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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 whether there is a 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=592) were utilised in the current study from a community sample of 1,002 same-sex female twins who had participated in three waves of data collection. Diagnoses pertaining to eating disorders were ascertained from the Eating Disorder Examination at Wave 3, diagnoses of MD were ascertained through interview at Wave 2, and continuous measures of early family life events were ascertained from self-report measures at Waves 1 and 3. Two case control designs were used to examine life event risk factors, 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.

Results: Across the two types of case control analyses, compared to controls: AN was associated with more comments from the family about weight and shape when growing up and higher levels of paternal protection; BN was associated with higher levels of parental conflict; MD was associated with higher levels of parental criticism.

Conclusion: Whilst some overlap among early life events was indicated, there was evidence to support some degree of non-overlap among life events associated with AN, BN and MD.

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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 design has been adopted previously in the area of eating disorders and life events. 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, whereas women with AN report more critical comments than psychiatric controls, as well as less parental contact and higher levels of parental under-involvement and expectations.[2] Higher levels of parental expectations when growing up are also reported by women with AN compared to their healthy sisters.[3]

The most powerful case control design is where monozygotic (MZ) twins discordant for the eating disorder are compared, as the reason for such differences is likely to be due to environment rather than genes, thus providing valuable information about putative risk factors that are likely to be 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 co-twin,[4] but no such differences were noted for MZ twins with lifetime AN compared to their unaffected co-twin.[5] Bulik and colleagues[6] 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.

The current paper seeks to add to 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, twins were treated as singletons

and four groups were compared, women with AN, BN, MD, and controls. Second, MZ twins discordant for either lifetime AN, BN or MD were compared with respect to differences in life events. The similarities in findings across these two designs are examined in order to see if there are life events unique to these disorders.

Methods

Participants

Data for this study comes from three waves collected from a cohort of 8536 twins (4268 pairs) twins born 1964 to 1971. These twins were enrolled with the Australian Twin Registry (ATR) between 1980 and 1982.[7] The first wave of data was collected between 1989 and 1992, when twins were aged 18 to 25 years, a Health and Lifestyle Questionnaire (HLQ) was mailed to 4269 twin pairs. The HLQ self-report questionnaire was primarily designed to measure drinking behaviour, and covered a wide range of health issues affecting younger people including family life events and eating disorders. Completed questionnaires were returned from 50.9% of females.

Between 1996 to 2000, when the median age of the sample was 30 years (range, 24-36 years), 4010 twins were given a diagnostic telephone interview in a second wave of data collection. Completed interviews were obtained from 77.7% of females. While education below university level and being a DZ rather than an MZ twin predicted reduced likelihood of participating in the self-report questionnaire, associations between psychiatric history and health behaviour variables were modest, and there was no association between BMI and questionnaire non-response.[7] 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 Wave 2 were approached to participate in Wave 3 which included a self-report questionnaire and a telephone interview. Data (self-report, interview, or both) were obtained from 46% of the sample (mean age 35 years (SD=2.11), ranging from 28

to 40 years), where participation at Wave 3 was not predicted by the number of eating problems at Wave 1.[8] This included 1002 completed interviews, 1016 completed self-report questionnaires, where 962 women 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.[9]

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 Alcoholism[15] 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 socio-demographic 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,[16] 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 behavioural 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.[8,17] It should be noted that the 14 (60.9%) of the AN group included women who did not meet the amenorrhea criterion.

Statistical Analyses

The first case control design investigated whether family life events differed significantly across the psychiatric diagnoses and control group. The four groups included: (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=173), and (4) twins with no lifetime AN, BN, or MD (n=376). Subjects (n=592) from MZ twin pairs were treated as singletons in the analyses. Therefore, in order to correct for correlated-observations, linear mixed-effects modelling in SPSS (fixed-effects models with non-residual 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.

The second case control design investigated differences in family life events within each of the three psychiatric diagnoses. As shown in **Table 3**, we selected MZ twin pairs who were discordant for the diagnosis of interest, and this included female twin pairs discordant for AN (N=14), BN (N=14), and MD (N=64), where any twins in the BN and MD groups with co-morbid eating disorder diagnoses were removed. Paired t-tests were used to investigate any differences between the family environment variables reported by each twin in the discordant pair. Given the small numbers of discordant AN and BN pairs and the decreased ability to find significant differences compared to the larger MD sample, effect sizes (ES) were also calculated, namely 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. Results were therefore considered to be worthy of note if they were (i) significant at $p<0.05$, and/or (ii) had a large ES.

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 to 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 amongst singleton MZ twins is summarised 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 BN reported significantly higher perceived parental expectations than controls, and this group along with the women with MD reported significantly higher levels of parental criticism than 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=0.07). Finally, no significant difference 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.

Second case control design

The results of the comparison of family functioning between MZ twins discordant for the psychiatric disorder of interest are summarised in **Table 3**. Twins with AN reported significantly higher levels of paternal protectiveness when growing up compared to their co-twin 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 un-affected co-twin, 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 co-twin.

Discussion

The current study used two matched case control designs to investigate whether any early family life events were unique to three different psychopathologies, including anorexia nervosa (AN), bulimia nervosa (BN) and major depression (MD). The strength of the current study is the use of two case control designs, including a rigorous discordant MZ design, in order to detect consistent patterns of life events across the three psychopathologies, and to identify any unique retrospective correlates. The discordant MZ design, where twins are genetically identical and are raised in the same family, indicates that differences arise from differences in environmental experience (in the absence of measurement error and random developmental processes).[18]

Across the two case control designs, our results suggested that, compared to controls, women with lifetime AN reported more comments from the family about weight and shape and higher levels of paternal protection. Consistent with a previous study[2] we found the frequency of comments was higher than controls but no different between women with AN or BN. However, results from our discordant MZ twins design suggested that the comments are

unique to the AN group in terms of its association with the disorder. Paternal over-involvement and a combined paternal care and protection measure have been previously found to be higher for women with AN compared to matched controls,[2] and our results also suggest paternal overprotection to be unique retrospective correlate for the AN group.

Whilst both women with MD and BN reported higher levels of parental conflict than controls, albeit this latter finding only approached significance, it was only women with BN who reported higher levels of parental conflict in the discordant MZ design, consistent with the findings of Bulik and colleagues.[6] Conflict between parents has previously been implicated in the development of BN of a twin population from the Virginia Twin Registry, using a parental report of conflict rather than the affected twins' reports, thus removing any effect of the eating psychopathology on the retrospective report of conflict.[19]

Whilst both women with MD and BN reported higher levels of parental criticism, it was only indicated as a retrospective correlate for MD in the discordant MZ design. This is consistent with the results of a recent study, where verbal victimisation such as the comment "someone called me something like stupid, lazy or ugly" was found to prospectively predict negative changes to children's attributional styles.[20] A conflictual parent-child relationship was found to be one of the maximally discriminating variables associated with depression in MZ twins discordant for MD. [18] A recent review of risk factors for unipolar depression concluded that there is consistent support for a relationship between the development of depression and both childhood emotional abuse from parents and negative parental inference about the causes and consequences of negative events in their child's life. [21]

Comments from the family about the amount eaten when growing up appeared to be elevated in all three diagnostic groups, representing a non-specific retrospective correlate. Contrary to two previous studies[2,3] which used a single item to assess parental expectations,[14] we did not find that women with AN reported higher expectations from parents when growing up. Rather, the women with BN perceived this to be the case, but this did not differ across any of the discordant MZ twin pairs. Neither was the finding reported by Wade and

colleagues[4] of women with lifetime BN reporting lower maternal care than their affected MZ twin replicated in the current study.

The results of this study should be interpreted in the context of six important limitations. First, the life events can not be clearly interpreted as preceding the development of the eating disorder. At least half of the AN cohort and 1 in 5 of the BN cohort developed this disorder before age 16, the age up to which the family life events were assessed. Occurrence of the life event before the disorder would imply some causal relationship, but overlap with, or occurrence after the emergence of an eating disorder, would suggest it could be a sequelae of the illness. While direction of causation modelling with ATR data has shown support for a model specifying recollected parental behaviour (as reported on the Parental Bonding Inventory) as the cause of psychological distress (including depression) rather than *vice versa*, [22] this hypothesis would require testing with our eating measures, where there is limited power to conduct such analyses. Second, data in this study are retrospective, so the ages of eating disorder initiation might not be recalled accurately or the reporting of life event data may be influenced by the subsequent experience of a psychiatric disorder.[23] Third, we had a moderate response rate (47%), commensurate with other large population studies in Australia[24] but lower than others.[25] 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.[26] However, those with poor outcome with respect to the eating disorder may have been under-represented 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.[27] Fifth, the internal reliability of PBI is poor, and so results pertaining to this measure should be interpreted with caution. 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, results from this study may reflect actual differences or perceived differences. This latter suggestion is consistent with findings from previous twin studies where common genetic factors have been found to influence depression and ratings of parenting.[28] In other words, a gene-environment interaction may be active, where genes that influence development of psychopathology also affect sensitivity to adverse life events, or a gene-environment correlation, whereby genes that influence psychopathology also increase exposure to stressful life events.[29] If this is the case, and given that such genetic factors are shared between MZ twins, it would of interest to delineate the sources of non-shared environment outside of the family (e.g., sexual or physical assaults) that "switches on" such genetic vulnerability. Another area of future research is 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 conflictual situations.

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Table 1

Description of the self-report variables examined as retrospective correlates

Variable	Description and Cronbach's alpha
Wave 1 – Life Events when growing up	
Maternal care Paternal care Maternal over-protectiveness Paternal over-protectiveness	Parental Bonding Inventory[10,11]: 3 care items and 4 protectiveness items each for mother and father (respective α=.69, .65, .69, .58)
Wave 3 – Life events in first 16 years	
Parental expectations Parental criticism	Frost Multidimensional Perfectionism Scale[12]: respectively 5 items (α=.86), and 4 items (α=.89)
Parental conflict	Revised Moos Family Environment Scale, conflict subscale[13]: 9 items (α=.73)
Comments about weight Comments about amount eaten	Risk Factor Interview[14]: 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".

Table 2

Comparison of family life events in the first 16 years for monozygotic twins (n=592)

Variable	AN (n=23)[a]	BN (n=20)[b]	MD (n=173)	Control (n=376)	F (p)
	M (SE)	M (SE)	M (SE)	M (SE)	
Comments about weight or shape	2.71 (0.19)[1]	2.78 (0.20)[1]	2.32 (0.07)	2.15 (0.05)[2]	6.31 (<0.001)
Comments on how much I ate	2.61 (0.16)[1]	2.65 (0.17)[1]	2.25 (0.06)[1]	1.92 (0.05)[2]	15.20 (<0.001)
Parental expectations	2.09 (0.11)	2.36 (0.12)[1]	2.16 (0.04)	2.03 (0.03)[2]	4.26 (0.006)
Parental criticism	1.86 (0.12)	2.09 (0.13)[1]	1.98 (0.05)[1]	1.71 (0.04)[2]	9.81 (<0.001)
Parental conflict	2.15 (0.08)	2.29 (0.09)	2.20 (0.04)[1]	2.07 (0.03)[2]	5.52 (0.001)
Maternal care	3.43 (0.15)	3.25 (0.16)	3.45 (0.05)	3.57 (0.04)	2.41 (0.07)
Paternal care	3.13 (0.17)	3.19 (0.18)	3.18 (0.06)	3.36 (0.05)	2.61 (0.05)
Maternal protection	2.09 (0.14)	2.03 (0.15)	1.99 (0.05)	1.93 (0.04)	0.74 (0.53)
Paternal protection	2.28 (0.14)[1]	1.80 (0.14)	1.95 (0.05)	1.89 (0.04)[2]	3.07 (0.03)

Note: OR=odds ratio, CI=confidence interval, AN=anorexia nervosa, BN=bulimia nervosa, MD=major depression, M=mean, SE=standard error.

[a] 9 women (39.1%) also had lifetime MD; [b]11 women (55%) also had lifetime MD.

Numerical superscripts that differ represent significant differences of $p<.05$ between groups using Bonferroni adjustments.

Table 3

Comparison of family functioning in the first 16 years for monozygotic twins discordant for (i) anorexia nervosa (AN), (ii) bulimia nervosa (BN), and (iii) major depression (MD)

Variable	Discordant AN twins (n=14 pairs)[a]			Discordant BN twins (n=14 pairs)[b]			Discordant MD twins (n=64)[c]		
	Affected twin M (SD)	Unaffected twin M (SD)	ES *d*	Affected twin M (SD)	Unaffected twin M (SD)	ES *d*	Affected twin M (SD)	Unaffected twin M (SD)	ES *d*
Comments about weight & shape	2.92 (0.64)	2.54 (1.05)	0.92	3.00 (0.82)	2.92 (0.95)	0.17	2.21 (0.96)	2.11 (0.96)	0.20
Comments about how much I ate	2.69 (0.85)	2.39 (0.87)	1.71	2.62 (0.87)	2.39 (0.96)	0.66	2.17 (0.82)	1.91 (0.73)	0.54[1]
Parental expectations	2.17 (0.55)	2.18 (0.44)	0.07	2.49 (0.83)	2.54 (0.76)	0.12	2.10 (0.52)	2.01 (0.56)	0
Parental criticism	1.98 (0.83)	2.04 (0.63)	0.19	2.10 (0.88)	2.12 (0.81)	0.05	1.94 (0.60)	1.72 (0.54)	0.69[1]
Parental conflict	2.25 (0.60)	2.26 (0.42)	0.04	2.26 (0.66)	2.07 (0.64)	1.06	2.21 (0.51)	2.15 (0.50)	0.37
Maternal care	3.42 (0.85)	3.31 (0.52)	0.30	3.28 (1.13)	3.30 (0.91)	0.04	3.58 (0.53)	3.60 (0.51)	0.05
Paternal care	3.08 (1.00)	2.97 (0.75)	0.22	3.63 (0.35)	3.37 (0.56)	0.86	3.08 (0.75)	3.18 (0.75)	0.29
Maternal overprotection	2.21 (0.85)	2.17 (0.66)	0.22	2.00 (0.89)	2.08 (0.67)	0.16	1.85 (0.61)	1.93 (0.67)	0.29
Paternal overprotection	2.32 (0.87)	1.91 (0.61)	1.63 [1]	1.61 (0.55)	1.75 (0.47)	0.37	1.94 (0.60)	1.96 (0.62)	0.07

Abbreviation: ES, effect size Cohen's *d* (0.2=small, 0.5=medium, 0.8=large); [1] $p \leq 0.05$

[a]2 pairs were removed as they were concordant for lifetime AN; [b] pairs who had comorbid AN were removed; [c] pairs who had AN or BN removed