental discord is created by the presence of AN, this is experienced only by the unaffected cotwin. If this latter suggestion is true, then family therapy approaches for AN may usefully pay some attention to the well-being of unaffected siblings among other sibling-related issues that are currently included in such therapy.²⁸

The results of this study should be interpreted in the context of five important limitations. First, the life events cannot be clearly interpreted as preceding the development of the eating disorder as they could also be a result of the eating disorder. However, our results are consistent with direction of causation modeling using ATR data that has shown support for a model specifying recollected parental behavior (as reported on the Parental Bonding Inventory) as the cause of psychological distress (including depression) rather than vice versa.²⁹ Second, data in this study are retrospective, thus influencing the accuracy of the data.²⁴ Third, we had a moderate response rate (46.3%), commensurate with other large population studies in Australia³⁰ but lower than others.³¹ There was no indication that a past history of disordered eating influenced response and neither did a previous study of Australian twins using interviews focused only on eating indicate that response was biased by previous eating problems.³² However, those with poor outcome with respect to the eating disorder may have been underrepresented in the current study. Fourth, while we used a highly reliable and valid eating disorder interview, the accuracy of the EDE for reporting retrospective eating disorder symptoms is unknown, though previous research has also shown that reliability of lifetime reporting is increased with the severity of the eating symptomatology.³³ Finally, the use of two single item measures relating to comments about food and weight increases the error variance related to these measures.

In summary, this study has contributed some suggestions for retrospective correlates that differ somewhat in emphasis from previous case control studies. It appears that comments about eating are not unique to any particular psychopathology but

that comments about appearance and low levels of care from fathers may be of etiological relevance to both AN and BN. Higher levels of control from fathers rather than high parental expectations may be a unique risk factor for AN, whereas parental expectations may be a unique retrospective correlate for BN. Family discord and criticism were associated with a variety of psychopathologies, with some support for the idea that criticism may be higher in families where there are eating disorders. A useful direction of future research suggested by the current study is investigation of whether the nature of the adverse life events that are associated with specific types of psychopathology gives some clue as to the specific vulnerabilities and core beliefs which can be tackled in therapy to provide maximum leverage in obtaining a decrease in eating disorder symptoms. For example, when working with people with AN it may be of relevance to tackle issues related to feeling controlled by powerful others, whereas when working with people with BN it may be useful to examine coping with the perceived expectations of others.

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