Childhood Sexual Abuse and Eating Disorders in Females

Findings From the Victorian Adolescent Health Cohort Study

Lena Sanci, MBBS, PhD, FRACGP; Carolyn Coffey, BSc, Grad Dip Epi; Craig Olsson, PhD; Sophie Reid, PhD; John B. Carlin, PhD; George Patton, MBBS, MD, FRANZCP

Objective: To examine the relationship between childhood sexual abuse (CSA) before the age of 16 years and later onset of bulimia and anorexia nervosa symptoms in females.

Design: A longitudinal cohort study of adolescents observed from August 1992 to March 2003. The cohort was defined in a 2-stage cluster sample using 44 Australian schools in Victoria.

Setting: Population based.

Participants: A total of 1936 persons participated at least once and survived to the age of 24 years, including 999 females. The mean (SD) age of females at the start of follow-up was 14.91 (0.39) years; and at completion, 24.03 (0.55) years.

Main Exposure: Self-reported CSA before the age of 16 years was ascertained retrospectively at the age of 24 years.

Outcome Measures: Incident Diagnostic and Statistical Manual of Mental Disorders (Fourth Edition)–defined partial syndromes of anorexia and bulimia nervosa were identified between waves 4 (mean age, 16.3 years) and 6 (mean age, 17.4 years) using the Branched Eating Disorder Test.

Results: The incidence of bulimic syndrome during adolescence was 2.5 (95% confidence interval, 0.80-8.0) times higher among those who reported 1 episode of CSA and 4.9 (95% confidence interval, 1.9-12.7) times higher among those who reported 2 or more episodes of CSA, compared with females reporting no episodes, adjusted for age and background factors. The association persisted after adjusting for possible confounders or mediators measured 6 months earlier, including psychiatric morbidity and dieting behavior. There was little evidence of an association between CSA and partial syndromes of incident anorexia nervosa.

Conclusion: Childhood sexual abuse seems to be a risk factor for the development of bulimic syndromes, not necessarily mediated by psychiatric morbidity or severe dieting.


Clinicians have long suspected that childhood sexual abuse (CSA) has a causal association with eating disorders, yet epidemiological and empirical studies have failed to provide consistent evidence for this association.

Early reviews reported discrepant findings. Some studies reported that CSA was no more prevalent in females with eating disorders than in other psychiatric groups or in the general population. Others found histories of abuse to be almost 2 times more common in females with anorexia or bulimia nervosa.

Methodological limitations probably contribute to inconsistent findings. Many studies used case-control designs with eating disorder cases taken from clinical settings but with control samples drawn from settings ranging from psychiatric clinics to primary care and population-based samples. Stronger associations were generally reported when the control group was nonclinical as opposed to findings of no difference when the control group consisted of clinical patients, particularly those with other psychopathological features.

Reports in later reviews vary considerably, not least because studies are cross-sectional and unable to measure a temporal association between CSA and eating disorder and studies adopt differing definitions of eating disorders and CSA. Conclusions from later reviews are that CSA is a nonspecific retrospective correlate of anorexia and bulimia nervosa, is a risk factor for bulimia nervosa with significant comorbidity, and cannot be a confirmed risk.
factor for eating disorder based on current evidence; and that there is a small significant relationship between CSA and eating disorder, but the nature of this association is difficult to determine. All reviews call for further prospective study of CSA as a risk factor for incident eating disorder.

Only one previous study7 was truly longitudinal. Johnson et al15 found that CSA was a risk factor for eating disorder during early adulthood in a community sample of 782 mothers and their offspring. Childhood abuse was ascertained by reports to a child protection registry and by maternal interview. Offspring were interviewed at the ages of 6, 14, 16, and 22 years, but there was temporal overlap between assessment of CSA and eating disorder in the adolescent age group, leaving the directional nature of the association between CSA and eating disorder unclear.

Ideally, a study of the relationship of CSA and eating disorders would take place around the time of peak incidence for eating disorders: 14 to 16 years for anorexia nervosa, whereas bulimia nervosa usually starts in adolescence and continues to occur beyond this point.16,17 We report on the relationship between sexual abuse before the age of 16 years and onset of anorexia or bulimia symptoms in later adolescence using data from an Australian longitudinal study of 999 adolescent females observed from the age of 14 years to the age of 24 years.

METHODS

SAMPLE

Between August 1992 and March 2003, we conducted an 8-wave cohort study of adolescent and young adult health in Victoria. Study protocols were approved by the Ethics in Human Research Committee of Royal Children's Hospital, Parkville. The cohort was defined in a 2-stage cluster sample, in which we selected 2 classrooms of 20 to 30 children at random from each of 44 schools drawn from a stratified frame of government, Catholic, and independent schools (60 905 students). School retention to year 9 (14 years) in the year of sampling was 98%. One class from each school entered the cohort in the latter part of the ninth school year (at the age of 14 years [wave 1]) with the second class 6 months later, early in the tenth school year (wave 2). Participants were subsequently reviewed at 4 six-month intervals during adolescence (waves 3-6), with 2 follow-up waves in young adulthood when participants were aged 20 to 21 years (wave 7) and 24 to 25 years (wave 8) (Figure). Written informed consent was obtained from parents at study commencement, and verbal informed consent was obtained from participants before each wave. In the adolescent data collection phase (1-6), participants self-administered the questionnaire on laptop computers, with telephone follow-up of those absent from school. The young adult phase (waves 7-8) of data collection was undertaken using computer-assisted telephone interviews.

Insufficient males were identified with an eating disorder in waves 4 to 6 (n=6) to allow meaningful analysis. Therefore, this report is restricted to an examination of eating disorder in females. From a sample of 1044 female students, 1000 (95.8%) participated at least once during the first 6 (adolescent) waves. In wave 8, 824 females (82.4% of teenaged participants) were interviewed between April 2002 and June 2003. Reasons for noncompletion were refusal (n=127), loss of contact (n=48), and death (n=1). The mean (SD) age of females at the start of follow-up (wave 1) was 14.91 (0.39) years; and at wave 8, it was 24.03 (0.55) years. In this report, we examined data for females from waves 3 to 6 and wave 8 in a surviving cohort of 999 females.

MEASURES

For eating disorder, because of the low prevalence of the full syndromes of anorexia and bulimia nervosa, partial syndromes were identified as cases. The Branched Eating Disorders Test, designed to define Diagnostic and Statistical Manual of Mental Disorders (Fourth Edition) criteria for eating disorder in adolescence, was used to assess symptoms of eating disorder during the previous 3 months.

The criteria for syndrome of anorexia nervosa were defined as follows: (1) body mass index (calculated as weight in kilograms divided by height in meters squared) below the 5th percentile for sex and age, (2) fear of weight gain despite hav-
Aim: To examine the relationship between childhood sexual abuse and eating disorder syndromes.

Methods:
1. Study design: Cross-sectional study.
2. Participants: 999 female participants aged 18-24 years.
3. Measures:
   - CSA (Childhood Sexual Abuse) assessed retrospectively at wave 8.
   - Eating Disordered Behaviors assessed at waves 1-6.
   - Other measures: Demographics, mental health, and history of abuse.

Results:
- Of the 999 female cohort participants, 12.1% were classified with 1 report of CSA and 8.2% were classified with 2 or more reports of CSA. Parental divorce and low parental education were more common in females reporting CSA (Table 1).
- Ninety-five female participants reported 1 episode and 69 reported 2 or more episodes of CSA without physical contact. Ninety-six females reported 1 episode and 70 reported 2 or more episodes of CSA without physical contact.

Analysis:
- Cross-sectional associations between background factors and reported incidence of CSA, and between CSA and cumulative incidence of eating disorder syndromes, were assessed using odds ratios with 95% confidence intervals (CIs). Multivariable discrete-time proportional hazards models were used to model associations between covariates of interest (including CSA) and the incidence of bulimic syndrome and its symptoms. This analysis allowed the inclusion of time-varying covariates (namely, behavior measured in the previous wave and age at wave). Because of the low incidence of the outcomes of interest, odds ratios and hazard ratios (HRs) can be interpreted as measures of relative risk.

Data collection was undertaken at a developmental point when young people are difficult to trace because of high mobility. There was low missingness on individual measures, but 30.7% of respondents missed at least one wave of data collection in the adolescent phase (waves 1-6), leading to potential bias in summary measures calculated from these data. To address this, we used the method of multiple imputation, with 5 complete data sets created by imputation using a multivariable normal model that incorporated all the variables of interest measured at all waves of data collection, along with the fixed covariates of sex, age, rural or urban residence, and parental education. Imputation was performed using a stand-alone software package (NORM) with adaptive rounding postimputation for binary measures.

Data analysis was undertaken with statistical software (Stata), with the multiple imputation analysis performed using software developed by one of us (J.B.C.) and colleagues. Estimates of all variables of interest were obtained by averaging across the 5 imputed data sets with Wald-type CIs calculated under multiple imputation using Rubin combination rules.

Results:
- Of the 999 female cohort participants, 12.1% were classified with 1 report of CSA and 8.2% were classified with 2 or more reports of CSA. Parental divorce and low parental education were more common in females reporting CSA (Table 1).
- Ninety-five female participants reported 1 episode and 69 reported 2 or more episodes of CSA without physical contact. Ninety-six females reported 1 episode and 70 reported 2 or more episodes of CSA with physical contact (Table 2). There was considerable overlap: 127 females (12.7%) reported contact and noncontact CSA at some level. In waves 4 through 6, 35 females (3.5%) were identified as new (incident) cases of bulimic syndrome and 32 (3.2%) as new (incident) cases of anorexia nervosa. Four individuals were identified as incident cases of both anorexia and bulimia syndrome in the same period. At wave 3, 19 and 29 females were identified with anorexic and bulimic syndromes, respectively, but were not eligible to be classified as incident cases and were excluded from further analysis.

Identification of incident cases of eating disorder occurred when most participants were 16 years or older (ie, having attended school in a non-Melbourne metropolitan location, having been born in a country other than Australia, parental divorce or separation by wave 6, or parental education of incomplete secondary schooling).
beyond the reference period for the CSA measure). However, the mean (SD) age for females at commencement of identification in wave 4 was 16.4 (0.4) years, with 189 females short of their 16th birthday, so that there was a slight overlap between identification of incident eating disorder and the reference period for CSA in the first wave of assessment.

There was little evidence of an association between any measure of CSA and new adolescent anorexic syndrome between waves 4 and 6 (Table 3), but the estimates were imprecise because of the low prevalence of exposures and outcome. We, therefore, did not examine this association further. Compared with female participants who reported no episodes of sexual abuse before the age of 16 years, those who reported 2 or more episodes had more than 5-fold elevated odds of new-onset bulimic syndrome (Table 2). Because the measures of noncontact and contact abuse were not mutually exclusive, and both showed similar patterns of risk to the overall measure of CSA, we examined only the effect of the summary measure of CSA on bulimic syndrome in subsequent analyses.

Table 1. Background Factors by Reported CSA Before the Age of 16 Years in a Cohort of 999 Females

<table>
<thead>
<tr>
<th>Background Factor</th>
<th>Total No. of Females</th>
<th>Those With No CSA (n=796)</th>
<th>Those With 1 Report of CSA (n=121)</th>
<th>Those With ≥2 Reports of CSA (n=82)</th>
</tr>
</thead>
<tbody>
<tr>
<td>School location</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Metropolitan</td>
<td>740</td>
<td>586 (73.6)</td>
<td>87 (71.9)</td>
<td>67 (81.7)</td>
</tr>
<tr>
<td>Nonmetropolitan</td>
<td>259</td>
<td>210 (26.4)</td>
<td>34 (28.1)</td>
<td>15 (18.3)</td>
</tr>
<tr>
<td>OR (95% CI)</td>
<td>NA</td>
<td>1 [Reference]</td>
<td>0.92 (0.56-1.5)</td>
<td>1.7 (0.91-3.0)</td>
</tr>
<tr>
<td>Place of birth</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Australia</td>
<td>866</td>
<td>690 (86.7)</td>
<td>106 (87.6)</td>
<td>70 (85.4)</td>
</tr>
<tr>
<td>Outside Australia</td>
<td>133</td>
<td>106 (13.3)</td>
<td>15 (12.4)</td>
<td>12 (14.6)</td>
</tr>
<tr>
<td>OR (95% CI)</td>
<td>NA</td>
<td>1 [Reference]</td>
<td>0.90 (0.44-1.8)</td>
<td>1.1 (0.53-2.3)</td>
</tr>
<tr>
<td>Parental divorce or separation by wave</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No</td>
<td>777</td>
<td>634 (79.6)</td>
<td>88 (72.7)</td>
<td>55 (67.1)</td>
</tr>
<tr>
<td>Yes</td>
<td>222</td>
<td>162 (20.4)</td>
<td>33 (27.3)</td>
<td>27 (32.9)</td>
</tr>
<tr>
<td>OR (95% CI)</td>
<td>NA</td>
<td>1 [Reference]</td>
<td>1.5 (0.92-2.4)</td>
<td>2.0 (1.2-3.3)</td>
</tr>
<tr>
<td>Parental high school completion</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>At least 1 parent completed</td>
<td>635</td>
<td>524 (65.8)</td>
<td>70 (57.9)</td>
<td>41 (50.0)</td>
</tr>
<tr>
<td>Neither parent completed</td>
<td>364</td>
<td>272 (34.2)</td>
<td>51 (42.1)</td>
<td>41 (50.0)</td>
</tr>
<tr>
<td>OR (95% CI)</td>
<td>NA</td>
<td>1 [Reference]</td>
<td>1.4 (0.91-2.2)</td>
<td>2.0 (1.2-3.1)</td>
</tr>
</tbody>
</table>

Abbreviations: CI, confidence interval; CSA, childhood sexual abuse; NA, data not applicable; OR, odds ratio.

a Frequencies were obtained by averaging across the imputed data sets.
b Data are given as number (percentage) of each group unless otherwise indicated.
c Risk category of the explanatory variable.
d Univariate ORs from multinomial logistic regression models (base category, “those with no CSA”).

Table 2. Cumulative Incidence of Partial Bulimic and Anorexic Syndromes at 16 Years or Older (Waves 4-6) in a Cohort of 999 Adolescent Females Reporting CSA Before the Age of 16 Years

<table>
<thead>
<tr>
<th>CSA Variable</th>
<th>Total No. of Females</th>
<th>Incident Bulimic Syndrome (n=35)</th>
<th>Incident Anorexic Syndrome (n=32)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>No. of Females</td>
<td>OR (95% CI)</td>
</tr>
<tr>
<td>CSA without physical contact</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>0 Reports</td>
<td>834</td>
<td>20</td>
<td>1 [Reference]</td>
</tr>
<tr>
<td>1 Report</td>
<td>95</td>
<td>7</td>
<td>2.5 (0.73-8.4)</td>
</tr>
<tr>
<td>≥2 Reports</td>
<td>69</td>
<td>8</td>
<td>4.6 (1.7-13.0)</td>
</tr>
<tr>
<td>CSA with physical contact</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>0 Reports</td>
<td>833</td>
<td>21</td>
<td>1 [Reference]</td>
</tr>
<tr>
<td>1 Report</td>
<td>96</td>
<td>6</td>
<td>2.9 (0.95-8.7)</td>
</tr>
<tr>
<td>≥2 Reports</td>
<td>70</td>
<td>8</td>
<td>5.3 (2.0-13.6)</td>
</tr>
<tr>
<td>Any CSA</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>0 Reports</td>
<td>796</td>
<td>18</td>
<td>1 [Reference]</td>
</tr>
<tr>
<td>1 Report</td>
<td>121</td>
<td>7</td>
<td>2.8 (0.85-8.9)</td>
</tr>
<tr>
<td>≥2 Reports</td>
<td>82</td>
<td>10</td>
<td>5.7 (2.2-14.6)</td>
</tr>
</tbody>
</table>

Abbreviations: See Table 1.
a Frequencies were obtained by averaging across the imputed data sets.
b Univariate ORs from logistic regression models based on an eligible sample of 970 females.
c Univariate ORs from logistic regression models based on an eligible sample of 980 females.
We examined the prospective association between CSA and new bulimic syndrome (Table 3) and the importance of time-varying putative mediators, including symptoms of anxiety and depression and dieting behavior, measured 6 months earlier.

After adjustment for background factors, females reporting 2 or more episodes of CSA were almost 5 times more likely to make the transition to bulimic syndrome than those reporting no abuse. This association was somewhat reduced by the addition of earlier anxiety and depression symptoms into the model, although CSA remained an independent predictor. The further addition of earlier dieting behavior into the model, although strongly predictive of the transition into disorder itself, did not substantially affect the association between incidence of bulimic syndrome and CSA.

This series of analyses was repeated using only complete (nonimputed) data from wave 8 (ie, restricting the imputed data sets to the 824 female participants who completed the final survey). There was no substantial difference in any estimate, but the standard errors increased, reflecting reduction in precision from loss of information. The estimates for 1 report of CSA were adjusted for background factors (HR, 2.6; 95% CI, 0.84–14.4), further adjusted for anxiety and depression symptoms in the previous wave (HR, 2.1; 95% CI, 0.71–6.2), and, finally, further adjusted for dieting behavior in the previous wave (HR, 2.3, 95% CI, 0.79–7.0). The estimates for 2 or more reports of sexual abuse were adjusted for background factors (HR, 4.8; 95% CI, 1.6–14), further adjusted for anxiety and depression symptoms in the previous wave (HR, 4.8; 95% CI, 1.6–14), further adjusted for anxiety (HR, 3.1; 95% CI, 1.7–5.3), and, finally, further adjusted for dieting behavior (HR, 3.6; 95% CI, 1.3–10.1), and further adjusted for dieting behavior in the previous wave (HR, 3.2; 95% CI, 1.2–8.4).

Females reporting 2 or more episodes of CSA were more than 4 times as likely to make the transition to purging than those with no CSA (Table 4). Further adjustment of this association for dieting behavior and symptoms of depression and anxiety in the previous wave reduced the estimate only marginally (HR, 2.7; 95% CI, 1.3–6.4). There was equivocal evidence of an increase in the likelihood of transition to overconcern about weight and to binge eating in females reporting 2 or more episodes of CSA.

Table 3. Predictive Associations Between CSA Before the Age of 16 y and Incidence of the Partial Bulimic Syndrome in Adolescence (Waves 4–6) in 970 Eligible Females

<table>
<thead>
<tr>
<th>Adjustment Variables</th>
<th>Prior Anxiety and Depression</th>
<th>Prior Dieting Behavior</th>
<th>Prevalent Measure</th>
<th>Explanatory Measure</th>
<th>Prior Anxiety and Depression</th>
</tr>
</thead>
<tbody>
<tr>
<td>0 reports</td>
<td>1 (Reference)</td>
<td>1 (Reference)</td>
<td>1 (Reference)</td>
<td>NA</td>
<td>4.8 (1.8–12.9)</td>
</tr>
<tr>
<td>1 report</td>
<td>2.5 (0.80–8.0)</td>
<td>2.0 (0.63–6.6)</td>
<td>2.0 (0.67–6.2)</td>
<td>NA</td>
<td>3.5 (1.3–9.4)</td>
</tr>
<tr>
<td>2 reports</td>
<td>4.9 (1.9–12.9)</td>
<td>3.5 (1.3–9.4)</td>
<td>3.1 (1.2–7.8)</td>
<td>NA</td>
<td>4.8 (1.8–12.9)</td>
</tr>
</tbody>
</table>

Table 4. Predictive Associations Between CSA Before the Age of 16 y and Incident Bulimic Syndrome Symptoms in Adolescence (Waves 4–6) in a Cohort of 999 Females

<table>
<thead>
<tr>
<th>No. of Reports of CSA</th>
<th>Incident Binge Eating (n=31)</th>
<th>Incident Purging (n=51)</th>
<th>Incident Overconcern About Weight (n=182)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>1 (Reference)</td>
<td>1 (Reference)</td>
<td>1 (Reference)</td>
</tr>
<tr>
<td>1</td>
<td>1.9 (0.64–5.8)</td>
<td>1.7 (0.36–8.0)</td>
<td>1.2 (0.63–2.3)</td>
</tr>
<tr>
<td>≥ 2</td>
<td>3.0 (0.39–23.6)</td>
<td>4.4 (1.9–10.3)</td>
<td>1.7 (0.95–3.0)</td>
</tr>
</tbody>
</table>

Abbreviations: CIS-R, Clinical Interview Schedule–Revised; CSA, childhood sexual abuse; NA, data not applicable.

Data are given as hazard ratio (95% confidence interval). Data are adjusted for age, parental divorce or separation, and parental education, using robust standard errors to allow for repeated measures within individuals.

COMMENT

This study provides evidence that CSA is a risk factor for bulimic disorders in young females. We found that reporting 2 or more episodes of CSA before the age of 16 years predicted a greater than 5-fold elevated cumulative risk of new bulimic syndrome during adolescence. In contrast, we found only equivocal evidence of an association between report of multiple episodes of CSA and the partial syndrome of anorexia nervosa. The association with bulimic syndrome was independent of background and family measures and persisted after adjusting for dietary behavior and symptoms of depression and anxiety, both common precursors of bulimic syndrome, measured 6 months before incident disorder. These factors reduced the association between CSA and incident bulimic syndrome marginally and, therefore, could be acting to some extent as mediators. In addition, we found that reporting 2 or more episodes of CSA was clearly predictive of incident purging behavior.

Our results augment evidence reported by Jacob et al, who found that in 4 of the 5 eligible studies, higher rates of CSA were present for patients with bulimia and anorexia nervosa, but that the evidence was much stronger for bulimia nervosa. The 6 studies reviewed by Wonderlich et al specifically to examine the hypothesis that...
CSA was more common in those with bulimia than in those with anorexia nervosa showed stronger associations for bulimia.

However, in a prospective study of the causes of drinking in adult females, Vogeltanz-Holm et al found no association between CSA and binge eating, dieting, and weight concern. A total of 709 females aged 16 to 45 years at baseline were reexamined 5 years later when they were asked about binge eating, intense dieting, and weight concerns. The mean age at follow-up was 34.7 years, so most incident cases of adolescent eating disorder would have been missed.

Suggested mechanisms for this association vary. Symptoms of eating disorder, such as binge eating, purging, or starving, may regulate continuing emotional distress following the experience of abuse. Alternatively, binge/purge cycles might function as an expression of anger or a symbolic "cleansing" of the self of the abusive experience, thereby allowing an individual to regain a stronger sense of self. Bulimia has also been viewed as a dissociative state in which awareness of CSA trauma is diminished.

Our study has addressed some of the methodological limitations of previous studies. It is a population-based cohort study of younger adolescents observed over a decade, allowing us to measure eating disorder and other time-varying factors prospectively. The use of proportional hazard models allowed us to examine the influence of time-dependent covariates. We examined possible mechanisms by adjusting for putative mediators between CSA and eating disorders measured before the transition to disorder. Finally, although the measure of CSA was retrospective, we examined the effect of different levels of abuse, as recommended by Smolak and Murnen.

There are, nevertheless, limitations to this study. We did not have available measures of temperament, personality, or self-denigration of the participants as children, potentially important mediating variables. Our measure of sexual abuse covered prepubertal and some postpubertal events, and we are not able to distinguish whether the timing of sexual abuse may have a differential effect on risks for later bulimic syndromes. In common with most, if not all, studies of CSA, we had to rely on the participants' retrospective report of events. This may be more likely to result in underreporting of CSA because survivors may find it too distressing to recall painful events and have, thus, repressed their experiences. We were unable to assess the possibility or the extent of recall bias or accuracy: whether those participants who experienced bulimia in adolescence were more inclined to remember CSA or whether participants were able to apply the reference period to events occurring some years earlier.

Perhaps most important, partial syndromes were identified as cases in this report, rather than full disorder. A recent review of studies of partial syndromes in adolescence reports that while most individuals go into spontaneous remission, a subset develops the full syndrome and compared with nonaffected individuals, those with partial syndromes are more at risk of developing a full eating disorder and in any case have risks for other psychopathological features. These are some examples from this review: one study of 16-year-old adolescents with partial syndrome found that after 12 months, 52% were in remission, 38% still had partial syndrome, and 7% had developed a full bulimia disorder; other studies found partial syndrome to be largely limited to adolescence, with between 1 in 3 and 1 in 10 persisting into adulthood; and another study showed that of the 1% of 17-year-old adolescents who had partial syndrome, 12.5% went on to meet criteria for full anorexia nervosa in early adulthood. Specific risk factors, including physical or sexual abuse, may also play a role in progression.

Our findings have clinical implications for the treatment, early intervention, and prevention of eating disorders in females with a CSA history. We concur with others who conclude there is a link between CSA and eating disorders on the need to consider the possibility of CSA when treatment of eating disorder is complicated and to specifically target the comorbidities of CSA before meaningful treatment of an eating disorder can begin.

Childhood sexual abuse has been linked to a range of conditions in which difficulties with emotional control are prominent. This seems to also be the case for eating disorders in that a clear association was found with bulimic, but not anorexic, symptoms in our study. We suggest that developing less impulsive strategies for dealing with difficult emotions may be an important facet of efforts to prevent eating disorders or reduce their impact in female adolescents with a history of sexual abuse.

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Correspondence: Lena Sanci, MBBS, PhD, FRACGP, Department of General Practice, University of Melbourne, 200 Berkeley St, Carlton, Victoria 3053, Australia (l.sanci@unimelb.edu.au).

Author Contributions: Ms Coffey and Dr Carlin had full access to all the data in the study and take responsibility for the integrity of the data and accuracy of the data analysis. Study concept and design: Sanci, Olsson, and Patton. Acquisition of data: Coffey, Olsson, and Patton. Analysis and interpretation of data: Sanci, Coffey, Olsson, Reid, Carlin, and Patton. Drafting of the manuscript: Sanci, Coffey, and Patton. Critical revision of the manuscript for important intellectual content: Sanci, Coffey, Olsson, Reid, Carlin, and Patton. Statistical analysis: Coffey and Carlin. Obtained funding: Olsson and Patton. Administrative, technical, and material support: Sanci, Coffey, Olsson, and Patton.

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Additional Information: The location of work was the Centre for Adolescent Health, Parkville, Victoria.

Additional Contributions: Philip Greenwood, PhD, assisted with the data processing, in particular preparation of imputed data sets.

REFERENCES


Heredity is what a man believes in until his son begins to behave like a delinquent.
—Presbyterian Life