

# 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.

*Arch Pediatr Adolesc Med.* 2008;162(3):261-267

### Author Affiliations:

Department of General Practice, University of Melbourne, Carlton (Dr Sanci); Centre for Adolescent Health (Ms Coffey and Drs Olsson, Reid, and Patton) and Clinical Epidemiology and Biostatistics Unit (Dr Carlin), Murdoch Childrens Research Institute, Parkville; and Department of Paediatrics, University of Melbourne, Melbourne (Dr Carlin), Victoria, Australia.

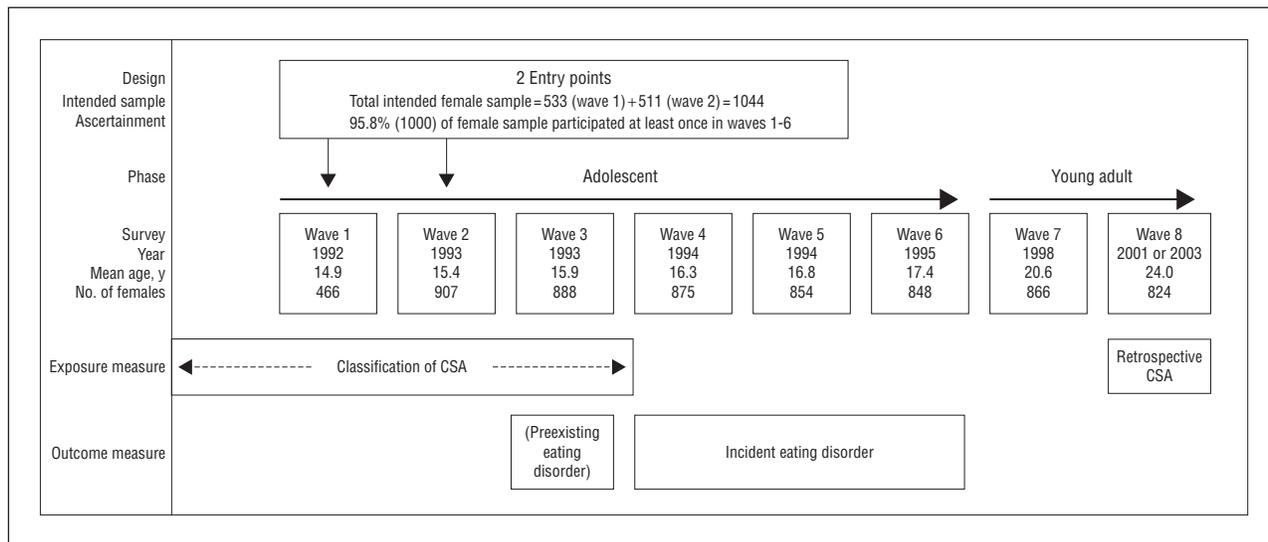
CLINICIANS HAVE LONG SUSPECTED that childhood sexual abuse (CSA) has a causal association with eating disorders,<sup>1-15</sup> yet epidemiological and empirical studies<sup>3,5</sup> have failed to provide consistent evidence for this association.

Early reviews<sup>2,3</sup> 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.<sup>1,5</sup> Many studies used case-control designs with eating disorder cases taken from clinical set-

tings but with control samples drawn from settings ranging from psychiatric clinics to primary care and population-based samples.<sup>1,5</sup> 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.<sup>5</sup>

Reports in later reviews vary considerably,<sup>1,5-7</sup> 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.<sup>5,7</sup> Conclusions from later reviews are that CSA is a nonspecific retrospective correlate of anorexia and bulimia nervosa,<sup>7</sup> is a risk factor for bulimia nervosa with significant comorbidity,<sup>1</sup> and cannot be a confirmed risk



**Figure.** Sampling and ascertainment of females in the Victorian Adolescent Health Cohort, August 1992 to March 2003, with exposure and outcome measures. CSA indicates childhood sexual abuse.

factor for eating disorder based on current evidence<sup>6</sup>; and that there is a small significant relationship between CSA and eating disorder, but the nature of this association is difficult to determine.<sup>5</sup> All reviews call for further prospective study of CSA as a risk factor for incident eating disorder.<sup>1,5-7</sup>

Only one previous study<sup>7</sup> was truly longitudinal. Johnson et al<sup>15</sup> found that CSA was a risk factor for eating disorder in 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.<sup>16,17</sup> 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<sup>18</sup> of 999 adolescent females observed from the age of 14 years to the age of 24 years.

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,<sup>19</sup> 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.

## 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

### 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-

ing a body mass index below the 25th percentile for sex and age, (3) overconcern with weight and body mass index below the 25th percentile for sex and age, and (4) secondary amenorrhea of at least 3 months' duration.

The criteria for syndrome of bulimia nervosa were defined as follows: (1) bingeing (frequent loss of control over eating at least weekly for at least 3 months), (2) purging (weight control by induced vomiting at least twice weekly and/or laxatives at least twice weekly and/or diuretics at least twice weekly and/or daily fasting and/or daily vigorous exercise, for at least 3 months), and (3) overconcern with weight (weight very important to feelings about self). A partial syndrome of eating disorder was defined when a subject met 2 *Diagnostic and Statistical Manual of Mental Disorders* (Fourth Edition) criteria for either bulimia or anorexia nervosa. Definitions of partial syndrome using the Branched Eating Disorders Test had high agreement with the Eating Disorders Examination in a community sample of schoolgirls in Australia (sensitivity, 1.0; specificity, 0.99; and positive predictive value, 0.7).<sup>20</sup> For simplicity of presentation, we refer to these partial syndromes as "bulimic syndrome" and "anorexic syndrome" throughout the article. The Branched Eating Disorders Test was administered from waves 3 through 6. New (incident) disorder was identified in each wave from waves 4 through 6 in females who had no previous disorder (ie, in wave 3).

Childhood sexual abuse was measured retrospectively at wave 8 (at the age of 24 years) using 6 items developed by Martin et al.<sup>21</sup> Participants were asked, "Before you were 16, did any adult or older person involve you in any unwanted incidents like: (i) inviting or requesting you to do something sexual; (ii) kissing or hugging you in a sexual way"; (iii) touching or fondling your private parts; (iv) showing their sex organs to you; (v) making them touch you in a sexual way; (vi) attempting or having sexual intercourse?" The response set was "never," "once," and "more than once." These items were reduced to 3 measures, all with categories of "no report," "one report," and "two or more reports": (1) "sexual abuse without physical contact," classified according to the individual's most severe response to questions i and iv; (2) "sexual abuse with physical contact," classified according to the individual's most severe response to questions ii, iii, v, and vi; and (3) "any sexual abuse," classified according to the individual's most severe response to all abuse questions with categories.

We measured CSA in adulthood because our state has a statutory requirement to report all abuse in children younger than 17 years to government services. To have informed parents and participants of this at the time carried a risk of selective refusal for those with abuse histories. Furthermore, participation in waves 1 through 6 required parental and school consent. Inclusion of CSA questions may have reduced our response rates. By the age of 24 years, we believed participants would be sufficiently comfortable to encounter these questions, yet not as remote from the experience as to limit recall.

Dieting behavior was assessed from waves 3 through 6 using the 9-item Adolescent Dieting Scale, covering common strategies for dieting restraint, each with a 4-point Likert response set (0, 1, 2, and 3).<sup>22</sup> Dieting was categorized on 3 levels: none or minimal (score, 0-2), moderate (score, 3-14), and severe (score, 15-27) dieting behavior.

Symptoms of depression and anxiety were assessed in each adolescent in waves 3 through 6 using the computerized revised Clinical Interview Schedule.<sup>23</sup> The total scores on the revised Clinical Interview Schedule were dichotomized, with scores greater than 11 delineating a mixed depression-anxiety state at a lower threshold than syndromes of major depression and anxiety disorder, but where clinical intervention would be appropriate.<sup>24,25</sup>

Background measures included incomplete secondary schooling (ie, having left school before the final possible year [year

12], 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).

## 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.<sup>26</sup> 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,<sup>27</sup> with 5 complete data sets created by imputation under 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.<sup>28</sup> Imputation was performed using a stand-alone software package (NORM<sup>29</sup>) with adaptive rounding postimputation for binary measures.<sup>30</sup>

Data analysis was undertaken with statistical software (Stata<sup>31</sup>), 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.<sup>28</sup>

## 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 anorexic syndrome. 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,

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

Background Factor	Total No. of Females <sup>a</sup>	Those With No CSA (n=796) <sup>a,b</sup>	Those With 1 Report of CSA (n=121) <sup>a,b</sup>	Those With ≥2 Reports of CSA (n=82) <sup>a,b</sup>
School location				
Metropolitan	740	586 (73.6)	87 (71.9)	67 (81.7)
Nonmetropolitan <sup>c</sup>	259	210 (26.4)	34 (28.1)	15 (18.3)
OR (95% CI) <sup>d</sup>	NA	1 [Reference]	0.92 (0.56-1.5)	1.7 (0.91-3.0)
Place of birth				
Australia	866	690 (86.7)	106 (87.6)	70 (85.4)
Outside Australia <sup>c</sup>	133	106 (13.3)	15 (12.4)	12 (14.6)
OR (95% CI) <sup>d</sup>	NA	1 [Reference]	0.90 (0.44-1.8)	1.1 (0.53-2.3)
Parental divorce or separation by wave 6				
No	777	634 (79.6)	88 (72.7)	55 (67.1)
Yes <sup>c</sup>	222	162 (20.4)	33 (27.3)	27 (32.9)
OR (95% CI) <sup>d</sup>	NA	1 [Reference]	1.5 (0.92-2.4)	2.0 (1.2-3.3)
Parental high school completion				
At least 1 parent completed	635	524 (65.8)	70 (57.9)	41 (50.0)
Neither parent completed <sup>c</sup>	364	272 (34.2)	51 (42.1)	41 (50.0)
OR (95% CI) <sup>d</sup>	NA	1 [Reference]	1.4 (0.91-2.2)	2.0 (1.2-3.1)

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

<sup>a</sup>Frequencies were obtained by averaging across the imputed data sets.

<sup>b</sup>Data are given as number (percentage) of each group unless otherwise indicated.

<sup>c</sup>Risk category of the explanatory variable.

<sup>d</sup>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**

CSA Variable	Total No. of Females <sup>a</sup>	Incident Bulimic Syndrome (n=35) <sup>a</sup>		Incident Anorexic Syndrome (n=32) <sup>a</sup>	
		No. of Females <sup>a</sup>	OR (95% CI) <sup>b</sup>	No. of Females <sup>a</sup>	OR (95% CI) <sup>c</sup>
CSA without physical contact					
0 Reports	834	20	1 [Reference]	25	1 [Reference]
1 Report	95	7	2.5 (0.73-8.4)	4	1.2 (0.19-7.7)
≥2 Reports	69	8	4.6 (1.7-13.0)	3	2.4 (0.80-7.2)
CSA with physical contact					
0 Reports	833	21	1 [Reference]	24	1 [Reference]
1 Report	96	6	2.9 (0.95-8.7)	4	1.1 (0.17-7.5)
≥2 Reports	70	8	5.3 (2.0-13.6)	5	1.6 (0.46-5.6)
Any CSA					
0 Reports	796	18	1 [Reference]	23	1 [Reference]
1 Report	121	7	2.8 (0.85-8.9)	4	1.0 (0.14-7.3)
≥2 Reports	82	10	5.7 (2.2-14.6)	5	2.0 (0.71-5.8)

Abbreviations: See Table 1.

<sup>a</sup>Frequencies were obtained by averaging across the imputed data sets.

<sup>b</sup>Univariate ORs from logistic regression models based on an eligible sample of 970 females.

<sup>c</sup>Univariate ORs from logistic regression models based on an eligible sample of 980 females.

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

Explanatory Measure	Adjustment Variables <sup>a</sup>		
	Background Factors Only	Prior Anxiety and Depression	Prior Anxiety and Depression and Dieting Behavior
<b>Before the Age of 16 y</b>			
Childhood sexual abuse			
0 Reports	1 [Reference]	1 [Reference]	1 [Reference]
1 Report	2.5 (0.80-8.0)	2.0 (0.63-6.6)	2.0 (0.67-6.2)
≥2 Reports	4.9 (1.9-12.9)	3.5 (1.3-9.4)	3.1 (1.2-7.8)
<b>Measured the Previous Wave</b>			
Symptoms of anxiety and depression (CIS-R score, > 11)	NA	4.8 (1.8-12.9)	3.5 (1.2-10.9)
Previous dieting behavior			
None or minimal	NA	NA	1 [Reference]
Moderate	NA	NA	3.5 (0.48-26.0)
Severe	NA	NA	14 (1.7-120.7)

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

<sup>a</sup>Data are given as hazard ratio (95% confidence interval). Data are adjusted for background factors and progressively for symptoms of anxiety and depression and dieting behavior in the previous wave. The hazard ratios are from multivariable discrete time proportional hazards models, with all models adjusted for age, parental divorce or separation, and parental education, using robust standard errors to allow for repeated measures within individuals.

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<sup>32</sup> 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 anxi-

**Table 4. Predictive Associations Between CSA Before the Age of 16 Years and Incident Bulimic Syndrome Symptoms in Adolescence (Waves 4-6) in a Cohort of 999 Females<sup>a</sup>**

No. of Reports of CSA	Incident Binge Eating (n=31) <sup>b,c</sup>	Incident Purging (n=51) <sup>b,d</sup>	Incident Overconcern About Weight (n=182) <sup>b,e</sup>
0	1 [Reference]	1 [Reference]	1 [Reference]
1	1.9 (0.64-5.8)	1.7 (0.36-8.0)	1.2 (0.63-2.3)
≥2	3.0 (0.39-23.6)	4.4 (1.9-10.3)	1.7 (0.95-3.0)

Abbreviation: CSA, childhood sexual abuse.

<sup>a</sup>Data are given as hazard ratio (95% confidence interval). Hazard ratios are from multivariable discrete time proportional hazards models, adjusted for age, parental divorce or separation, and education, using robust standard errors to allow for repeated measures within individuals.

<sup>b</sup>Frequencies were obtained by averaging across the imputed data sets.

<sup>c</sup>Analysis based on an eligible sample of 971 females.

<sup>d</sup>Analysis based on an eligible sample of 938 females.

<sup>e</sup>Analysis based on an eligible sample of 696 females.

ety and depression symptom in the previous wave (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.

## 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 dieting behavior and symptoms of depression and anxiety, both common precursors of bulimic syndrome,<sup>33</sup> 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 Jacobi et al,<sup>7</sup> 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<sup>1</sup> 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<sup>12</sup> 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.<sup>1</sup> 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.<sup>3</sup> Bulimia has also been viewed as a dissociative state in which awareness of CSA trauma is diminished.<sup>1,3,9</sup>

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.<sup>6</sup> We examined possible mechanisms by adjusting for putative mediators between CSA and eating disorders measured before the transition to disorder.<sup>14</sup> Finally, although the measure of CSA was retrospective, we examined the effect of different levels of abuse, as recommended by Smolak and Murnen.<sup>5</sup>

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.<sup>4,5,9</sup> 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.<sup>2,32</sup> 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.<sup>2</sup> 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<sup>34</sup> and in any case have risks for other psy-

chopathological features.<sup>35</sup> 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.<sup>34</sup> Specific risk factors, including physical or sexual abuse, may also play a role in progression.<sup>34</sup>

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.<sup>1</sup>

Childhood sexual abuse has been linked to a range of conditions in which difficulties with emotional control are prominent.<sup>36</sup> 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.

**Accepted for Publication:** September 6, 2007.

**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.

**Financial Disclosure:** None reported.

**Funding/Support:** This study was supported by the National Health and Medical Research Council; the Centre for Excellence in Eating Disorders (Ms Coffey); and a research grant from beyondblue, the National Depression Initiative (Dr Reid). Dr Olsson is the recipient of a Victorian Health Promotion Foundation Public Health Fellowship, and Dr Patton holds a professorial chair in Adolescent Health Research supported by the Victorian Health Promotion Foundation.

**Role of the Sponsor:** The funding bodies had no role in the design and conduct of the study; in the collection, analysis, and interpretation of the data; or in the preparation, review, or approval of the manuscript.

**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

1. Wonderlich SA, Brewerton TD, Jocic Z, Danskey BS, Abbott DW. Relationship of childhood sexual abuse and eating disorders. *J Am Acad Child Adolesc Psychiatry.* 1997;36(8):1107-1115.
2. Connors ME, Morse W. Sexual abuse and eating disorders: a review. *Int J Eat Disord.* 1993;13(1):1-11.
3. Everill JT, Waller G. Reported sexual abuse and eating psychopathology: a review of the evidence for a causal link. *Int J Eat Disord.* 1995;18(1):1-11.
4. Molinari E. Eating disorders and sexual abuse. *Eat Weight Disord.* 2001;6(2):68-80.
5. Smolak L, Murnen SK. A meta-analytic examination of the relationship between child sexual abuse and eating disorders. *Int J Eat Disord.* 2002;31(2):136-150.
6. Stice E. Risk and maintenance factors for eating pathology: a meta-analytic review. *Psychol Bull.* 2002;128(5):825-848.
7. Jacobi C, Hayward C, de Zwann M, Kraemer HC, Agras WS. Coming to terms with risk factors for eating disorders: application of risk terminology and suggestions for a general taxonomy. *Psychol Bull.* 2004;130(1):19-65.
8. deGroot JM, Kennedy S, Rodin G, McVey G. Correlates of sexual abuse in women with anorexia nervosa and bulimia nervosa. *Can J Psychiatry.* 1992;37(7):516-518.
9. Waller G, Hamilton K, Rose N, Sumra J, Baldwin G. Sexual abuse and body-image distortion in the eating disorders. *Br J Clin Psychol.* 1993;32(pt 3):350-353.
10. Rorty M, Yager J, Rossotto E. Childhood sexual, physical, and psychological abuse in bulimia nervosa. *Am J Psychiatry.* 1994;151(8):1122-1126.
11. Welch SL. Childhood sexual and physical abuse as risk factors for the development of bulimia nervosa: a community-based case control study. *Child Abuse Negl.* 1996;20(7):633-642.
12. Vogelanz-Holm ND, Wonderlich SA, Lewis BA, et al. Longitudinal predictors of binge eating, intense dieting, and weight concerns in a national sample of women. *Behav Ther.* 2000;31(2):221-235.
13. Romans SE, Gendall KA, Martin JL, Mullen PE. Child sexual abuse and later disordered eating: a New Zealand epidemiological study. *Int J Eat Disord.* 2001;29(4):380-392.
14. Rayworth BB, Wise LA, Harlow BL. Childhood abuse and risk of eating disorders in women. *Epidemiology.* 2004;15(3):271-278.
15. Johnson JG, Cohen P, Kasen S, Brook JS. Childhood adversities associated with risk for eating disorders or weight problems during adolescence or early adulthood. *Am J Psychiatry.* 2002;159(3):394-400.
16. Killen JD, Hayward C, Litt I, et al. Is puberty a risk factor for eating disorders? *Am J Dis Child.* 1992;146(3):323-325.
17. Striegel-Moore RH, Bulik CM. Risk factors for eating disorders. *Am Psychol.* 2007;62(3):181-198.
18. Patton GC, Coffey C, Sawyer SM. The outcome of adolescent eating disorders: findings from the Victorian Adolescent Health Cohort Study. *Eur Child Adolesc Psychiatry.* 2003;12(suppl 1):125-129.
19. Paperny DM, Aono JY, Lehman RM, Hammar SL, Risser J. Computer-assisted detection and intervention in adolescent high-risk health behaviors. *J Pediatr.* 1990;116(3):456-462.
20. Selzer R, Hamill C, Bowes G, Patton G. The Branched Eating Disorders Test: validity in a nonclinical population. *Int J Eat Disord.* 1996;20(1):57-64.
21. Martin J, Anderson J, Romans S, Mullen P, O'Shea M. Asking about child sexual abuse: methodological implications of a two stage survey. *Child Abuse Negl.* 1993;17(3):383-392.
22. Patton GC, Carlin JB, Shao Q, et al. Adolescent dieting: healthy weight control or borderline eating disorder? *J Child Psychol Psychiatry.* 1997;38(3):299-306.
23. Mann AH, Wakeling A, Wood K, Monck E, Dobbs R, Szmukler G. Screening for abnormal eating attitudes and psychiatric morbidity in an unselected population of 15-year-old schoolgirls. *Psychol Med.* 1983;13(3):573-580.
24. Harrington R, Fudge H, Rutter M, Pickles A, Hill J. Adult outcomes of childhood and adolescent depression, II: links with antisocial disorders. *J Am Acad Child Adolesc Psychiatry.* 1991;30(3):434-439.
25. Lewis G, Pelosi AJ, Araya R, Dunn G. Measuring psychiatric disorder in the community: a standardized assessment for use by lay interviewers. *Psychol Med.* 1992;22(2):465-486.
26. Carlin JB, Wolfe R, Coffey C, Patton GC. Analysis of binary outcomes in longitudinal studies using weighted estimating equations and discrete-time survival methods: prevalence and incidence of smoking in an adolescent cohort. *Stat Med.* 1999;18(19):2655-2679.
27. Rubin DB. *Multiple Imputation for Non-Response in Surveys.* New York, NY: John Wiley & Sons Inc; 1987.
28. Schafer JL. *Analysis of Incomplete Multivariate Data.* London, England: CRC Press; 1997:147-192.
29. Schafer JL. NORM (1999). <http://www.stat.psu.edu/~jls/misoftwa.html>. Accessed August 10, 2007.
30. Bernaards CA, Belin TR, Schafer JL. Robustness of a multivariate normal approximation for imputation of incomplete binary data. *Stat Med.* 2007;26(6):1368-1382.
31. Stata Corp. *Stata Statistical Software [Release 9].* College Station, TX: Stata Corp; 2006.
32. Fergusson DM, Lynskey MT, Horwood LJ. Childhood sexual abuse and psychiatric disorder in young adulthood, I: prevalence of sexual abuse and factors associated with sexual abuse. *J Am Acad Child Adolesc Psychiatry.* 1996;35(10):1355-1364.
33. Patton GC, Selzer R, Coffey C, Carlin J, Wolfe R. Onset of adolescent eating disorders: population based cohort study over 3 years. *BMJ.* 1999;318(7186):765-768.
34. Chamay-Weber C, Narring F, Michaud PA. Partial eating disorders among adolescents: a review. *J Adolesc Health.* 2005;37(5):416-427.
35. Lewinsohn PM, Striegel-Moore RH, Seeley JR. Epidemiology and natural course of eating disorders in young women from adolescence to young adulthood. *J Am Acad Child Adolesc Psychiatry.* 2000;39(10):1284-1292.
36. Kendall-Tackett KA, Meyer Williams L, Finkelhor D. Impact of sexual abuse on children: a review and synthesis of recent empirical studies. *Psychol Bull.* 1993;113(1):164-180.

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