Child Sexual Abuse and Later Disordered Eating: A New Zealand Epidemiological Study

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Abstract: **Objective:** *This community-based study examined how some women who have experienced childhood sexual abuse (CSA) develop an eating disorder (ED), whereas others develop depression and anxiety, and others show no adverse psychological sequelae.* **Methods:** *A two-stage random community sampling strategy was used to select two groups of women: (1) women with CSA prior to age 16 years and (2) a comparison group of women reporting no abuse. Both groups completed the Parental Bonding Instrument (PBI), the Present State Examination, and additional ICD–10 eating disorders questions. Information on the nature and frequency of the CSA was obtained at interview. CSA women with ED (CSA+ED) were compared with CSA women without ED (CSA–noED) and with CSA women with anxiety and/or depression (psychiatric comparison group).* **Results:** *Higher rates of EDs in women who have experienced CSA were confirmed in this study. Belonging to a younger age cohort, experiencing menarche at an early age, and high paternal overcontrol on the PBI independently increased the risk of developing an ED in women who had experienced CSA. Low maternal care was specifically associated with the development of anorexia nervosa, whereas early age of menarche differentiated women with bulimia nervosa. Younger age and early age of menarche also differentiated the CSA+ED women from the psychiatric comparison group.* **Discussion:** *Early maturation and paternal overcontrol emerged as risk factors for ED development in women with CSA. Although these variables are also risk factors in the general population, women with CSA may be vulnerable to ED development because these risk factors are particular domains of concern that emanate from experiences of CSA. © 2001 by John Wiley & Sons, Inc.* Int J Eat Disord 29: 380–392, 2001.

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INTRODUCTION

The literature on possible links between eating disorders and previous child sexual abuse (CSA) began with reports in the late 1980s and early 1990s. In these studies, women

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being treated for an eating disorder were assessed for the prevalence of CSA (Bulik, Sullivan, & Rorty, 1989; Calam & Slade, 1987, 1989; Finn, Hartman, Leon, & Lawson, 1986; Hall, Tice, Beresford, Wooley, & Hall, 1989; Oppenheimer, Howells, Palmer, & Chaloner, 1985; Palmer, Oppenheimer, Dignon, Chaloner, & Howells, 1990; Root & Fallon, 1988; Steiger & Zanko, 1990). The majority reported high rates of CSA among eating-disordered individuals. Since these early studies, several authors have reviewed the work on CSA and eating disorders (Brown, 1997; Connors & Morse, 1993; Pope & Hudson, 1992; Wonderlich, Brewerton, Jocic, Dansky, & Abbott, 1997). Generally, it is agreed that there has been an excessive reliance on clinical samples, which have produced the strongest links between CSA and eating disorders, but which are troubled by referral bias. Throughout the 1990s, several studies using samples drawn from the community have emerged (Bushnell, Wells, & Oakely-Browne, 1992; Garfinkel et al., 1995; Welch & Fairburn, 1994; Wonderlich, Wilsnack, Wilsnack, & Harris, 1996).

In a randomly selected New Zealand community sample, Bushnell et al. (1992) found an increased rate of many adult mental symptoms, including bulimia nervosa (BN), among the 13% who had experienced intrafamilial CSA. An English study that recruited participants through their general practitioners (Welch & Fairburn, 1994), found that CSA was reported more frequently by BN patients than normal general practice controls. However, there was no difference in CSA status between the BN patients and patients with general psychiatric disorders. Community-based studies of BN in Canada (Garfinkel et al., 1995) and the United Kingdom (Fairburn, Welch, Doll, Davies, & O'Connor, 1997) found that CSA was significantly more common in BN patients than in comparison subjects. However, rates of CSA in BN patients were similar to psychiatric comparison women, once again suggesting the presence of a general risk factor not specific to eating disorders. Wonderlich et al. (1996) found that 29% of a nationally representative U.S. sample who reported CSA were also more likely to also report binge eating and purging than nonabused women. These authors concluded that CSA, by itself, is unlikely to be sufficient to cause bulimic behaviors. Finally, Perkins and Luster (1999) found purging behavior to be associated with CSA in a representative sample of middle and high school girls. However, this relationship was not significant when physical abuse and other familial factors were included in the model.

Overall, the findings of these community-based studies have suggested that CSA is a risk factor for psychiatric disorder among young adult women, which includes, but is not specific for, BN. The only study that examined anorexia nervosa (AN) also demonstrated increased rates of abuse in AN patients compared with healthy controls, and a similar rate of abuse in AN patients compared with psychiatric controls (Fairburn, Cooper, Doll, & Welch, 1999).

In the past decade, there have been several nonrandom studies (Kinzl, Traweger, Guenther, & Biebl, 1994; Palmer, 1992; Rorty, Yager, & Rossotto, 1994) that examined the impact of CSA on eating disorder subtype, severity, comorbidity, and treatment response. Several studies (Favaro, Dalle Grave, & Santonastaso, 1998; Garfinkel et al., 1996; Pribor & Dinwiddle, 1992; Steiger & Zanko, 1990) have reported a stronger link between CSA and eating disorders involving binging and purging rather than restricting behaviors, whereas other studies have not (Palmer et al., 1990; Schmidt, Tiller, & Treasure, 1993; Vize & Cooper, 1995). More severe bulimic symptoms have (Waller, 1992), and have not (Bushnell et al., 1992; Favaro et al., 1998; Sullivan, Bulik, Carter, & Joyce, 1995), been associated with abuse experiences. In terms of comorbidity, eating disorder patients in clinical samples who have experienced CSA have higher rates of disorders that are characterized by poor impulse control (Favaro & Santonastaso, 1997; Folsom et al., 1993; Sullivan et al.,

1995; Waller, 1991, 1993). With respect to treatment outcome, Anderson, LaPorte, Brandt, and Crawford (1997) found that BN patients who experienced CSA initially improved as much as those without CSA. However, they were less likely to achieve complete abstinence from bulimic behaviors and they were more likely to relapse.

There is a need for controlled random community surveys with good response rates and careful delineation of both CSA and disordered eating using semistructured interviews with validated instruments. The study described here addresses the issues of specificity and referral bias. Because eating disorders are relatively uncommon in a random community sample, a specially designed study is required to investigate the associations between eating disorders and adverse childhood experiences. This study used a two-stage methodology, which ensured adequate numbers while protecting the representativeness of the sample through random sampling. These data enabled us to examine why some women who were sexually abused as children develop an eating disorder whereas others develop depression and anxiety or other syndromes. We sought to identify the factors that may place women who have experienced CSA at specific risk of eating disorder development.

METHODS

Study Design

A two-stage design in a random community sample of New Zealand women was used to investigate the relationship between a woman reporting CSA before the age of 16 and a later history of either AN or BN. The study is described in detail elsewhere (Anderson, Martin, Mullen, Romans, & Herbison, 1993; Mullen, Martin, Anderson, Romans, & Herbison, 1993). In the first phase, 3,000 women were randomly selected from Otago, New Zealand, electoral rolls. They were mailed a sociodemographic questionnaire that included screening items for CSA. New Zealand law requires that all adults 18 years and older are on the electoral roll. In the second phase, two groups of women were selected for interview: women who indicated that they had experienced CSA (CSA group) and a comparison group of women randomly selected from those who reported no abuse either as a child or as an adult (no CSA group).

Assessment of Psychopathology

The interview covered the details of factors considered relevant to the participant's adult mental health. The quality of the participant's relationship with each parent was quantified at interview with the Parental Bonding Instrument (PBI) for parents or parent substitutes (Parker, Tupling, & Brown, 1979). Current (within the past month) psychiatric disorder was assessed using the short form of the Present State Examination (PSE; Wing, Cooper, & Sartorius, 1974; Wing, Nixon, Mann, & Leff, 1977) and past psychiatric disorder was assessed using the longitudinal form of the PSE (McGuffin, Katz, & Aldrich, 1986). As the short PSE does not document eating disorders, additional questions from the ICD-10 schedule (World Health Organization, 1992) on AN and BN were added. Specifically, AN questions probed for overconcern about eating too much, fear of getting fat, lowest weight and duration of lowest weight, actions taken to avoid getting too fat, and amenorrhea. BN questions probed for eating large amounts of food within an hour (binge eating), loss of

control during binge eating, compensatory measures taken after binge eating, frequency and duration of binge eating, dread of fatness, and dissatisfaction with weight and/or shape.

Assessment and Classification of CSA

During the interview, data were collected on the nature and frequency of CSA, the relationship of the abuser to the subject, and whether force, actual or threatened, was involved. CSA information was then classified into several grades of severity: (A) noncontact abuse (lewd conversation, exposure) and nongenital contact (touching breasts, buttocks, and sexualized kissing). Subjects reported that these categories of CSA were not included in the abuse sample in this analysis; (B) touching of the child's genitals; touching of the assailant's genitalia; attempted sexual intercourse (where the child was immobilized, clothing was removed, and dual genital contact was attempted); sexual intercourse (any act involving penile penetration of the vaginal or anal area, whether or not ejaculation occurred).

Statistical Analyses

Bivariate statistics (chi-square and Fisher's exact test for contingency tables with small cells for discrete variables and analysis of variance [ANOVA] for continuous variables) were used to compare the frequency of eating disorders in the CSA sample versus the no CSA population sample. They were also used to compare sociodemographic variables, family and childhood characteristics, the nature of the CSA, and psychopathology in CSA women with (CSA+ED) and without (CSA–noED) an eating disorder. The CSA+ED women were further categorized into those with anorexia (CSA+AN) and bulimia nervosa (CSA+BN). Finally, sociodemographic variables, family and childhood characteristics, and the nature of the CSA in CSA+ED women were compared with a psychiatric group. Logistic regression was then used to assess which variables made an independent contribution to the likelihood that a CSA victim would develop an eating disorder.

RESULTS

Prevalence of CSA and Disordered Eating

The sampling and prevalence results of the original study can be obtained in detail elsewhere (Anderson et al., 1993). In brief, satisfactory response rates (postal 73%) were obtained. Nongenital CSA was described by 12.2% of the original sample, genital CSA (nonintercourse) by 12.5%, and sexual intercourse by 7.1% (attempted 3.5%, completed 3.8%). A total of 254 women who had experienced CSA and 223 women with no CSA were interviewed. The response rate for those invited for interview was 80% and there was no significant difference in the proportion of women with and without CSA who declined interview.

A comparison of the prevalence of disordered eating between the women with and without a history of CSA is shown in Table 1. There was a higher frequency of both AN and BN in the CSA group than in the group with no CSA. Sixteen of the nineteen (84.2 %) women who gave a history of AN were from the CSA group (relative risk [RR] 5.00, 95% CI 1.44–17.37). The abuse data were incomplete for one subject, who is not included in the

Table 1. Comparison of eating disorder prevalence rates between women with and without a history of CSA

Diagnosis	Overall	CSA	No CSA	CSA Vs. No CSA	
				χ^2	p
N	477	254	223	—	—
Anorexia nervosa	19 (3.9%)	16 (6.3%)	3 (1.3%)	7.6	.006
Bulimia nervosa	26 (5.5%)	19 (7.8%)	7 (3.1%)	4.5	.03
Anorexia + bulimia nervosa	6 (1.3%)	5 (2.0%)	1 (0.4%)	2.2	.1
Any eating disorder	38 (8.2%)	30 (11.8%)	9 (4.0%)	9.6	.002

Note: CSA = childhood sexual abuse.

analysis. The mean age of onset of AN was 20.5 ± 5.3 years (range 14–32 years) with a mean duration of 1.4 ± 1.7 years (range 0.5–7 years). Of the 26 women who gave a history of BN, 19 (73.1%) were from the CSA group (RR 2.55, 95% CI 1.05–6.18). The abuse data were missing or inadequate for classification for two subjects. The mean age of onset of BN was 22.8 ± 7.7 years (range 13–42 years), with a mean duration of 5.1 ± 6.5 years (range <1–29.0 years). Of the 6 women who suffered from both types of eating disorders, 5 (83.3 %) had experienced CSA (Table 1).

Comparison of CSA Women With and Without an Eating Disorder

Sociodemographic Characteristics

The CSA+ED women were more likely to be younger (<30 years old [53%]) than the CSA−noED women (22.8%; $\chi^2 = 15.2$, $p = .0001$). More CSA+ED women were single compared with CSA−noED women (34% vs. 12.1%, $\chi^2 = 9.6$, $p = .002$). Among the ED subgroups, both the CSA+BN (37%, $\chi^2 = 8.2$, $p = .004$) and CSA+AN (31%, $\chi^2 = 3.8$, $p = .05$) were more frequently single than the CSA−noED women. When the younger age of the CSA+ED women was controlled for, the high frequency of unmarried women in each of the CSA+ED subgroups was not significant (data not shown).

There were no differences between any of the CSA+ED groups and the CSA−noED women for their own or parental socioeconomic status (SES) ranking, high school, or tertiary qualifications and the proportions in paid and part-time work.

Parental and Family Characteristics

As shown in Table 2, the frequency of parental separation in childhood was higher and major parental rows and violence were reported more often in the CSA+ED than in the CSA−noED women. The higher rates of parental separation compared with CSA−noED women were observed in each of the ED subgroups. Compared with the CSA−noED group, only the CSA+AN subgroup had significantly higher frequencies of major parental rows and violence. More CSA+ED than CSA−noED women had also lived outside of their nuclear family in childhood.

All of the CSA+ED groups reported significantly higher maternal and paternal control on the PBI subscales than the CSA−noED women. The only significant difference in parental care was lower maternal care in the CSA+AN compared with the CSA−noED women (Table 2).

Table 2. Comparison of parental variables in childhood between the eating-disordered and non–eating-disordered CSA groups

Variable	CSA + ED Combined	CSA + AN	CSA + BN	CSA–noED
N	30	16	19	224
Parents separated	26.7%*	31.3%*	31.5%*	12.9%
Parental rows	45.8%*	56.3%*	35.3%	21.9%
Parental violence	44.2%**	54.4%**	33.2%	21.9%
Lived outside nuclear family in childhood	40%*	41.3%*	39.2%*	23.7%
PBI variables				
Maternal care	17.5 ± 7.1	16.2 ± 10.2*	21.8 ± 10.5	24.0 ± 9.0
Maternal control	16.8 ± 7.6*	17.1 ± 7.3**	16.4 ± 6.4*	14.5 ± 5.8
Paternal care	19.8 ± 11.1	20.8 ± 11.9	18.8 ± 8.5	22.3 ± 9.3
Paternal control	19.1 ± 9.0**	17.7 ± 8.4*	19.5 ± 9.3**	13.5 ± 7.6

Note: CSA = childhood sexual abuse; ED = eating disorder; AN = anorexia nervosa; BN = bulimia nervosa; PBI = Parental Bonding Instrument. Data shown are percent for discrete variables and *M* + *SD* for continuous variables.

Statistics (χ^2 or *F*-ratio) are for comparison between each of the eating disorder groups with the CSA–noED group. *$p < .05$. **$p < .01$.

Early Physical Characteristics and Personality

There were no differences in the CSA+ED and CSA–noED groups in their attitudes toward puberty, age of first (nonabuse) intercourse, whether sex education was received from parents, ease with which menstruation was discussed with mother, and child health problems (data not shown). Compared with CSA–noED women, significantly more of the CSA+ED women experienced menarche before they were 12 years old (46.7% vs. 21.2%, $\chi^2 = 21.2$, $p = .01$). This finding was significant for the CSA+BN (68.4%, $p = 0.001$) but not the CSA+AN (37.5%, $p = 0.2$) subgroup.

Each of the CSA+ED (36.7%, $\chi^2 = 12.2$, $p = .002$), CSA+AN (31.3%, $\chi^2 = 8.1$, $p = .02$), and CSA+BN (47.4%, $\chi^2 = 17.8$, $p = .0001$) groups was more likely to report a lack of confidence in childhood compared with CSA–noED (13.8%) women. The CSA+ED and CSA–noED women did not differ on descriptions of themselves as loners, as followers, or of being shy. However, the CSA+BN women were slightly less likely to endorse shyness than the CSA–noED women (15.8% vs. 28.1%, $\chi^2 = 2.9$, $p = .09$) and were significantly less shy than the CSA+AN women (31.3%, $\chi^2 = 3.5$, $p = .05$).

CSA Variables and Help Seeking

The CSA variables examined included age of CSA onset, frequency, type, duration, relationship of the abuser to the victim, abuser tactics, feelings at the time of the abuse, reaction when told someone about the abuse, and help seeking. Table 3 shows a comparison of CSA-related variables between the CSA+ED and CSA–noED groups that emerged as significant. CSA+ED women were more likely to have been abused 10 or more times and to have experienced feelings of disgust at the time of abuse than CSA–noED women. There was a trend for more intrusive forms of CSA (attempted or completed intercourse) to be more common in CSA+ED women ($p = .06$).

Compared with the CSA–noED group, the CSA+AN women were more likely to have been abused by their fathers or stepfathers, to have experienced CSA of attempted or completed intercourse, and to have been abused 10 or more times. They were also more likely to report feelings of disgust at the time of abuse (Table 3). For the CSA+BN women,

Table 3. Comparison of CSA variables between the eating-disordered and non–eating-disordered CSA groups

Variable	CSA + ED Combined	CSA + AN	CSA + BN	CSA–noED
N	30	16	19	224
Abused 10+ times	30%*	37.5%*	21.9%	11.3%
Felt disgust at time of abuse	33.3%**	43.8%***	36.8.3%***	10.7%
Intrusive form of abuse	23.3%	31.3%*	18.2%	11.2%
Abused by father/stepfather	12.1%	25%*	9%	6.6%
Abused before 11 years old	60.1%	48.2%	78%	39%

Note: CSA = childhood sexual abuse; ED = eating disorder; AN = anorexia nervosa; BN = bulimia nervosa. Statistics (χ^2) are for comparison between each of the eating disorder groups with CSA–noED group. *$p < .05$. **$p < .01$. ***$p < .001$.

the victim's age at the time of CSA was significant; more CSA+BN than CSA–noED women reported abuse before 11 years of age. Similar to the CSA+AN women, CSA+BN women were also more likely to report feelings of disgust at the time of abuse than the CSA–noED women (Table 3).

There was a trend for more CSA+AN women to have sought help from a professional for an emotional disorder at any stage than CSA–noED women (56% vs. 33.3%, $\chi^2 = 3.47$, $p = .06$), whereas more CSA–BN women had clearly done so (57.9% vs. 32.6%, $\chi^2 = 4.96$, $p = .03$).

Psychiatric Comorbidity

Rates of psychiatric comorbidity in the CSA+ED and the CSA–noED groups are compared in Table 4. Higher lifetime prevalence rates of major depression, anxiety, and suicide attempts were observed in all of the eating disorder groups. However, the higher prevalence of major depression in the CSA+AN group did not reach statistical significance ($p = .08$). Despite higher rates of social anxiety and phobias in all of the CSA+ED groups, only the CSA+AN women had significantly higher rates of any anxiety disorder. Rates of substance dependence did not differ between the groups.

Table 4. Lifetime psychiatric comorbidity in the eating disordered and non-eating disordered CSA groups

Psychopathology	CSA + ED Combined	CSA + AN	CSA + BN	CSA–noED
N	30	16	19	224
Major Depression	78%*	75%	84.2%*	54%
Anxiety	36.7%	56.3%	36.8%	26.8%
Panic disorder	13.3%***	12.5%*	21.1%****	2.7%
Phobias	20%**	50%*	42.1%*	4.5%
Social anxiety	30%**	37.5%*	42.1%**	16.1%
Obsessionality[a]	13.3%	12.5%	10.5%	5.4%
Substance dependence	16.7%	18.8%	15.8%	8.5%
Attempted suicide	30%****	37.5%****	31.6%**	6.7%

Note: CSA = childhood sexual abuse; ED = eating disorder; AN = anorexia nervosa; BN = bulimia nervosa.
[a]Includes obsessional cleanliness, checking, and ideas combined.
Statistics (χ^2) are for comparison between each of the eating disorder groups and the CSA–noED group. *$p < .05$. **$p < .01$. ***$p < .001$. ****$p < .0001$.

Mathematical Model

Logistic regression showed that younger age ($r = -.23$, $p = .002$), paternal control ($r = .20$, $p = .006$), and earlier age of menarche ($r = -.18$, $p = .01$) all produced independent effects on the development of any eating disorder among women with CSA. In addition to these risk factors, low maternal care ($r = -.18$, $p = .03$) produced an independent effect for predicting the development of AN. Early age of menarche ($r = -.20$, $p = .02$) was an independent predictor of developing BN.

Comparison Between CSA+ED and Psychiatric Comparison Women

In the following analyses, CSA+ED women were compared on a number of variables with CSA women with a lifetime diagnosis of depression/anxiety (CSA+DEPANX) but who did not give a history of an eating disorder. The figures among the CSA groups are CSA+ED (with or without depression/anxiety, $n = 30$, 11.8% of the CSA sample) and CSA+DEPANX ($n = 136$, 53.5% of the CSA sample).

Sociodemographic Characteristics

The CSA+ED and CSA+DEPANX groups did not differ significantly with respect to their own or parental SES, high school or tertiary qualifications, and the proportions in paid and part-time work. More CSA+ED women were under 40 years of age at the time of interview than the CSA+DEPANX women (86% vs. 47.8%, $\chi^2 = 15.1$, $p = .001$). The CSA+ED women were more often unmarried and separated/divorced than the CSA+DEPANX group ($\chi^2 = 10.34$, $p = .006$), but this was explained by the younger age of the CSA+ED women.

Parental and Family Characteristics

In the CSA+ED group, major parental rows (46.7% vs. 21.9%, $\chi^2 = 7.8$, $p = .02$) and parental violence (46.7% vs. 26.5%, $\chi^2 = 4.8$, $p = .03$) were reported more frequently than in the CSA+DEPANX women. Higher frequencies of parental separation in childhood (26.7% vs. 13.2 %, $\chi^2 = 3.4$, $p = .07$) and living outside of the nuclear family (40% vs. 24.3%, $\chi^2 = 3.1$, $p = .08$) in the CSA+ED women were not significantly different from the CSA+DEPANX women. The only variable on the PBI that differed significantly between groups was an elevated score on the maternal control subscale in the CSA+ED women (16.8 ± 7.6 vs. 14.9 ± 5.9, F ratio = 4.6, $p = .02$).

Early Physical Characteristics and Personality

There were no differences in the CSA+ED and CSA+DEPANX groups in their attitudes toward puberty, age of first (nonabuse) intercourse, whether sex education was received from parents, and child health problems (data not shown). The CSA+ED women were more likely to report menarche onset at age younger than 12 years than CSA+DEPANX women (46.7% vs. 22.1%, $\chi^2 = 4.4$, $p = .03$).

The childhood personality items on shyness, leadership, and follower did not differ between the CSA+ED and CSA+DEPANX groups. CSA+ED women were more likely than CSA+DEPANX women to regard themselves as extraverted in childhood (36.7% vs. 15.4%, $\chi^2 = 7.2$, $p = .03$). The CSA+BN subgroup accounted for this relationship. A lack of confidence was also reported more frequently in the CSA+ED women than in the CSA+DEPANX women (36.7% vs. 14.5%, $\chi^2 = 8.1$, $p = .02$).

CSA and Help-Seeking Variables

There were no differences between CSA+ED and CSA+DEPANX women on the intrusiveness (attempted and completed intercourse) of CSA, relationship of perpetrator to victim, onset of abuse, or abuser tactics (data not shown). The only CSA variable that significantly differed between the CSA+ED and CSA+DEPANX women was a higher frequency of feelings of disgust in the CSA+ED women (33% vs. 11%, $\chi^2 = 20.0$, $p = .04$). More CSA+ED women than CSA+DEPANX women reported a duration of abuse longer than 1 year but this was not statistically significant (60% vs. 33.8%, $\chi^2 = 18.3$, $p = .1$). No differences were found between CSA+ED and CSA+DEPANX groups for help-seeking variables or for seeing a professional.

Psychiatric Comorbidity

The CSA+ED group was significantly more likely to have ever made a minor or major suicide attempt (30% versus 6.6%, $\chi^2 = 15.4$, $p = .001$) than were the CSA+DEPANX women. Lifetime rates of social anxiety (30% vs. 16.9%, $\chi^2 = 6.3$, $p = .04$) and panic disorder (13.3% vs. 3.7%, $\chi^2 = 9.8$, $p = .01$) were also significantly higher in the CSA+ED women than in the CSA+DEPANX women. Lifetime rates of substance dependence, obsessional ideas, depression, and having any anxiety disorder were not significantly different between the groups (data not shown).

Mathematical Model

Logistic regression showed that belonging to a younger age cohort ($r = -0.24$, $p = .002$) and early age of menarche ($r = -.18$, $p = 0.02$) independently predicted the development of an eating disorder as opposed to the development of an anxiety disorder or depression.

DISCUSSION

This community-based study confirmed higher rates of eating disorders in women who have experienced CSA. It also examined how some women sexually abused as children develop an eating disorder whereas others develop depression and anxiety. A younger age cohort, onset of menarche at an early age, and paternal overcontrol independently increased the risk of developing an eating disorder in women who had experienced CSA. In addition, low maternal care was specifically associated with the development of AN, whereas early age of menarche was the only specific variable that differentiated women with BN. A younger age cohort and early age of menarche onset also differentiated the CSA+ED women from the CSA women who had only anxiety or depression (psychiatric comparison women).

Epidemiological studies have found lifetime and current rates of eating disorders to be higher in more recent birth cohorts (Bushnell, Wells, Hornblow, Oakley-Browne, & Joyce, 1990), which indicates a growing prevalence in recent years. Our data suggest that this trend observed in general population samples is also seen within one high-risk group of CSA women. In addition, our data suggest that among women with CSA, belonging to a younger age cohort was not a risk factor for other psychiatric disorders, but was specific to eating disorder development.

Low care and high control on the PBI have been associated with dysfunctional psychosocial development (Parker, 1983). Similar to the findings of Calam, Waller, Slade, and Newton (1989), paternal, but not maternal, overcontrol emerged as a risk factor for eating disorder development in this study. Fairburn et al. (1999, 1997) identified high levels of

both maternal and paternal overcontrol in a community-based sample of women with BN and AN compared with controls. Interestingly, lower levels of perceived control have also been observed in eating-disordered women with a history of sexual abuse than those without a history of abuse (Waller, 1998). Parental overcontrol has been hypothesized to impede the development of autonomy in children (Costanzo & Woody, 1985). These children may perceive themselves to have the inability to gain control over their own lives, which is construed as a major variable in some formulations of the etiology of eating disorders (Slade, 1982).

This study did not demonstrate a direct association between prior discomfort with their bodies (as reflected by difficulty in discussing menstruation with their mothers) and eating disorder development in CSA women. However, early age of menarche was found to be a risk factor for eating disorder development in women who had experienced CSA. Menarche prior to 12 years of age was reported as a risk factor for BN in a community-based case-control study (Fairburn et al., 1997). It has also previously been implicated in the development of AN (Crisp, 1970). Early menarche involves early exposure to the increase in body fat and changes in body shape that are associated with puberty. This in turn may precipitate dieting, itself a risk factor for the development of eating disorders (Heatherton & Polivy, 1990). It is of note that menarche tends to occur earlier in overweight and obese girls (Scott & Johnston, 1982) and in women who report CSA (Romans, Martin, & Bouwer, 2001). Although we did not assess childhood body weight in this study, it is possible that overweight in childhood mediates the relationship between early menarche onset and eating disorders in women with CSA. Similar to the findings for belonging to a younger age cohort, early menarche was not a risk factor for other psychiatric disorders, but was specific to eating disorder development.

Although a number of variables other than age of menarche and parental control have been identified as risk factors for eating disorders (Halmi, 1997; Johnson & Wonderlich, 1990; Leon, Klump, & Fulkerson, 1997; Streigel-Moore, 1997), why have these two risk factors emerged in women with a history of CSA in this study? It has been hypothesized that eating disorders represent both an attempt to gain a sense of control (Bruch, 1973; King, 1989) and to avoid sexual maturation (Crisp, 1967), which may be the very domains of concern that emanate from experiences of CSA. Perceived loss of control (Beckman & Burns, 1990) and heightened concerns about sexual maturation (Brown, 1997) have been described as consequences of CSA.

This study did not find independent relationships between CSA variables (e.g., nature and duration) and eating disorder status. Although a high frequency of abuse and feelings of disgust at the time of abuse were associated with eating disorders, these variables were not significant in the overall model. Although women with CSA are at increased risk for developing an eating disorder, this is likely due to an increased risk of having any psychiatric disorder because no specific CSA variables contributed to risk within a sample of CSA women.

We acknowledge several limitations of this study. These data were retrospectively obtained and focused on the effects of abuse prior to 16 years of age. However, abuse in adulthood may be less relevant to eating disorder development because the onset of the majority of eating disorder cases occurs in adolescence and early adulthood. Eating disorder variables such as symptom severity and outcome were not assessed. Thus, we are unable to determine the influence of CSA variables on such factors. We did not determine restricting versus bulimic behaviors among AN women, nor could we determine the relative influence of CSA variables on determining the subtype of AN. Because our

sampling strategy was based on community cases of CSA rather than on cases of eating disorders, small numbers of eating disorders cases may also limit our findings.

In summary, the findings of this study suggest that the risk factors for eating disorder development in women with a history of CSA are similar to those that operate in the general population. The challenge for future work may be to determine more precisely the mechanisms by which early sexual maturation, perceived lack of control, and CSA are linked to eating disorder development.

REFERENCES

Anderson, J., Martin, J., Mullen, P., Romans, S., & Herbison, P. (1993). The prevalence of childhood sexual experiences in a community sample of women. Journal of the American Academy of Child and Adolescent Psychiatry, 32, 911–919.

Anderson, K.P., LaPorte, D.J., Brandt, H., & Crawford, S. (1997). Sexual abuse and bulimia: Response to inpatient treatment and preliminary outcome. Journal of Psychiatry Research, 31 (6), 621–633.

Beckman, K.A., & Burns, G.L. (1990). Relation of sexual abuse and bulimia in college age women. International Journal of Eating Disorders, 9, 467–492.

Brown, L. (1997). Child sexual abuse and eating disorders: A review of the links and personal comments on the treatment process. Australian and New Zealand Journal of Psychiatry, 31, 194–199.

Bruch, H. (1973). Eating disorders: Obesity, anorexia nervosa and the person within. New York: Basic Books.

Bulik, C.M., Sullivan, P.F., & Rorty, M. (1989). Childhood sexual abuse in women with women with bulimia. Journal of Clinical Psychiatry, 50, 460–464.

Bushnell, J.A., Wells, J.E., Hornblow, A.R., Oakley-Browne, M.A., & Joyce, P.R. (1990). Prevalence of three bulimia syndromes in the general population. Psychological Medicine, 20, 671–680.

Bushnell, J.A., Wells, J.E., & Oakely-Browne, M. (1992). Long term effects of intrafamilial sexual abuse in childhood. Acta Psychiatrica Scandinavica, 85, 136–142.

Calam, R., & Slade, P. (1987). Eating problems and sexual experience: Some relationships. British Review of Bulimia and Anorexia Nervosa, 2, 37–43.

Calam, R., & Slade, P. (1989). Eating problems in female undergraduates. International Journal of Eating Disorders, 8, 391–397.

Calam, R., Waller, G., Slade, P., & Newton, T. (1989). Eating disorders and perceived relationships with parents. International Journal of Eating Disorders, 9 (5), 479–485.

Connors, M., & Morse, W. (1993). Sexual abuse and eating disorders: A review. International Journal of Eating Disorders, 13, 1–11.

Costanzo, P.R., & Woody, E.Z. (1985). Domain-specific parenting styles and their impact on the child's development of particular deviance: The example of obesity proneness. Journal of Social and Clinical psychology, 3 (4), 425–445.

Crisp, A.H. (1967). Anorexia nervosa. Hospital Medicine, 1, 713–718.

Crisp, A.H. (1970). Reported birth weights and growth rates in a group of patients with primary anorexia nervosa. Journal of Psychosomatic Research, 14, 23–50.

Fairburn, C.G., Cooper, Z., Doll, H.A., & Welch, S.L. (1999). Risk factors for anorexia nervosa. Archives of General Psychiatry, 56, 468–476.

Fairburn, C.G., Welch, S.L., Doll, H.A., Davies, B.A., & O'Connnor, M.E. (1997). Risk factors for bulimia nervosa. Archives of General Psychiatry, 54, 509–517.

Favaro, A., Dalle Grave, R., & Santonastaso, P. (1998). Impact of a history of physical and sexual abuse in eating disordered and asymptomatic subjects. Acta Psychiatrica Scandinavica, 97, 358–363.

Favaro, A., & Santonastaso, P. (1997). Suicidality in eating disorders: Clinical and psychological correlates. Acta Psychiatrica Scandinavica, 95, 508–514.

Finn, S., Hartman, M., Leon, G., & Lawson, L. (1986). Eating disorders and sexual abuse: Lack of confirmation for a clinical hypothesis. International Journal of Eating Disorders, 5, 1051–1060.

Folsom, V., Khran, D., Nairm, K., Gold, L., Demitrack, M.A., & Silk, K.R. (1993). The impact of physical and sexual abuse on eating disordered and psychiatric symptoms: A comparison of eating disordered and psychiatric inpatients. International Journal of Eating Disorders, 13, 249–257.

Garfinkel, P.E., Lin, E., Goering, P., Spegg, C., Goldbloom, D.S., Kennedy, S., Kaplan, A.S., & Woodside, D.B. (1995). Bulimia nervosa in a Canadian community sample: Prevalence and comparison of subgroups. American Journal of Psychiatry, 152, 1052–1058.

Garfinkel, P.E., Lin, E., Goering, P., Spegg, C., Goldbloom, D.S., Kennedy, S., Kaplan, A.S., & Woodside, D.B. (1996). Purging and nonpurging forms of bulimia nervosa in a community sample. International Journal of Eating Disorders, 20 (3), 231–238.

Hall, R., Tice, L., Beresford, T., Wooley, B., & Hall, A. (1989). Sexual abuse in patients with anorexia and bulimia. Psychosomatics, 30 (1), 73–79.
Halmi, K.A. (1997). Models to conceptualize risk factors for bulimia nervosa. Archives of General Psychiatry, 54, 507–508.
Heatherton, T.F., & Polivy, J. (1990). Chronic dieting and eating disorders: A spiral model. In J.H. Crowther, D.L. Tennenbaum, S.E. Hobfoll, & M.A. Parris Stephens (Eds.), The etiology of bulimia nervosa: The individual and family context (pp. 157–177). OH: Hemisphere.
Johnson, C., & Wonderlich, S. (1990). Personality characteristics as a risk factor in the development of eating disorders. In J.H. Crowther, D.L. Tennenbaum, S.E. Hobfoll, & M.A. Parris Stephens (Eds.), The etiology of bulimia nervosa: The individual and familial context (pp. 179–196). Washington, D.C.: Hemisphere Publishing Corporation.
King, M. (1989). Locus of control in women with eating pathology. Psychological Medicine, 12, 183–187.
Kinzl, J., Traweger, C., Guenther, V., & Biebl, W. (1994). Family background and sexual abuse associated with eating disorders. American Journal of Psychiatry, 151, 1127–1131.
Leon, G.R., Keel, P.K., K., Klump, K.L., & Fulkerson, J.A. (1997). The future of risk factor research in understanding the etiology of eating disorders. Psychopharmacology Bulletin, 33 (3), 405–411.
McGuffin, P.E., Katz, R., & Aldrich, J. (1986). Past and present state examinations: The assessment of "lifetime ever" psychopathology. Psychological Medicine, 6, 461–465.
Mullen, P.E., Martin, J.L., Anderson, J.C., Romans, S.E., & Herbison, G.P. (1993). Child sexual abuse and mental health in adult life. British Journal of Psychiatry, 163, 721–732.
Oppenheimer, R., Howells, K., Palmer, R.L., & Chaloner, D.A. (1985). Adverse sexual experience in childhood and clinical eating disorders: A preliminary description. Journal of Psychiatric Research, 19, 357–361.
Palmer, R.L. (1992). Effects of child sexual abuse on adult mental health. British Journal of Hospital Medicine, 48 (1), 9–10.
Palmer, R.L., Oppenheimer, R., Dignon, A., Chaloner, D., & Howells, K. (1990). Childhood sexual experience with adults reported by women with eating disorders: An extended series. British Journal of Psychiatry, 156, 699–703.
Parker, G. (1983). Parental overprotection. A risk factor for psychosocial development. New York: Grune and Stratton.
Parker, G., Tupling, H., & Brown, L.B. (1979). A Parental Bonding Instrument. British Journal of Medical Psychology, 52, 1–10.
Perkins, D.F., & Luster, T. (1999). The relationship between sexual abuse and purging: Findings from community-wide surveys of female adolescents. Child Abuse and Neglect, 23 (4), 371–382.
Pope, H., & Hudson, J. (1992). Is childhood sexual abuse a risk factor for bulimia nervosa? American Journal of Psychiatry, 149, 455–463.
Pribor, E.F., & Dinwiddle, S.H. (1992). Psychiatric correlates of incest in childhood. American Journal of Psychiatry, 149, 52–56.
Romans, S.E., Martin, J.M., & Bouwer, C. (2001). Age of menarche and childhood adversity. Manuscript in preparation.
Root, M., & Fallon, P. (1988). The incidence of victimization experiences in a bulimic sample. Journal of Interpersonal Violence, 3, 161–173.
Rorty, M., Yager, J., & Rossotto, E. (1994). Childhood sexual, physical and psychological abuse and their relationship to comorbid psychopathology in bulimia nervosa. International Journal of Eating Disorders, 16 (4), 317–334.
Schmidt, U., Tiller, J., & Treasure, J. (1993). Setting the scene for eating disorders: Childhood care, classification and course of illness. Psychological Medicine, 23, 663–672.
Scott, E.C., & Johnston, F.E. (1982). Critical fat, menarche, and the maintenance of menstrual cycles: A review. Journal of Adolescent Health Care, 2, 249–260.
Slade, P.D. (1982). Towards a functional analysis of anorexia and bulimia nervosa. British Journal of Clinical Psychology, 21, 167–179.
Steiger, H., & Zanko, M. (1990). Sexual trauma among eating-disordered, psychiatric and normal female groups. Journal of Interpersonal Violence, 5, 74–86.
Streigel-Moore, R.H. (1997). Risk factors for eating disorders. Annals of the New York Academy of Sciences, 817, 98–109.
Sullivan, P.F., Bulik, C.M., Carter, F.C., & Joyce, P.R. (1995). The significance of a prior history of sexual abuse in bulimia nervosa. British Journal of Psychiatry, 167, 679–682.
Vize, C.M., & Cooper, P.J. (1995). Sexual abuse in patients with eating disorder, patients with depression, and normal controls. British Journal of Psychiatry, 167, 80–85.
Waller, G. (1991). Sexual abuse as a factor in eating disorders. British Journal of Psychiatry, 159, 664–671.
Waller, G. (1992). Sexual abuse and severity of bulimic symptoms. British Journal of Psychiatry, 161, 90–93.
Waller, G. (1993). Sexual abuse and eating disorders. Borderline personality disorder as a mediating factor? British Journal of Psychiatry, 162, 771–775.
Waller, G. (1998). Perceived control in eating disorders: Relationship with reported sexual abuse. International Journal of Eating Disorders, 23, 213–216.

Welch, S., & Fairburn, C. (1994). Sexual abuse and bulimia nervosa: Three integrated case control comparisons. American Journal of Psychiatry, 151, 402–407.
Wing, J.K., Cooper, J.E., & Sartorius, N. (1974). The measurement and classification of psychiatric symptoms. London: Cambridge University Press.
Wing, J.K., Nixon, J.M., Mann, S.A., & Leff, J.P. (1977). Reliability of the PSE used in a population study. Psychological Medicine, 7, 505–516.
Wonderlich, S.A., Brewerton, T.D., Jocic, Z., Dansky, B.S., & Abbott, D.W. (1997). Relationship of childhood sexual abuse and eating disorders. Journal of the American Academy of Child and Adolescent Psychiatry, 36 (8), 1107–1115.
Wonderlich, S.A., Wilsnack, R.W., Wilsnack, S.C., & Harris, T.R. (1996). Childhood sexual abuse and bulimic behavior in a nationally representative sample. American Journal of Public Health, 86 (8, Pt. 1), 1082–1086.
World Health Organization. (1992). The ICD-10 classification of mental and behavioral disorders—clinical descriptions and diagnostic guidelines. Geneva: Author.