Chapter 23

Child sexual abuse

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Summary

This chapter summarizes the evidence of a relationship between child sexual abuse and subsequent mental disorder. Child sexual abuse (CSA) typically includes unwanted and inappropriate sexual solicitation of, or exposure to, a child by an older person; genital touching or fondling; or penetration in terms of oral, anal or vaginal intercourse or attempted intercourse. CSA can vary along a number of dimensions including frequency, duration, age at onset, and relationship of victim to perpetrator. However, the most common dimension used to define CSA is type of abuse. Three categories are commonly reported in the literature. Non-contact abuse encompasses a range of acts and includes inappropriate sexual solicitation or indecent exposure. The two other categories are contact abuse, which includes touching or fondling, and intercourse, which includes oral, anal or vaginal intercourse. In this chapter the upper age limit used to define childhood was 18 years. The theoretical minimum exposure was defined as no abuse.

The disease outcomes chosen for the current analysis were depression, panic disorder, post-traumatic stress disorder (PTSD), alcohol abuse/dependence, drug abuse/dependence and suicide attempts. Evidence for causality came from twin studies, prospective studies and representative community studies. In particular, the three twin studies available provided strong evidence of a causal relationship as they inherently controlled for the genetic and family environment factors that are also associated with mental disorders (Dinwiddie et al. 2000; Kendler et al. 2000; Nelson et al. 2002). These studies provide evidence of significant associations between CSA and depression, panic disorder, alcohol abuse/dependence, drug abuse/dependence and suicide attempts. While PTSD was not considered as an outcome in the twin studies, data from a prospective study (Silverman et al. 1996) and three representative community studies (Davidson et al. 1991; Molnar et al. 2001; Saunders

et al. 1999) provided consistent evidence that a strong association exists. There was insufficient evidence to support the relationship with obsessive–compulsive disorder (OCD) and so it was excluded. A number of other mental disorder outcomes that were not considered here have been linked to child sexual abuse. Eating disorders have long been conceptualized as a response to a dysfunctional family environment, of which child sexual abuse can be a part. A number of studies have also looked at the association between child sexual abuse and personality disorders, particularly antisocial and borderline personality disorder. Additionally, child sexual abuse does not only produce an increased risk of mental disorder. There has been anecdotal and experimental evidence suggesting that CSA increases the probability of negative psychological outcomes such as poor self-esteem, lack of a sense of control or agency, difficulties with intimacy and continuing sexual difficulties.

Review articles on the prevalence of child sexual abuse have commonly reported a range of prevalence anywhere from 2% to 62%. Previous studies have demonstrated a significant difference in the prevalence of CSA, depending on a number of methodological factors, including method of data collection, number of questions used to assess CSA, definition of childhood and the type of sample assessed. Therefore, multiple linear regression analyses were employed separately for males and females to identify the methodological characteristics that significantly contribute to the variation in prevalence. Any unwanted variation was removed by adjusting individual prevalence estimates accordingly. These "adjusted" prevalence estimates were then broken down into the three levels of abuse (non-contact, contact and intercourse) and the eight age groups. Meta-analysis was then used to combine the estimates within each country. These country estimates were weighted by the population of the country and combined to provide the final subregional[1] estimates. Prevalence estimates were higher in females than in males and varied across subregions with the highest prevalence estimates found in AFR-E and SEAR-D.

Relative risk estimates were derived from studies examining the relationship between CSA and psychiatric outcomes. Where relative risks were reported only for overall exposure to CSA, these were extrapolated into the three levels of abuse. Few studies adjusted for the confounding effects of family dysfunction. An external method of adjustment was applied to the relative risks from those studies that did not control for family dysfunction. The small number of studies available for analysis meant that separate relative risks for different age, sex and subregional groups could not be obtained. Relative risks were stratified by psychiatric outcome before being combined using meta-analysis. Results showed that the relative risks are not significantly different across types of mental disorder suggesting that CSA is not particularly associated with any one disorder. Additionally, across types of abuse there was a general trend for increased risk to be associated with "increased" exposure to CSA.

That is, as more severe forms of CSA were experienced the risks for developing a mental disorder increased. After external adjustment the contact and intercourse categories of abuse remained significant across most disorders but non-contact abuse became non-significant.

Across the world CSA contributed to between 4% and 5% of the burden of disease in males and between 7% and 8% of the burden of disease in females, for each of the conditions depression, alcohol abuse/dependence and drug abuse/dependence. The attributable fractions were higher for panic disorder (7% for males and 13% for females) and higher still for PTSD (21% for males and 33% for females). For suicide attempts attributable fractions were 6% for males and 11% for females. There were slight regional variations in the amount of burden that could be attributed to CSA, with AFR-E and SEAR-D having higher attributable fractions. Prevalence was estimated to be higher in these subregions. However, data for these subregions came from a few studies that were methodologically poor. The burden of disease attributed to CSA was greater in the younger age groups and declined in the older age groups. Since risk was assumed to be constant across age, this merely reflected the age distribution of the mental disorder disease burden, which impacts largely on the younger age groups due to its early onset and chronic nature. Avoidable burden would be the same as attributable since it was assumed that the prevalence of CSA does not change over time under a "business-as-usual" scenario.

1. Introduction

1.1 Definitions of risk factor

Definitions of exposure to child sexual abuse vary. Patterns of interpersonal behaviour are being described, not the results of measuring a physical attribute like body mass index, hypertension or blood lead levels. In physical inactivity we accept the report of a person's habitual behaviour and calculate the health consequences of that behaviour to that person. In CSA we quantify one person's behaviour with a child, and usually have to rely on the retrospective report of that child when adult. We then estimate the health consequences of that occurrence. It is important to point out that, even in prospective studies, data on CSA are gathered retrospectively. It is unethical, and in many countries illegal, to prospectively identify CSA and not intervene. The problems of measurement have proven to be difficult but not insurmountable.

In its broadest sense CSA includes unwanted and inappropriate sexual solicitation of, or exposure to, a child by an older person (non-contact abuse), genital touching or fondling (contact abuse), and penetration in terms of oral, anal or vaginal intercourse or attempted intercourse (intercourse). Many studies have used a narrow definition of CSA to include contact abuse and intercourse only. Definitions of CSA also differ

depending on the cut-off age used to define childhood. While in most countries 18 years of age is the legal cut-off used to define childhood, in many countries the age of consent, especially for sexual activity, is lower. However, the most widely-reported definition of childhood in large population surveys of CSA is 18 years or less. Very few studies provided estimates of prevalence by different age groups and none provided estimates of mental disorder risk by different age groups. Furthermore, as described below, it has been shown that the first onset of CSA is less likely to occur between the ages of 15 and 18 years than in younger children. For these reasons the cut-off age used to define childhood in this chapter was set at 18 years.

1.2 Choice of exposure variable, reasons and implications

The causes of adult mental disorders have proven difficult to define. There is considerable evidence from longitudinal and twin studies that both genetic and environmental factors are implicated in different proportions in different disorders. Because we are dealing with human behaviour there is also evidence of substantial gene–environment interaction (Kendler et al. 2000; Rutter 1999). Advances in medicine generally have been simplified by the availability of animal models of the condition, which allow the biology to be explicated. Animal models of mental disorders just do not exist, and progress in understanding has to be made by association and inference and does not come from experimental paradigms.

It is simplistic to assume that genetic contributions are immutable. Few would claim that the genetic factors are, except in the case of Huntington chorea and some dementias, a full and sufficient explanation. Genetic factors act to enhance vulnerability. For example, the indicated prevention programmes in anxiety disorders almost certainly work by inhibiting the expression of that genetic vulnerability. Nevertheless, it is inherently more plausible to identify a risk factor that is purely environmental, as CSA is, when looking for a risk factor that might be avertible.

In broad terms, the risk factors for mental disorders can be grouped into vulnerability produced by temperament, by adversity and by deprivation. Trauma and CSA are examples of adversity; family dysfunction and neglect are examples of deprivation (Bryant and Range 1995; Kessler et al. 1997). Temperament, adversity and deprivation co-occur more often than is predicted by chance and, as these impact throughout childhood and adolescence in complex ways, their influence in adult functioning is not likely to be simple or linear (Mullen et al. 1996, 2000; Rutter 1999).

CSA is no exception to this rule. It is more frequent in situations in which the other factors are present. Fortunately, it is possible to estimate the independent contribution of CSA to adult mental disorders from both

twin studies on twin pairs discordant for CSA (where both genetics and family environment like deprivation and other adversity are controlled by virtue of the twin method), and longitudinal studies of young people growing up (where data were prospectively gathered on temperament, non-CSA adversity and deprivation) (Borowsky et al. 1999; Kendler et al. 2000; Molnar et al. 2001; Mullen et al. 1993, 1996; Yama et al. 1995; Zuravin and Fontanella 1999). The link between CSA and adult mental disorders has been established, even after controlling for these other determinants of adult mental disorders.

Two other issues make CSA an appropriate exposure variable to choose. First, it is not rare—most reviews have concluded that close to one child in six experiences an episode of CSA as defined using the broad definition above (Fergusson and Mullen 1999). Second, there is an extensive literature on CSA, much of it recent and much using the definitions presented above, which made a systematic review possible. The literature on deprivation or other adversity is nowhere near as extensive, coherent or accessible.

1.3 Choice of theoretical minimum

Given the nature of CSA and the way it is defined in this chapter, the only acceptable theoretical minimum is zero.

2. Estimating risk factor levels

2.1 Methods

The methods used to identify sources and studies for estimation of both risk factor levels and risk factor–disease relationships are presented in the following section.

2.2 Criteria for considering sources and studies

The following inclusion criteria were used:

- any study which determined the prevalence of childhood sexual abuse in any sample;
- any study which determined both the presence and absence of CSA and the subsequent presence and absence of our chosen outcomes;
- any review chapters or reports published in the last 10 years where the topic was CSA (books where the central topic was CSA were included regardless of year of publication);
- methodological papers to assist with the interpretation of the results; and
- meta-analyses of original research results.

The following exclusion criteria were then applied before collection of the articles:

- prevalence studies with total sample sizes of less than 100 (unless data was from an underrepresented country); and
- studies where the population was sampled on the basis of the presence of one of our chosen outcomes.

The first exclusion criterion was applied on the basis of the recommendation of Fergusson and Mullen (1999) and because samples of less than 100 may not provide accurate prevalence estimates for CSA. The second exclusion criterion was applied because studies that sampled on the basis of the presence or absence of an outcome could not be used to calculate a relative risk for that outcome (Streiner 1998). In epidemiological terms this means we could not determine the number of CSA-exposed individuals who developed the outcome of interest. Instead, we determined the number of people with outcome A who were exposed to CSA, thus answering a different question. Case–control studies were therefore only included where "cases" were individuals exposed to CSA and "controls" were those who were not exposed.

2.3 Search strategy for identification of studies

Several strategies were used to locate studies for this chapter. First, computer searches of 16 databases were conducted. Databases searched were the following: Medline; Embase; Psychinfo; E-psych; Healthstar; Cinahl; Cochrane; Social Work Abstracts; Health & Society; Family & Society; General Science Abstracts; Cambridge Life Sciences; Family Studies; Dissertation Abstracts; Child Abuse; Child Welfare & Adoption; Social Sciences Citation Index.

Subject heading (SH) and key word (KW) searches were carried out in two stages and were defined as follows:

Stage 1

SH: Child Sexual Abuse

OR

KW: "child*" (child, childhood, children) *AND* KW: ["sexual abuse", "sexual assault", "molestation", "incest"]

AND

Stage 2 (Risk factor levels)

SH: Epidemiology *OR* KW: ["epidemiology", "prevalence", "incidence"]

Stage 2 (Risk factor–disease relationships)

SH: Depression, *OR* KW: "depress*" (depression, depressive)

SH: Anxiety Disorders, *OR* SH: [Panic Disorder, Agoraphobia, Obsessive Compulsive Disorder, post-traumatic stress disorder] *OR* KW: ["panic disorder", "agoraphobia", "obsessive compulsive disorder", "OCD", "post-traumatic stress disorder", "PTSD"]

SH: [Substance Related Disorders, Alcoholism] *OR* KW: ["alcohol abuse", "alcohol dependence", "alcohol use", "substance abuse", "substance dependence", "substance use", "drug abuse", "drug dependence", "drug use"]

SH: [Suicide, Suicide Attempted], *OR* KW: ["suicid*" (suicide, suicidal)]

These searches generated a list of over 12 000 studies, of which approximately 4000 were duplicates. The abstracts of the remaining 8000 were examined to isolate potentially appropriate studies. The inclusion criteria were applied and the number of potentially relevant articles was reduced to approximately 1000. Many of the articles excluded at this step were those that either did not measure CSA separately from other types of abuse, or did not include a non-exposed control group.

After the exclusion criteria were applied to the 1000 articles, an initial sample of 460 articles was identified for collection. As the articles were reviewed the reference lists were examined in an attempt to uncover additional studies. The tables of contents of *Child Abuse and Neglect*, the leading journal in the area of child sexual abuse, were searched to locate any articles missed in searches. Experts in the area were contacted for information on unpublished data or for data from countries underrepresented in the usual databases and journals. These experts were located through the membership directory of the International Society for Prevention of Child Abuse and Neglect (ISPCAN), an organization sponsored by UNICEF-NY, Child Protection Division. The Society has over 300 members. All members were contacted via email or fax, with the exception of those from countries where we had adequate published data. A request for data was also put out on a "child maltreatment researchers" list on the Internet which yielded further information. The researchers had access to translation services so articles were not excluded on the basis of language.

The final database consisted of 604 articles. Of these, a further 91 were excluded after collection on the basis of the exclusion criteria defined above (CSA not reported separately from other types of abuse or from abuse as an adult $n = 33$; subjects sampled on the basis of outcome $n = 33$; no non-exposed control group $n = 11$; sample size too small $n = 11$; individual case study data only $n = 3$). In these cases it was not possible to determine from the abstract alone whether the study

should be included. This produced a final set of 513 articles or reports. They were as follows:

- 103 reviews or meta-analyses;
- 55 methodological papers;
- 179 coded studies;
- 48 studies not coded as data had been collected from another paper or report;
- 52 prevalence studies not coded as data on CSA were derived from a secondary source such as official reports or records;
- 13 studies not coded as subjects were gathered from a special sample;
- 48 studies not coded as the outcome data were either not adequately measured or outcome was measured using a continuous as opposed to a categorical measure; and
- 15 studies not coded, as we were unable to obtain copies of the relevant papers or reports.

The 179 coded studies were coded for risk factor levels, risk factor–disease relationships or both.

2.4 Methods for obtaining estimates where more than one data source exists

More than one estimate was available for each subregion. Therefore a meta-analysis was conducted to combine estimates. However, before this was carried out the prevalence estimates were adjusted to remove any differences that can be explained by variations in methodology employed in the studies.

Coding of studies

Prevalence of CSA was obtained from each study included in this analysis. Where possible, an overall prevalence of any CSA was coded as well as a breakdown into the mutually exclusive groups of non-contact, contact and intercourse. A number of methodological characteristics were also coded from each study. These included the type of sample, the representativeness of the sample, age and sex distributions, the method of data collection employed, how childhood was defined, survey response rate, how many questions were used to elicit the presence of CSA and whether restrictions were placed on the definition of abuse.

Correcting for methodological variability within studies

Previous studies have noted that the prevalence of CSA differs depending on a number of methodological factors, including method of data collection, number of questions used to assess CSA, definition of child-

hood and the type of sample assessed (Bolen and Scannapieco 1999; Gorey and Leslie 1997; Haugaard and Emery 1989; Wynkoop et al. 1995). In order to examine the methodological characteristics that influence the variability in prevalence, multiple linear regression analyses were conducted. As it was assumed that the impact of these factors may differ for males and females, separate regression models were fitted for all estimates of prevalence for males ($N = 93$ estimates) and all estimates of prevalence for females ($N = 143$ estimates). Too few data points existed to carry out the regression analyses in each of the levels of exposure. Thus, the independent variable was prevalence of any CSA (coded as either broad or narrow definition).

Model-building steps

1. Each independent variable was screened for adequate cell sizes as well as its relationship with all other independent variables. Cells were collapsed where necessary and choices were made between two variables when they were significantly related (Table 23.1 displays the variables chosen for the regression analysis and their categories).
2. The distribution of the dependent variable was tested for skewness, kurtosis and univariate outliers. Three outliers were removed from the analysis.
3. All variables were tested univariately and then entered together into a single model. A process of backward elimination was employed to remove the least significant variable at each step until all variables left in the final model were statistically significant at the 0.05 level. As shown in Table 23.1 two of the dependent variables entered into the model were "subregion" and "CSA definition". Variability in prevalence across different World Health Organization (WHO) regions and across the broad and narrow definitions of CSA was considered to be important. Thus, these variables were left in the multivariate model even if they were non-significant.

Results

The final model examining the multivariate effects of methodological factors accounted for 27% of the variance in prevalence of CSA in males and 22% of the variance in prevalence of CSA in females (beta weights and significance levels for the statistically significant variables in the multivariate model are shown in Table 23.1). The variables remaining in the "males" model were "subregion" ($F = 0.68$, $df = 3$, $P = 0.5688$), "CSA definition" ($F = 1.98$, $df = 1$, $P = 0.1637$) and "sample type" ($F = 6.13$, $df = 4$, $P = 0.0002$). These results indicate that studies of male college samples and general practitioner (GP) attendees report a significantly higher overall prevalence of CSA than studies of community samples (12% and 12% vs 6%). The variables remaining in the "females" model

Table 23.1 Characteristics of the dependent variables used in the regression analysis

		Variables left in final model for males		*Variables left in final model for females*	
Dependent variable	*Categories (for categorical variables)*	*Beta weights*	*P-value*	*Beta weights*	*P-value*
Subregion	1 = AMR-A	0.00	—	0.00	—
	2 = EUR-A	–1.16	0.4616	–8.18	0.0002*
	3 = WPR-A	0.95	0.6499	1.57	0.5897
	4 = All other subregions	3.63	0.2988	5.75	0.2553
CSA definition	1 = Broad (non-contact, contact or intercourse)	0.0	—	0.00	—
	2 = Narrow (contact or intercourse)	–1.70	0.1637	–4.60	0.0247*
Sample type	1 = Community volunteers	0.00	—	Null	Null
	2 = College students	4.52	0.0126*		
	3 = School students	–1.54	0.1982		
	4 = GP attendees	5.77	0.0145*		
	5 = All other samples	0.69	0.7509		
Number of questions	1 = One	Null	Null	–7.72	0.0003*
	2 = More than one			0.00	—
Definition of childhood	1 = <18 years old	Null	Null	Null	Null
	2 = <16 years old				
	3 = <14 years old				
	4 = Not reported				
Restrictions on CSA definition	1 = None	Null	Null	Null	Null
	2 = Coercion only				
	3 = Age only				
	4 = Age or coercion				
Sample size	Continuous variable	Null	Null	Null	Null
Response rate	Continuous variable	Null	Null	Null	Null
Current age	Continuous variable	Null	Null	Null	Null

Null Non-significant.

— Reference category.

* Significant at the $P < 0.05$ level.

were "subregion" ($F = 7.18$, $df = 3$, $P = 0.0002$), "CSA definition" ($F = 5.17$, $df = 1$, $P = 0.0247$) and "Number of questions" ($F = 13.65$, $df = 1$, $P = 0.0003$). These results indicate reported prevalence is lower among women in some European countries compared to North America (15% vs 22%), among studies that employed a narrow definition of CSA

(19%) than those that employed a broad CSA definition (23%), and those which used only one question to assess the presence of CSA (14%) than those with more than one question (23%).

The finding that CSA definition independently contributed to variance in prevalence for females but not males is unusual. The difference between the broad and narrow prevalence estimates is wholly explained by whether or not non-contact forms of abuse are included in the definition. Therefore, if non-contact abuse is rare as the only form of abuse experienced in males then it is likely that including non-contact abuse in a definition of CSA in males would not substantially alter the overall prevalence. However, the pattern across types of abuse for males does not indicate this to be the case (40% of all CSA in males is non-contact CSA). A more plausible explanation is that the low overall prevalence in males coupled with the smaller number of estimates and their substantial variance contributed to the lack of effect observed for different CSA definitions in males. One might expect that had more estimates been available in males a significant difference may have emerged.

The finding that studies that included more than one question about abuse yielded higher prevalence rates is widely supported in the CSA literature (Fergusson and Mullen 1999; Mullen et al. 2000; Peters et al. 1986; Plunkett and Oates 1990); it is also reported that the higher rates observed in such studies are likely to be more accurate (Bolen and Scannapieco 1999). Within this context, adjusting the prevalence estimates from studies that used only one question to more closely reflect those that used multiple questions (rather than the other way around) is likely to be a more accurate reflection of true prevalence in the population. Once again, the lack of effect in male estimates may be a function of the smaller number of estimates, especially given that the number of questions did significantly predict prevalence in males in the univariate analyses.

More intriguing is the finding that the prevalence in females was lower in EUR-A countries than in North America, from where the majority of the world's prevalence estimates come. Finkelhor (1994) reported on the international epidemiology of CSA and concluded that between-country differences were more likely to be due to methodological differences than reflective of true differences in prevalence. However, the fact that the effect in the present analysis remains after controlling for methodological factors may reflect true differences between these cultural groups. Moreover, in light of the larger number of studies available for the present analysis, it does appear to be true that international differences in prevalence exist. Within this context, it is important to note that there were substantial variations in CSA prevalence within subregions even when controlling for methodological variations, and this is reflected in the modest proportion of explained variance for the final model (22%). Therefore, the lower observed prevalence may, in fact, be a function of a non-examined explanatory variable. Moreover, a substantial number

of subregions were either underrepresented or not represented at all in this analysis, and it is therefore difficult to interpret these findings in a broader international context.

Weighting of prevalence estimates

The results of the above regression analyses were used to weight the overall CSA prevalence estimates for both males and females. Specifically, the unstandardized regression coefficients from the two final models were used to adjust the raw prevalence estimates. This was achieved by subtracting the coefficients from the prevalence for all levels of a variable that were statistically significant (e.g. if the prevalence in males was derived from a college sample the adjusted prevalence would be the raw prevalence minus 4.52 percentage points). The variables of "subregion" and "CSA definition" did not contribute to this adjustment because they do not reflect differences in methodological quality. This produced the desired effect of controlling for any influence these factors had on other variables while at the same time keeping any variance in prevalence due to these factors.

It is worth noting that implicit in this process of adjustment was the assumption that estimates from the group used as the reference group more closely reflected the true population prevalence. Thus, estimates for males from college and GP samples were adjusted to more closely reflect the prevalence observed in community samples. For females, prevalence estimates were adjusted to more closely reflect those observed in studies where more than one question to define CSA was asked.

Extrapolation across levels of exposure

Once weighted prevalence estimates were obtained for each study they were divided into the mutually exclusive groups of non-contact, contact and intercourse using the following method. The relationship between each level of exposure, expressed as a proportion of the overall prevalence, was calculated for all studies that reported prevalence of CSA by type of exposure. When a study reported prevalence based on a broad definition, this was apportioned into non-contact CSA, contact CSA and intercourse (eight studies for males, 12 studies for females). When a study reported prevalence based on a narrow definition this was apportioned into contact CSA and intercourse (12 studies for males, 20 studies for females). These proportions were then applied to those studies only reporting an overall prevalence to obtain an estimate of prevalence for each level of exposure in each study. A small number of studies reported proportions in the three levels of exposure that were opposite in direction to all other studies, and these were excluded from the present calculations. Sensitivity analyses were carried out to assess the impact of these two apportioning fractions (e.g. including all studies, excluding studies where the proportion was opposite to all other studies) on the prevalence in each of the three levels of exposure. The results of these

Table 23.2 The impact of different apportioning fractions on the prevalence of CSA in the three levels of exposure

	Method 1		*Method 2*	
	Apportioning fraction	*Prevalence*	*Apportioning fraction*	*Prevalence*
Males				
Non-contact CSA	0.297	2.6	0.387	3.1
Contact CSA	0.448	4.0	0.378	3.7
Intercourse	0.255	2.0	0.235	1.9
Females				
Non-contact CSA	0.279	6.7	0.291	6.8
Contact CSA	0.500	12.7	0.512	13.2
Intercourse	0.221	5.8	0.197	5.3

Method 1: Proportions derived from all studies (n = 12 for males, n = 20 for females).

Method 2: Proportions derived from all studies (n = 10 for males, n = 17 for females) except those in opposite direction to expected proportions (this method was used in the final calculations).

analyses are shown in Table 23.2. The overall impact of these excluded studies on each level of exposure was minimal. They were therefore not included in the calculation of this apportioning fraction.

The observed relationship between non-contact, contact and intercourse for both males and females indicated that intercourse was the least common form of abuse, a finding that has been widely reported (Fergusson and Mullen 1999; Mullen et al. 2000).

Extrapolation of combined estimates across age

Exposure to CSA, by definition, occurs in childhood. Thus, during childhood the prevalence of CSA represents current exposure, and reflects cumulative exposure to CSA from birth until current age. The prevalence of CSA in persons aged ≥18 years necessarily represents past exposure and reflects cumulative exposure from birth until the age of 18 years. Therefore prevalence of CSA will vary across age groups from 0–17 years dependent on both the age at which exposure to CSA usually begins (age at onset) and the duration of CSA throughout childhood. Because exposure is fixed at the age of 18 years, the prevalence of CSA across age groups after 18 years will vary only if the prevalence of CSA is changing over time. For example, if birth cohorts between 1956 and 1970 (current age 30–44 years) were more likely to be exposed to CSA in childhood than birth cohorts before 1956 (current age ≥45 years) then the prevalence of CSA for these age groups would differ.

The age groups required for reporting by WHO therefore necessitated three separate steps for calculation of exposure across age groups. The first step estimated prevalence in the 0–4-, 5–14-, and 15–17-year age groups. The second step estimated prevalence in the 18–29-, 30–44-, 45–59-, 60–69-, 70–79- and ≥80-year age groups. The third step esti-

mated a combined prevalence in the 15–17- and 18–29-year age groups to obtain prevalence for the age group 15–29 years.

Step 1: Estimating prevalence in persons aged 0–17 years

No data exist on the prevalence of CSA in different childhood age categories. Age is usually examined in terms of the age at first onset of the abuse or the duration of abuse. In order to estimate prevalence of CSA across different childhood age categories, all studies that contained information about onset or duration were examined. The main difficulty arose from the use of disparate age categories that did not always conform to those required for the WHO estimates. The following steps were carried out to examine this issue.

Age at onset of abuse. A total of 22 studies presented data on age at onset of abuse. Although categorization of age varied considerably across studies, onset was consistently more prevalent in the 5–14-year age group. This pattern was the same for both males and females. It is noted that the validity of self-reported abuse with an onset before the age of 5 years should be considered, at best, speculative. Age at onset for different levels of exposure and between different subregions could not be examined with the available data. Therefore, the following procedures were applied equally to males and females, across all levels of exposure and across all subregions.

- Two studies presented data in the 0–4-year age group. These studies indicate that approximately 6.5% of abuse begins in this age group. If the age band is extended to include 0–5 year olds (six studies in total) then the equivalent estimate is 9.7%. The midpoint of these two values indicates that approximately 8.1 % of all cases of CSA have their onset before the age of 5 years.
- Three studies presented data for those aged ≥15 years. These studies show that approximately 19.7% of abuse begins in this age group. If the age band is restricted to those aged ≥16 years (six studies in total) then the equivalent estimate is 18.8%. The midpoint of these two values indicates that approximately 19.3% of all cases of CSA have their onset after the age of 14 years.
- The middle age group was derived from the above calculations to give a value of 72.6% (100%–8.1%–19.3%), which is consistent with the overall pattern observed.

Duration of abuse. If prevalence of CSA in different childhood age groups is based only on the age at onset, then abuse that begins in one age group and continues into the next will not be counted. One way to account for this is to adjust for duration, that is, to include a proportion of cases with long duration in more than one age category.

Finkelhor (1979) estimated that 16% of those who experience CSA experience it on more than one occasion and for a duration of more than one week. Bagley and Mallick (2000) estimated this figure at approximately 20%. However, of most interest to the current analysis were the studies that reported the number of CSA cases where duration was at least one year. Two studies with such estimates were found (Collings 1997; Risin and Koss 1987), and they placed the prevalence of CSA cases with a duration of more than one year at 6.3% and 12% of all CSA cases. The midpoint of these values indicated that approximately 9.2% or one in ten cases of CSA would continue for more than one year (Risin and Koss 1987). Finkelhor (1979) further examined duration by level of exposure and indicated that non-contact abuse was the least likely to continue for more than one year while intercourse was the most likely (4.3% for non-contact, 5.8% for contact and 8.8% for intercourse, representing respective proportions of 0.68, 0.92 and 1.40, compared to the overall figure of 6.3%).

Combining onset and duration. Weights derived from age at abuse (0.081, 0.726 and 0.193) were combined with information about duration in the following way: 9.2% of cases in the 0–4-year age group were carried over to the 5–14-year age group using the above proportions for each level of abuse (0.68, 0.92 and 1.40). The same calculations were made in carrying over cases from the 5–14-year age group to the 15–18-year age group.

Step 2: Estimating prevalence in persons aged >18 years

For the reasons outlined earlier, prevalence of CSA in adults will only vary if the prevalence of CSA is changing over time. Several reviews have attempted to examine cohort effects in order to address this issue (Bagley 1990, 1995; Bagley and Ramsay 1985; Bickerton et al. 1991; Feldman et al. 1991; Fergusson et al. 2000). Three of these reviews concluded that the prevalence of CSA could be increasing over time while three reviews also concluded that there is no evidence to support a change in prevalence over time. This pattern is not explained by the publication dates of these reviews and many of the authors also pointed out that it is difficult to interpret these results without reference to a potential reporting phenomenon. That is, women in older age groups may be less willing or less able to report experiences of sexual abuse in childhood.

This issue was also examined empirically in the current data set. Each estimate for males and females was assigned a year of birth, calculated by subtracting the mean age of the sample from the year of publication (or where available the year the survey was conducted). This variable was then examined in a linear regression to determine whether year of birth explained any of the variance in prevalence. The continuous year of birth variable was also converted to a categorical variable with birth cohort defined according to the current age categories provided by

WHO. Birth cohort did not explain any variance in prevalence estimates for either males or females.

Given both these findings indicated no clear trend in prevalence of CSA over time, it was decided that estimates of prevalence would be combined across adult age groups. This had the effect of providing more stable estimates, particularly from underrepresented subregions.

Step 3: Combining estimates for 15–17 and 18–29 year olds

In order to combine estimates of prevalence in these two groups, it was necessary to determine the proportion of the population that fell into these two age groups for each subregion. This was calculated for each subregion represented in the data and was done separately for males and females. The data were obtained from the estimates provided by WHO and were based on population figures for the year 2000. As these data were only available for 15–19 year olds, as opposed to 15–17 year olds, population proportions were calculated using this age group.

Combining estimates within countries

Once estimates of prevalence were apportioned into the three levels of exposure and across the eight separate age groups (yielding 24 separate prevalence estimates for each study), prevalences were combined within countries. The prevalence estimates were combined using meta-analysis with STATA Intercooled 7. For ease of calculation the "meta" macro was used (Sharp and Sterne 1997). Heterogeneity between studies within each country was tested using the chi-squared statistic. When only two or three studies were available for combination, the between-study variance was estimated with poor precision (Cooper and Hedges 1994). Countries with less than five estimates were combined using a fixed-effects model, and countries with five or more estimates, and statistical heterogeneity, were combined using a random-effects model.

Combining estimates within subregions

In order to combine prevalence estimates between countries within each subregion, each country estimate was weighted for the population of that country. This meant that prevalence estimates from countries with large populations were given more weight in the final estimates. A combined estimate was obtained for each subregion by calculating a mean for each level of exposure in each age group for males and females.

2.5 Methods for obtaining estimates where no data existed

Extrapolation across subregions was one of the most difficult issues encountered in the construction of the prevalence estimates and arguably represents one of the greatest threats to the validity of the estimates in subregions where no data were available. For two out of the 14 subregions no prevalence data were found (EMR-B and EMR-D). These sub-

regions represent vastly different cultural, socioeconomic and geographic populations. In the absence of data it is impossible to speculate on how these differences might have impacted on the prevalence of CSA. No estimates were obtained for any countries in the Middle East. Therefore the estimate that was used comes from Turkey (EUR-B), which was considered the most appropriate in the absence of any other data. This extrapolation should be considered conjectural and the resultant estimates of prevalence in these subregions should be quoted with caution. It should also be noted that there was an uneven distribution of estimates in the remaining 12 subregions with a small number of countries making up a large proportion of the total number of estimates.

2.6 Description of studies, including methodological qualities

Prevalence studies included in the analysis are presented in Table 23.3. They are presented in three levels according to the type of sample used and the representativeness of this sample. The levels are defined as follows.

Level A: Representative community samples—samples of adolescents or adults where the article explicitly stated that the sample was representative of the population from which it was drawn. In general this was achieved through the use of complex sampling procedures or weighting.

Level B: Other community samples (representativeness not known)—samples of adolescents or adults where it was not known whether the samples were truly representative of the population from which they were drawn.

Level C: Community subgroups and convenience samples—samples of adolescents or adults drawn from a subgroup within the community based on factors such as ethnicity, educational or socioeconomic status.

2.7 Characteristics of excluded studies

There were 48 studies that were not coded due to their outcome measurement methods. Where the outcome was measured using a continuous measure for which there were established diagnostic cut-off points, authors were contacted and 2×2 tables were requested. The majority of authors responded and supplied data and these studies were included in the 179 coded studies. The 48 remaining were either not coded because authors could not supply data or authors were not contacted because the measure used could not be mapped to diagnostic criteria.

The characteristics of the 15 studies that could not be obtained are presented in Table 23.4. Inter-library loans were requested where items were only available interstate or overseas but several articles had not arrived at the time this chapter was being compiled.

Table 23.3 Characteristics of studies included in prevalence analysis

	Sample				*CSA data collection*		
Study authors	*Type*	*Number*	*% female*	*Mean age (years)*	*Method*	*No. of questions*	*Childhood definition*
AFR-D (Cameroon $n = 1$)							
Level C: Community subgroups and convenience samples							
Menick and Ngoh (1998)	School students	1 688	54	12	Self-report	>1	<15
AFR-E (Ethiopia $n = 1$, South Africa $n = 3$)							
Level B: Other community samples (representativeness not known)							
Mulugeta et al. (1998)	School students	719	100	16	Self-report	>1	<18
Level C: Community subgroups and convenience samples							
Collings (1997)	College students	640	100	20	Self-report	>1	<18
Collings (1991)	College students	284	0	20	Postal SR	>1	<18
Madu and Peltzer (2000)	School students	414	52	19	Self-report	>1	<18
AMR-A (Canada $n = 7$, USA $n = 72$)							
Level A: Representative community samples							
Davidson et al. (1991)	Adults	2 985	54	42	Face-to-face	>1	<16
Vogeltanz et al. (1999)	Adults	733	100	—	Face-to-face	>1	<18
Molnar et al. (2001)	Adults	5 877	50	35	Self-report	>1	<18
Siegel et al. (1987)	Adults	3 132	53	42	Face-to-face	>1	<16
Finkelhor et al. (1990)	Adults	2 626	56	—	Telephone	>1	<18
MacMillan et al. (1997)	Adults	9 953	51	40	Face-to-face	>1	<18
Wyatt (1985)	Adults	248	100	27	Face-to-face	>1	<18
Wyatt et al. (1999)	Adults	338	100	30	Face-to-face	>1	<18
Saunders et al. (1999)	Adults	4 008	100	45	Telephone	>1	<18
Brown et al. (1999)	Adults	639	48	18	Face-to-face	1	<18
Finkelhor and Dziuba-Leatherman (1994)	Adolescents	2 072	48	13	Telephone	>1	<17
Boney-McCoy and Finkelhor (1996)	Adolescents	1 457	47	13	Telephone	>1	<16
Kilpatrick et al. (2000)	Adolescents	4 023	49	15	Telephone	>1	<17
Risin and Koss (1987)	College students	2 972	0	21	Self-report	>1	<14
Fromuth and Burkhart (1989)	College students	253	0	20	Self-report	>1	<17
Harrison et al. (1997)	School students	122 824	51	15	Self-report	>1	<18
Bagley et al. (1995)	School students	2 112	49	15	Self-report	>1	<18
Blum et al. (1988)	School students	36 283	49	15	Self-report	1	<18
American School Health Association (1989)	School students	3 490	50	13	Self-report	1	<18
Nelson et al. (1994)	School students	2 332	51	16	Self-report	>1	<18
Hernandez (1992)	School students	3 179	48.3	14	Self-report	>1	<15
Bensley et al. (1999)	School students	4 790	48	16	Self-report	1	<18

Restriction on CSA definition	Prevalence in males (%)					Prevalence in females (%)				
	Any broad	*Any narrow*	*Non-contact only*	*Contact only*	*Intercourse*	*Any broad*	*Any narrow*	*Non-contact only*	*Contact only*	*Intercourse*
No	9.6	—	—	—	—	21.3	—	—	—	—
Yes	—	—	—	—	—	—	—	—	—	5.2
Yes	—	—	—	—	—	—	34.8	—	29.0	5.8
No	28.9	9.2	19.7	4.3	4.9	—	—	—	—	—
Yes	—	56.0[a]	—	42.5	13.5	—	53.2	—	35.6	17.6
No	—	—	—	—	1.1[b]	—	—	—	—	1.1[b]
Yes	—	—	—	—	—	24.0	17.7	6.3	—	—
No	—	2.5	—	1.9	0.9	—	13.5	—	8.5	5.0
No	—	3.8	—	—	—	—	6.8	—	—	—
No	16.0	—	—	—	—	27.0	—	—	—	—
Yes	6.7	—	—	—	—	19.5	—	—	—	—
Yes	—	—	—	—	—	62.0[a]	46.0	16.0	—	—
Yes	—	—	—	—	—	—	34.0	—	—	—
No	—	—	—	—	—	—	—	—	—	8.5
Yes	—	3.4[b]	—	—	—	—	3.4[b]	—	—	—
No	5.9	—	—	—	0.0	15.3	—	—	—	1.3
No	3.1	—	—	—	—	9.7	—	—	—	—
Yes	—	8.0[b]	—	—	—	—	8.0[b]	—	—	—
No	7.3	4.7	2.5	2.5	2.2	—	—	—	—	—
Yes	15.0	—	—	—	—	—	—	—	—	—
Yes	—	4.3	—	—	—	—	11.5	—	—	—
No	—	9.8	—	—	—	—	23.6	—	—	—
Yes	—	2.0	—	—	—	—	14.0	—	—	—
Yes	—	—	—	—	6.2	—	—	—	—	18.5
No	8.1	—	—	—	—	33.1	—	—	—	—
Yes	—	10.0[b]	—	—	—	—	10.0[b]	—	—	—
Yes	—	5.0	—	—	—	—	23.8	—	—	—

continued

Table 23.3 Characteristics of studies included in prevalence analysis (*continued*)

	Sample				*CSA data collection*		
Study authors	*Type*	*Number*	*% female*	*Mean age (years)*	*Method*	*No. of questions*	*Childhood definition*
Level B: Other community samples (representativeness not known)							
L. George and I. Winfield-Laird, unpublished document, 1986	Adults	1157	100	41	Face-to-face	1	<16
Keckley Market Research, unpublished document, 1983	Adults	603	—	—	Telephone	1	<18
Murphy (1997)	Adults	818	51	—	Telephone	1	<18
Wolf (1992)	Adults	637	56	—	Telephone	1	<16
Essock-Vitale and McGuire (1985)	Adults	300	100	40	Face-to-face	1	<18
Saunders et al. (1992)	Adults	391	100	42	Face-to-face	1	<18
Bagley and Ramsay (1985)	Adults	377	100	40	Face-to-face	>1	<17
Bagley (1991)	Adults	750	100	23	Face-to-face	>1	<17
Kercher and McShane (1984)	Adults	1054	56	—	Postal SR	1	<18
Russell (1983)	Adults	930	100	—	Face-to-face	>1	<18
Bagley et al. (1994)	Adults	750	0	23	Self-report	>1	<17
Roosa et al. (1998)	Adults	2003	100	20	Self-report	>1	<18
Bagley (1995)	Adults	1833	56	—	Self-report	>1	<17
Watts and Ellis (1993)	School students	670	100	15	Self-report	>1	<18
Erickson and Rapkin (1991)	School students	1197	50	15	Self-report	>1	<18
Lodico et al. (1996)	School students	6224	48	16	Self-report	>1	<18
Level C: Community subgroups and convenience samples							
Kendler et al. (2000)	Adults twins	1411	100	40	Postal SR	>1	<16
Silverman et al. (1996)	Adults (long study)	375	50	21	Face-to-face	>1	<18
White and Strange (1993)	College students	131	100	20	Postal SR	>1	<17
Peters and Range (1995)	College students	266	51	—	Self-report	>1	<12
Thakkar et al. (2000)	College students	707	100	19	Self-report	>1	<15
Schaaf and McCanne (1998)	College students	238	100	19	Self-report	>1	<15
Finkelhor (1979)	College students	796	67	21	Self-report	>1	<17
deLahunta (1996)	College students	787	38	—	Postal SR	—	—
Arroyo (1997)	College students	221	100	25	Face-to-face	>1	<18
Briere and Runtz (1988)	College students	278	100	20	Self-report	>1	<15
Wellman (1993)	College students	824	80	20	Self-report	>1	<18
Edwards and Alexander (1992)	College students	103	100	23	Self-report	>1	<18
Fritz et al. (1981)	College students	952	57	—	Self-report	>1	<14

	Prevalence in males (%)					Prevalence in females (%)				
Restriction on CSA definition	*Any broad*	*Any narrow*	*Non-contact only*	*Contact only*	*Intercourse*	*Any broad*	*Any narrow*	*Non-contact only*	*Contact only*	*Intercourse*
—	—	—	—	—	—	—	2.0	—	—	—
Yes	7.0	—	—	—	—	11.0	—	—	—	—
Yes	—	3.0	—	—	—	—	13.0	—	—	—
No	9.0	—	—	—	—	27.0	—	—	—	—
No	—	—	—	—	—	—	17.3	—	—	—
No	—	—	—	—	—	33.5	24.6	9.0	14.6	10.0
Yes	—	—	—	—	—	—	21.7	—	—	—
No	—	—	—	—	—	—	32.0	—	—	—
Yes	3.0	—	—	—	—	11.6	—	—	—	—
Yes	—	—	—	—	—	54.0	38.0	16.0	—	—
Yes	—	15.5	—	—	—	—	—	—	—	—
No	—	—	—	—	—	—	39.0	—	16.0	23.0
Yes	—	8.2	—	—	—	—	17.6	—	—	—
No	—	—	—	—	—	—	14.5	—	—	—
No	—	15.0[b]	—	—	—	—	15.0[b]	—	—	—
Yes	4.2	—	—	—	—	16.5	—	—	—	—
Yes	—	—	—	—	—	30.3	22.5	7.8	14.1	8.4
Yes	—	1.1[c]	—	—	—	—	12.3[c]	—	—	—
Yes	—	—	—	—	—	33.5	—	—	—	—
Yes	19.1	12.2	6.9	—	—	31.9	19.3	12.6	—	—
Yes	—	—	—	—	—	—	13.5	—	—	—
No	—	—	—	—	—	12.2	—	—	—	—
Yes	8.6	—	—	—	—	19.2	—	—	—	—
No	—	3.5[c]	—	—	—	—	10.8[c]	—	—	—
No	—	—	—	—	—	31.2	—	—	—	—
Yes	—	—	—	—	—	—	14.8	—	—	—
No	23.0	13.4	9.6	—	—	15.0	5.6	9.4	—	—
Yes	—	—	—	—	—	—	43.6	—	—	—
Yes	—	4.8	—	—	—	—	7.7	—	—	—

continued

Table 23.3 Characteristics of studies included in prevalence analysis (*continued*)

	Sample				*CSA data collection*		
Study authors	*Type*	*Number*	*% female*	*Mean age (years)*	*Method*	*No. of questions*	*Childhood definition*
Haugaard and Emery (1989)	College students	1089	61	19	Self-report	>1	<17
Duane et al. (1997)	College students	958	61	—	Self-report	>1	<13
Stepakoff (1998)	College students	393	100	20	Postal SR	>1	<17
Boudewyn and Liem (1995)	College students	438	61	25	Self-report	>1	<14
Bryant and Range (1997)	College students	486	74	24	Self-report	>1	<18
Bolstad and Zinbarg (1997)	College students	117	100	26	Self-report	>1	<15
Fromuth (1986)	College students	482	100	20	Self-report	>1	<16
Sedney and Brooks (1984)	College students	301	100	19	Self-report	—	—
Hibbard et al. (1988)	School students	712	50	14	Self-report	1	<18
Riggs et al. (1990)	School students	600	52	16	Postal SR	1	<18
Greenwood et al. (1990)	GP attendees	100	59	42	Face-to-face	>1	—
Walch and Broadhead (1992)	GP attendees	405	100	29	Self-report	—	<18
Kellogg and Hoffman (1995)	GP attendees	142	60	20	Self-report	1	<18
Gould et al. (1994)	GP attendees	292	71	48	Self-report	>1	<17
Felitti et al. (1998)	GP attendees	9508	54	56	Postal SR	>1	—
Kilpatrick (1986)	Misc. comm. groups	501	100	28	Self-report	>1	<15
DiVasto et al. (1984)	Misc. comm. groups	500	100	27	Self-report	1	<13
Moeller et al. (1993)	Clinic sample	668	100	34	Postal SR	—	<18
Bayatpour et al. (1992)	Clinic sample	352	100	15	Face-to-face	1	<18
Descamps et al. (2000)	Lesbian women	1925	100	35	Postal SR	—	—
Blum et al. (1992)	American Indian and Alaska Native youth	13454	51	15	Self-report	—	<18
Robin et al. (1997)	American Indians	375	58	37	Face-to-face	>1	<16
Greenwald and Leitenberg (1990)	Nurses	1500	100	—	Self-report	—	<16
Hall et al. (1993)	Low income women	203	100	27	Face-to-face	>1	<18
Zuravin and Fontanella (1999)	Low income women	513	100	30	Face-to-face	>1	<14
Wingood and DiClemente (1997)	African-American women	165	100	24	Face-to-face	1	<16
Romero et al. (1999)	Latina women	300	100	32	Face-to-face	>1	<18
AMR-B (Brazil $n = 2$, Costa Rica $n = 1$, Dominican Republic $n = 1$, El Salvador $n = 1$, Mexico $n = 1$)							
Level A: Representative community samples							
Ramos-Lira et al. (1998)	School students	61779	47	14	Self-report	>1	<18

	Prevalence in males (%)					Prevalence in females (%)				
Restriction on CSA definition	*Any broad*	*Any narrow*	*Non-contact only*	*Contact only*	*Intercourse*	*Any broad*	*Any narrow*	*Non-contact only*	*Contact only*	*Intercourse*
Yes	5.0	—	—	—	—	11.9	—	—	—	—
No	—	4.0	—	—	—	—	4.3	—	—	—
Yes	—	—	—	—	—	—	14.9	—	10.1	4.8
Yes	—	16.2	—	—	—	—	23.8	—	—	—
No	28.2[b]	—	—	—	—	28.2[b]	—	—	—	—
Yes	—	—	—	—	—	31.6	—	—	—	—
Yes	—	—	—	—	—	22.0	—	—	—	—
—	—	—	—	—	—	—	16.9[c]	—	—	—
No	—	8.0[b,c]	—	—	—	—	8.0[b,c]	—	—	—
No	8.1[b]	—	—	—	—	8.1[b]	—	—	—	—
No	0.0	—	—	—	—	16.9	—	—	—	—
Yes	—	—	—	—	—	35.6	—	—	—	6.1
No	—	16.0	—	—	—	—	39.0	—	—	—
No	—	12.0	—	—	—	—	30.0	—	—	—
Yes	—	22.0[b]	—	15.1[b]	6.9[b]	—	22.0[b]	—	15.1[b]	6.9[b]
No	—	—	—	—	—	55.0	—	—	—	1.8
No	—	—	—	—	—	—	—	—	—	1.8
Yes	—	—	—	—	—	19.8	—	—	—	—
No	—	—	—	—	—	14.8	—	—	—	—
—	—	—	—	—	—	—	28.7	—	—	—
Yes	—	10.0	—	—	—	—	21.6	—	—	—
Yes	14.0	—	—	—	—	49.0	—	—	—	—
Yes	—	—	—	—	—	3.6	—	—	—	0.7
No	—	—	—	—	—	—	22.0	—	—	—
No	—	—	—	—	—	—	20.7	—	8.0	12.7
No	—	—	—	—	—	—	—	—	—	12.7
No	—	—	—	—	—	33.0	—	—	—	8.6
Yes	—	4.3	—	—	—	—	4.3	—	—	—

continued

Table 23.3 Characteristics of studies included in prevalence analysis (*continued*)

	Sample				*CSA data collection*		
Study authors	*Type*	*Number*	*% female*	*Mean age (years)*	*Method*	*No. of questions*	*Childhood definition*
Level B: Other community samples (representativeness not known)							
WHO (2001)	Adults	1172	100	32	Face-to-face	>1	<15
WHO (2001)	Adults	1473	100	32	Face-to-face	>1	<15
Z.A. Ruiz et al., unpublished document, 1986	College students	893	54	23	Self-report	—	—
Level C: Community subgroups and convenience samples							
Barthauer and Leventhal (1999)	Adults	83	100	35	Face-to-face	>1	<18
Krugman et al. (1992)	College students	497	45	20	Self-report	>1	<19
AMR-D (Nicaragua $n = 1$, Peru $n = 2$)							
Level A: Representative community samples							
Olsson et al. (2000)	Adults	336	60	34	Self-report	4	<19
Level B: Other community samples (representativeness not known)							
WHO (2001)	Adults	1415	100	32	Face-to-face	>1	<15
WHO (2001)	Adults	1847	100	32	Face-to-face	>1	<15
EMR-B (no estimates available)							
EMR-D (no estimates available)							
EUR-A (Austria $n = 1$, Belgium $n = 1$, Czech Republic $n = 1$, Denmark $n = 1$, Finland $n = 1$, France $n = 3$, Germany $n = 3$, Greece $n = 1$, Ireland $n = 1$, Israel $n = 1$, Netherlands $n = 1$, Norway $n = 4$, Spain $n = 2$, Sweden $n = 4$, Switzerland $n = 2$, United Kingdom $n = 9$)							
Level A: Representative community samples							
Vandewege et al. (1988)	Adults	956	100	35	Face-to-face	—	—
Cawson et al. (2000)	Adults	2869	57	21	Computer	—	<13
Baker and Duncan (1985)	Adults	2019	52	40	Face-to-face	>1	<16
Edgardh and Ormstad (2000)	Adults	1943	58	17	Self-report	>1	<17
Ernst et al. (1993)	Adults	421	47	28	Face-to-face	1	<16
Halperin et al. (1996)	Adults	1116	51	15	Self-report	>1	<17
Spak et al. (1998)	Adults	316	100	—	Face-to-face	1	<18
Lopez et al. (1995)	Adults	1821	47	39	Face-to-face	>1	<17
Garnefski and Arends (1998)	Adults	13894	50	15	Self-report	>1	<19
Weiss and Zverina (1997)	Adults	1719	50	38	Postal SR	>1	<15
Bouhet et al. (1992)	Adults	1511	51	39	Postal SR	>1	<18
Hill et al. (2000)	Adults	862	100	31	Postal SR	>1	<16
Bendixen et al. (1994)	College students	996	51	23	Self-report	>1	—
Choquet et al. (1997)	School students	8140	51	16	Self-report	>1	<16
Sariola and Uutela (1994)	School students	6913	52	16	Self-report	>1	<15

	Prevalence in males (%)					Prevalence in females (%)				
Restriction on CSA definition	*Any broad*	*Any narrow*	*Non-contact only*	*Contact only*	*Intercourse*	*Any broad*	*Any narrow*	*Non-contact only*	*Contact only*	*Intercourse*
—	—	—	—	—	—	7.8	—	—	—	—
—	—	—	—	—	—	5.8	—	—	—	—
—	33.0[b]	—	—	—	—	33.0[b]	—	—	—	—
No	—	—	—	—	—	17.0	—	—	—	9.6
No	—	12.8	—	—	—	—	32.2	—	—	—
Yes	20.0	—	—	—	—	26.0	—	—	—	—
—	—	—	—	—	—	19.5	—	—	—	—
—	—	—	—	—	—	7.9	—	—	—	—
—	—	—	—	—	—	19.0	—	—	—	—
Yes	—	11.0	—	—	—	—	21.0	—	—	—
No	8.0	—	—	—	0.7	12.0	—	—	—	0.8
Yes	3.1	—	—	—	—	11.2	—	—	—	—
Yes	1.8	—	—	—	—	4.9	—	—	—	—
No	10.9	3.3	7.7	2.2	1.1	33.8	20.4	13.4	14.8	5.6
No	—	—	—	—	—	9.8	—	—	—	—
—	14.0	12.0	2.0	8.2	3.8	27.0	19.7	7.3	12.7	7.0
Yes	—	2.2	—	—	—	—	8.2	—	—	—
No	—	4.6	—	4.3	0.3	—	8.4	—	7.5	0.9
No	4.6	3.1	1.4	—	—	7.8	5.2	2.6	—	—
No	—	—	—	—	—	—	17.5	—	11.9	5.6
Yes	3.5	—	—	—	—	19.4	—	—	—	—
Yes	—	—	—	—	0.9	—	—	—	—	0.7
Yes	3.3	—	—	—	—	7.6	—	—	—	—

continued

Table 23.3 Characteristics of studies included in prevalence analysis (*continued*)

	Sample				*CSA data collection*		
Study authors	*Type*	*Number*	*% female*	*Mean age (years)*	*Method*	*No. of questions*	*Childhood definition*
Level B: Other community samples (representativeness not known)							
Leth (2001)	Adults	1 235	54	—	Postal SR	—	<18
H. Holter, unpublished document, 1990	Adults	1 017	—	—	Postal SR	1	—
Rönström (1985)	Adults	938	—	—	Self-report	—	—
Schei (1990)	Adults	118	100	33	Face-to-face	1	—
J. Kinzl and W. Biebl, unpublished data	College students	1 125	—	—	Self-report	—	—
Agathonos et al. (1992)	College students	746	—	—	Self-report	>1	—
de Paul et al. (1995)	College students	403	74	21	Postal SR	>1	<13
Lazartigues et al. (1989)	College students	963	58	20	Postal SR	>1	<16
Schoetensack et al. (1992)	College students	1 841	48	21	Self-report	>1	—
Kelly et al. (1991)	College students	1 244	62	19	Self-report	>1	<18
Pederson and Aas (1995)	School students	465	54	19	Postal SR	>1	<13
Schein et al. (2000)	GP attendees	1 005	65	36	Self-report	>1	<18
Level C: Community subgroups and convenience samples							
Market Research Bureau of Ireland (1987)	Adults	500	—	—	Self-report	>1	<18
Richter-Appelt and Tiefensee (1996)	College students	1 068	42	24	Self-report	—	<12
Raupp and Eggers (1993)	College students	1 009	50	—	Self-report	—	<18
Bickerton et al. (1991)	GP attendees	1 232	100	—	Face-to-face	>1	—
Coxell et al. (1999)	GP attendees	2 474	0	46	Computer	>1	<16
Palmer et al. (1994)	GP attendees	115	0	32	Self-report	>1	<16
Risberg et al. (1999)	GP attendees	175	100	41	Postal SR	1	<18
Palmer et al. (1993)	GP attendees	120	100	30	Face-to-face	>1	<16
Brown and Harris (1993)	Low income women	404	100	—	Face-to-face	>1	<17
EUR-B (Turkey $n = 1$)							
Level C: Community subgroups and convenience samples							
Elal et al. (2000)	College students	1 597	62	—	Self-report	>1	<18
EUR-C (Russia $n = 1$)							
Level C: Community subgroups and convenience samples							
N. Lvoff and V. Lvoff, unpublished document, 1998	College students	723	50	—	Self-report	—	<18

	Prevalence in males (%)					Prevalence in females (%)				
Restriction on CSA definition	*Any broad*	*Any narrow*	*Non-contact only*	*Contact only*	*Intercourse*	*Any broad*	*Any narrow*	*Non-contact only*	*Contact only*	*Intercourse*
Yes	7.0	6.5	0.4	2.2	4.3	14.0	12.1	2.0	5.2	6.9
No	—	9.0	—	—	—	—	19.0	—	—	—
—	3.0	—	—	—	—	11.0	—	—	—	—
No	—	—	—	—	—	14.0	—	—	—	—
—	19.0	—	—	—	—	36.0	—	—	—	—
No	6.0	—	—	—	—	16.0	—	—	—	—
No	3.9	—	—	—	—	6.4	—	—	—	—
Yes	3.4	—	—	—	—	10.2	—	—	—	—
Yes	5.8	3.9	1.9	3.0	0.9	16.1	10.8	5.3	8.9	1.9
No	27.0	11.0	16.0	—	—	59.0[a]	27.0	32.0	—	—
Yes	0.5	0.0	0.5	—	—	6.8	6.0	0.8	—	—
Yes	15.7	—	—	—	—	30.7	—	—	—	—
—	5.0	—	—	—	—	7.0	—	—	—	—
No	4.0	—	—	—	—	23.0	—	—	—	—
No	6.2	—	2.4	2.3	1.5	25.2	—	11.2	11.7	2.3
Yes	—	—	—	—	—	—	3.5	—	—	—
Yes	—	13.0	—	—	—	—	—	—	—	—
Yes	13.9	—	—	—	4.3	—	—	—	—	—
No	—	—	—	—	—	6.8	5.7	1.1	—	—
Yes	—	—	—	—	—	33.3	20.8	12.5	15.8	5.0
Yes	—	—	—	—	—	—	6.9	—	—	—
Yes	16.0	—	—	—	—	28.0	—	—	—	—
—	9.0	—	—	—	—	27.0	—	—	—	—

continued

Table 23.3 Characteristics of studies included in prevalence analysis (*continued*)

	Sample				*CSA data collection*		
Study authors	*Type*	*Number*	*% female*	*Mean age (years)*	*Method*	*No. of questions*	*Childhood definition*
SEAR-B (Indonesia $n = 1$, Sri Lanka $n = 1$, Thailand $n = 2$)							
Level B: Other community samples (representativeness not known)							
WHO (2001)	Adults	1536	100	32	Face-to-face	>1	<15
WHO (2001)	Adults	1282	100	32	Face-to-face	>1	<15
WHO (2001)	Adults	765	100	32	Face-to-face	>1	<15
Level C: Community subgroups and convenience samples							
Miles (2000)	School students	145	43	15	—	1	<18
SEAR-D (India $n = 3$)							
Level C: Community subgroups and convenience samples							
Interventions for support (2001)	School students	198	75	15	Self-report	—	<18
Interventions for support (2001)	School students	426	100	15	Self-report	—	<18
Castelino (1985)	College students	133	50	25	Self-report	—	<13
WPR-A (Australia $n = 8$, New Zealand $n = 4$)							
Level A: Representative community samples							
Anderson et al. (1993)	Adults	497	100	42	Face-to-face	>1	<16
Mullen et al. (1988)	Adults	314	100	—	Face-to-face	>1	<13
Goldman and Goldman (1988)	College students	991	61	22	Self-report	>1	<16
Mazza et al. (1996)	GP attendees	2181	100	—	Self-report	>1	<16
Level B: Other community samples (representativeness not known)							
Fleming (1997)	Adults	710	100	40	Postal SR	>1	<16
Martin et al. (1993)	Adults	1376	100	—	Postal SR	>1	<16
Level C: Community subgroups and convenience samples							
Dinwiddie et al. (2000)	Adults	5946	65	43	Telephone	1	<18
Nelson et al. (2002)	Adult twins	3982	58	30	Telephone	>1	<16
Fergusson et al. (1996a)	Adolescents	1019	51	18	Face-to-face	>1	<16
Higgins and McCabe (1994)	College students	253	79	21	Self-report	>1	<18
Goldman and Padayachi (1997)	College students	427	67	21	Self-report	>1	<17
Martin (1996)	School students	352	43	15	Self-report	1	<18
WPR-B (China $n = 1$, Malaysia $n = 1$)							
Level C: Community subgroups and convenience samples							
So-kum Tang (2000)	College students	2038	57	21	Self-report	>1	<17
Singh et al. (1996)	College students	616	77	22	Self-report	>1	<18

Postal SR Postal self-report.

— No data.

[a] Prevalence estimate not included in regression analysis based on outlier analysis (>3 standard deviations away from the mean).

	Prevalence in males (%)					Prevalence in females (%)				
Restriction on CSA definition	*Any broad*	*Any narrow*	*Non-contact only*	*Contact only*	*Intercourse*	*Any broad*	*Any narrow*	*Non-contact only*	*Contact only*	*Intercourse*
—	—	—	—	—	—	7.6	—	—	—	—
—	—	—	—	—	—	4.7	—	—	—	—
—	—	—	—	—	—	6.1	—	—	—	—
No	10.0[b]	—	—	—	—	10.0[b]	—	—	—	—
—	59.0	—	—	—	—	78.0	—	—	—	—
—	—	—	—	—	—	—	65.0	—	—	—
—	26.0	—	—	—	—	26.0	—	—	—	—
Yes	—	—	—	—	—	31.9	24.3	6.8	17.0	7.3
No	—	—	—	—	—	—	9.9	—	—	—
Yes	9.0	—	—	—	—	27.6	—	—	—	—
No	—	—	—	—	—	39.0	—	—	—	6.0
Yes	—	—	—	—	—	32.3	20.3	12.0	18.3	2.0
Yes	—	—	—	—	—	31.9	25.1	6.8	22.0	3.1
No	—	2.5	—	—	—	—	5.9	—	—	—
Yes	—	5.4	—	—	—	—	16.7	—	—	—
Yes	3.4	3.0	0.4	1.6	1.4	17.3	13.0	4.3	7.4	5.6
Yes	22.0	—	—	—	—	24.0	—	—	—	—
Yes	18.6	13.0	5.6	—	—	45.0	39.0	6.0	—	—
No	—	4.5	—	—	—	—	13.2	—	—	—
No	33.3[a]	—	—	—	3.0	28.2	—	—	—	5.8
No	2.1	2.1	0.0	2.1	0.0	8.8	5.5	2.3	5.1	0.4

[b] Estimates of prevalence not provided for males and females separately. Estimate for all persons displayed in the table. Regression modelling used estimates derived from methods outlined in section 3.

[c] Definition of CSA not provided, therefore assumed to be narrow.

Table 23.4 Characteristics of 15 excluded studies

Subregion	*Country*	*Author(s)*	*Type of study*	*Sample size*	*Sample type*
AMR-A	Canada	Berry (1997)	Prevalence	327	College students
AMR-A	Canada	Committee on Sexual Offences Against Children and Youths (1984)	Prevalence	2 008	Community adults
AMR-A	USA	Hernandez et al. (1993)	Prevalence and risk factor	2 973	School students
AMR-A	USA	Hibbard et al. (1990)	Prevalence and risk factor	3 998	School students
AMR-A	USA	Lenihan (1996)	Prevalence	1 687	College students
AMR-A	USA	Locke (1996)	Risk factor	—	College students
AMR-A	USA	Priest (1991)	Prevalence	—	College students
EUR-A	Austria	Friedrich et al. (1997)	Prevalence	—	Adult women
EUR-A	Italy	Meledandri et al. (1996)	Prevalence	—	—
EUR-B	Czech Republic	Pothe et al. (2000)	Prevalence	1 112	Community adults
WPR-A	Australia	Baldini (1996)	—	—	Aboriginal communities
WPR-A	Australia	Barton (1987)	Prevalence	>1 000	College students
WPR-B	China	Wang et al. (1994)	Prevalence	—	—
NA	NA	Garabedian (1994)	Risk factor	—	—
NA	NA	Garabedian (1994)	Risk factor	—	Adult women

NA Not applicable.

— No data.

2.8 Estimates by age, sex and subregion

Table 23.5 presents the final estimates of CSA prevalence by age, sex, level of exposure and subregion. It is presented in the format required for the WHO estimates. Table 23.6 presents the same data with level of abuse and age categories collapsed. This allows an easier comparison across subregions for both males and females. From Table 23.6 a number of interesting findings emerge. First, it can be seen that, on average, the prevalence of CSA is higher in females than in males. This is a commonly reported phenomenon. As demonstrated in the regression analysis, differences in prevalence also exist between subregions. Due to the paucity of prevalence estimates in subregions other than AMR-A, EUR-A and WPR-A it is not possible to look at differences between all subregions. However, the pattern of results does suggest that a high prevalence of CSA is found in AFR-E and SEAR-D. It should be noted that the

Table 23.5 CSA prevalence estimates (%) by subregion, sex, level of exposure and age group

			Age group (years)							
Subregion	*Sex*	*Level*	*0–4*	*5–14*	*15–29*	*30–44*	*45–59*	*60–69*	*70–79*	*≥80*
AFR-D	Male	1	0.3	2.7	2.5	3.7	3.7	3.7	3.7	3.7
		2	0.3	2.6	2.5	3.6	3.6	3.6	3.6	3.6
		3	0.2	1.6	1.6	2.3	2.3	2.3	2.3	2.3
	Female	1	0.5	4.5	4.3	6.2	6.2	6.2	6.2	6.2
		2	0.9	7.9	7.5	10.9	10.9	10.9	10.9	10.9
		3	0.3	3.0	2.9	4.2	4.2	4.2	4.2	4.2
AFR-E	Male	1	1.3	12.1	11.8	16.6	16.6	16.6	16.6	16.6
		2	0.5	4.8	4.6	7.1	7.1	7.1	7.1	7.1
		3	0.5	4.2	4.2	5.9	5.9	5.9	5.9	5.9
	Female	1	0.5	4.5	4.3	6.2	6.2	6.2	6.2	6.2
		2	2.5	22.2	21.6	30.5	30.5	30.5	30.5	30.5
		3	0.5	4.3	4.1	6.0	6.0	6.0	6.0	6.0
AMR-A	Male	1	0.1	1.9	1.9	2.7	2.7	2.7	2.7	2.7
		2	0.2	1.8	1.8	2.5	2.5	2.5	2.5	2.5
		3	0.1	1.0	1.0	1.4	1.4	1.4	1.4	1.4
	Female	1	0.4	5.4	5.3	7.5	7.5	7.5	7.5	7.5
		2	0.9	9.7	9.7	13.6	13.6	13.6	13.6	13.6
		3	0.4	3.9	4.0	5.5	5.5	5.5	5.5	5.5
AMR-B	Male	1	0.5	4.3	4.2	5.9	5.9	5.9	5.9	5.9
		2	0.3	2.3	2.3	3.2	3.2	3.2	3.2	3.2
		3	0.1	1.2	1.2	1.6	1.6	1.6	1.6	1.6
	Female	1	0.2	1.8	1.8	2.4	2.4	2.4	2.4	2.4
		2	0.3	3.0	3.0	4.2	4.2	4.2	4.2	4.2
		3	0.1	1.3	1.3	1.7	1.7	1.7	1.7	1.7
AMR-D	Male	1	0.6	5.6	5.2	7.7	7.7	7.7	7.7	7.7
		2	0.6	5.5	5.1	7.6	7.6	7.6	7.6	7.6
		3	0.4	3.4	3.2	4.7	4.7	4.7	4.7	4.7
	Female	1	0.3	2.8	2.7	3.9	3.9	3.9	3.9	3.9
		2	0.5	4.9	4.8	6.8	6.8	6.8	6.8	6.8
		3	0.2	1.9	1.8	2.6	2.6	2.6	2.6	2.6
EMR-B	Male	1	0.4	3.2	3.2	4.4	4.4	4.4	4.4	4.4
		2	0.4	3.2	3.2	4.3	4.3	4.3	4.3	4.3
		3	0.2	2.0	2.0	2.7	2.7	2.7	2.7	2.7
	Female	1	0.7	5.9	5.9	8.2	8.2	8.2	8.2	8.2
		2	1.2	10.4	10.4	14.3	14.3	14.3	14.3	14.3
		3	0.4	4.0	4.0	5.5	5.5	5.5	5.5	5.5
EMR-D	Male	1	0.4	3.2	3.2	4.4	4.4	4.4	4.4	4.4
		2	0.4	3.2	3.2	4.3	4.3	4.3	4.3	4.3
		3	0.2	2.0	2.0	2.7	2.7	2.7	2.7	2.7
	Female	1	0.7	5.9	5.9	8.2	8.2	8.2	8.2	8.2
		2	1.2	10.4	10.4	14.3	14.3	14.3	14.3	14.3
		3	0.4	4.0	4.0	5.5	5.5	5.5	5.5	5.5

continued

Table 23.5 CSA prevalence estimates (%) by subregion, sex, level of exposure and age group (*continued*)

			Age group (years)							
Subregion	*Sex*	*Level*	*0–4*	*5–14*	*15–29*	*30–44*	*45–59*	*60–69*	*70–79*	*≥80*
EUR-A	Male	1	0.1	0.9	0.9	1.2	1.2	1.2	1.2	1.2
		2	0.1	1.2	1.2	1.6	1.6	1.6	1.6	1.6
		3	0.1	0.7	0.7	0.9	0.9	0.9	0.9	0.9
	Female	1	0.4	3.5	3.6	4.9	4.9	4.9	4.9	4.9
		2	0.7	6.0	6.2	8.3	8.3	8.3	8.3	8.3
		3	0.2	1.9	2.0	2.7	2.7	2.7	2.7	2.7
EUR-B	Male	1	0.4	3.2	3.2	4.4	4.4	4.4	4.4	4.4
		2	0.4	3.2	3.2	4.3	4.3	4.3	4.3	4.3
		3	0.2	2.0	2.0	2.7	2.7	2.7	2.7	2.7
	Female	1	0.7	5.9	5.9	8.2	8.2	8.2	8.2	8.2
		2	1.2	10.4	10.4	14.3	14.3	14.3	14.3	14.3
		3	0.4	4.0	4.0	5.5	5.5	5.5	5.5	5.5
EUR-C	Male	1	0.3	2.5	2.5	3.5	3.5	3.5	3.5	3.5
		2	0.3	2.5	2.4	3.4	3.4	3.4	3.4	3.4
		3	0.2	1.5	1.5	2.1	2.1	2.1	2.1	2.1
	Female	1	0.6	5.7	5.6	7.9	7.9	7.9	7.9	7.9
		2	1.1	10.0	9.9	13.8	13.8	13.8	13.8	13.8
		3	0.4	3.9	3.8	5.3	5.3	5.3	5.3	5.3
SEAR-B	Male	1	0.2	1.7	1.6	2.3	2.3	2.3	2.3	2.3
		2	0.2	1.6	1.6	2.3	2.3	2.3	2.3	2.3
		3	0.1	1.0	1.0	1.4	1.4	1.4	1.4	1.4
	Female	1	0.2	1.5	1.5	2.1	2.1	2.1	2.1	2.1
		2	0.3	2.6	2.6	3.6	3.6	3.6	3.6	3.6
		3	0.1	1.0	1.0	1.4	1.4	1.4	1.4	1.4
SEAR-D	Male	1	1.1	9.8	9.5	13.6	13.6	13.6	13.6	13.6
		2	1.0	9.5	9.3	13.3	13.3	13.3	13.3	13.3
		3	0.7	5.9	5.7	8.2	8.2	8.2	8.2	8.2
	Female	1	1.1	10.5	10.2	14.7	14.7	14.7	14.7	14.7
		2	3.0	27.7	26.8	39.1	39.1	39.1	39.1	39.1
		3	1.1	10.0	9.7	13.9	13.9	13.9	13.9	13.9
WPR-A	Male	1	0.2	1.4	1.4	1.9	1.9	1.9	1.9	1.9
		2	0.2	1.8	1.8	2.5	2.5	2.5	2.5	2.5
		3	0.1	0.9	0.9	1.4	1.4	1.4	1.4	1.4
	Female	1	0.8	6.3	6.4	8.6	8.6	8.6	8.6	8.6
		2	1.0	11.4	11.6	15.8	15.8	15.8	15.8	15.8
		3	0.3	3.3	3.4	4.6	4.6	4.6	4.6	4.6
WPR-B	Male	1	1.1	9.6	9.9	13.2	13.2	13.2	13.2	13.2
		2	1.0	9.2	9.5	12.7	12.7	12.7	12.7	12.7
		3	0.2	1.9	1.9	2.6	2.6	2.6	2.6	2.6
	Female	1	0.6	5.8	6.0	8.0	8.0	8.0	8.0	8.0
		2	1.1	10.3	10.6	14.1	14.1	14.1	14.1	14.1
		3	0.5	4.1	4.3	5.7	5.7	5.7	5.7	5.7

Table 23.6 CSA prevalence estimates (%) by subregion and sex

	Broad estimate	
Subregion	*Males*	*Females*
AFR-D	9.6	21.3
AFR-E	29.8	42.7
AMR-A	6.7	26.5
AMR-B	10.7	8.4
AMR-D	20.0	13.3
EMR-B[a]	11.5	28.0
EMR-D[a]	11.5	28.0
EUR-A	3.8	15.8
EUR-B	11.5	28.0
EUR-C	9.0	27.0
SEAR-B	6.0	7.1
SEAR-D	35.0	67.7
WPR-A	5.9	29.1
WPR-B	28.6	27.8

[a] Subregions for which no data were available. Estimates were used from EUR-C.

estimates from AFR-E and SEAR-D come from very few studies that were relatively poor methodologically. This makes the estimates for these subregions at best highly uncertain. More studies are obviously needed to confirm whether or not the prevalence is higher in these subregions of the world. Some of these subregional differences appear dependent on sex. For example, in AMR-B, AMR-D and WPR-B the studies reported a higher prevalence in males than females. Only a few studies contributed to the estimates in these subregions and without measures of variability around the estimates it was difficult to draw firm conclusions.

2.9 Quantitative and qualitative sources of uncertainty

Uncertainty in the current analysis came from several sources. Studies of the prevalence of CSA varied in terms of methodological characteristics. Regression analyses demonstrated that several methodological factors contributed to the variability in prevalence estimates. This was the major quantitative source of uncertainty and was reduced by adjusting the prevalence estimates to more closely reflect ideal methodology. Meta-analysis was used as a method of quantifying the uncertainty around the final prevalence estimates, taking into account sample size or variability between studies, whichever is appropriate given the homogeneity of the estimates being combined. Other sources of uncertainty arose from

decisions made regarding inclusion and exclusion criteria, and methods of extrapolation across age, sex and subregion. These decisions and their rationale have been documented in the relevant sections. It should also be noted that the estimates of uncertainty will necessarily be underestimated, as data do not exist for every country in all subregions.

3. Estimating risk factor–disease relationships

3.1 Outcomes to be assessed: including evidence for causality and reasons for exclusion of related outcomes

Outcomes included and excluded

The choice of outcomes to be assessed was guided by two principles. First, choice of outcomes was limited to those diseases or outcomes that were included in the Global Burden of Disease (GBD) study. Second, outcomes were limited to those for which there was sufficient evidence of a causal relationship with CSA, the exposure variable. Section 3.4 presents a detailed assessment of the evidence for causality between CSA and six mental disorders (depression, panic disorder, agoraphobia, PTSD, and alcohol and drug abuse or dependence) and suicide attempts.

Child sexual abuse has also been linked to other mental disorders, including OCD, eating disorders and personality disorders. Three studies examined the relationship between CSA and OCD (Arata 1999; Saunders et al. 1992; Stein et al. 1988), however the evidence was equivocal and further research is required to confirm any association or lack thereof. Eating disorders have long been conceptualized as a response to a dysfunctional family environment. While it was originally thought that CSA played a pivotal role in the development of eating disorders, recent community studies have reported only a modest association between CSA and subsequent development of eating disorders after controlling for confounding influences of the family and social environment (Mullen et al. 1996; Wonderlich et al. 1997). CSA has also been linked to personality disorders, with a couple of studies finding positive associations between CSA and antisocial personality disorder (Scott 1992) and borderline personality disorder (Johnson et al. 1999). These disorders are currently not included in the GBD project and therefore were not examined here.

CSA does not only produce an increased risk of mental disorder. There is anecdotal and epidemiological evidence that CSA increases the probability of negative psychological outcomes such as poor self-esteem (Romans et al. 1997), lack of a sense of control or agency (Mullen and Fleming 1998), difficulties with intimacy and continuing sexual difficul-

ties that demonstrate the far reaching damage that occurs in some individuals (Mullen et al. 1996). These outcomes were outside the scope of this analysis and several meta-analyses examining the relationship between CSA and aspects of psychological adjustment offer a more comprehensive review of these psychological correlates (Jumper 1995; Paolucci et al. 2001; Rind et al. 1998). But this does not infer that they are in any way less harmful or less important to the person than the mental disorders identified in this report. The impact of CSA on adult life has also been studied in terms of non-mental health consequences. These include the increased risk of developing sexually transmitted diseases, teenage pregnancies, multiple sexual partnerships and sexual revictimization (Gorcey et al. 1986; Nagy et al. 1995). It has been suggested that a history of CSA, particularly of the more intrusive types, interrupts the child's development of sexuality and normal sexual relationships (Fleming et al. 1999). However, these were also outside the scope of this analysis.

General assessment of causality

Below, Hill's (1965) criteria for causality were applied to the case of CSA and adult mental disorders. The remainder of this section outlines each of these criteria as they relate to research in the area of child sexual abuse. A more detailed assessment of causality for each outcome is included in section 3.4.

Temporality

To satisfy the requirement of temporality, exposure to child sexual abuse must occur prior to the onset of adult mental disorder. By definition, some mental disorders are neither present nor easy to diagnose in children; therefore the time of onset of these disorders relative to child sexual abuse is a moot point. However, it is generally acknowledged that pre-existing vulnerabilities or predisposition to adult mental disorder exist even in childhood (Caspi et al. 1996). Long-term prospective studies that follow children from a young age and control for these vulnerabilities therefore provide the best evidence regarding temporality. While a number of research projects have been prospective in design, the majority of studies of child sexual abuse have been cross-sectional and retrospective. Traditionally, cross-sectional studies are not considered sources of evidence for temporality. However, by definition, exposure to child sexual abuse occurs during childhood and therefore prior to onset of an adult psychiatric disorder. It is for this reason that cross-sectional studies that indicate a relationship between child sexual abuse and adult mental disorder can still be indicators of temporality.

It is important to point out that even in prospective studies, data on CSA are gathered retrospectively. It is unethical, and in many countries illegal, to prospectively identify CSA and not intervene. However, retrospective studies rely on recall of memories and can therefore suffer from

unreliable data. Moreover, reports of CSA are often ascertained contemporaneously with assessment of disorder, which leaves recall open to bias in which those with disorder are more prone to recall CSA (Mullen et al. 2000). The issue of potentially unreliable recall represents one of the central threats to the validity of the published literature on CSA. Unfortunately, given that very few cases of CSA are reported to other adults, and even fewer to authorities, the validity of retrospective of CSA is very difficult to establish. Fergusson and Mullen (1999) recommended that one way of approaching this issue was to question the same individuals on multiple occasions to examine the consistency of their reports. Although this issue has rarely been examined, evidence suggests moderate-to-good consistency of CSA reports over time (Fergusson and Mullen 1999). Moreover, evidence also indicates that unreliability commonly arises from false negative reports (Fergusson et al. 2000) rather than false positive reports. Presumably greater validity would also be achieved if the presence of disorder were not determined at the same time as reports of CSA were obtained. As almost none of the prospective studies of CSA separated the ascertainment of disorder and reports of CSA, it is difficult to comment on what effect, if any, this may have had on results published to date. In summary, given that retrospective reports of CSA are virtually the only measure of CSA available in the literature, they must be accepted within the context of the caveats stated.

Strength

Several prospective studies (Fergusson et al. 1996b; Silverman et al. 1996) and several large studies with representative samples (Molnar et al. 2001; Saunders et al. 1999; Stein et al. 1988; Wilsnack et al. 1997) have found an association between CSA and mental disorders. These studies have reported odds ratios (ORs) of between 1.1 for depression (Kendler et al. 2000) and 10.2 for PTSD (Molnar et al. 2001). Despite this variability, CSA has been found to have at least a moderate effect on the outcomes studied (see Table 23.7 for a summary of the evidence). The strength of the relationship between CSA and mental disorders or suicide attempts is generally reduced when the effects of mediating variables are taken into account. This is particularly evident for non-contact forms of abuse.

Elimination of other possible causes

Child sexual abuse often co-occurs within the context of other family dysfunction, social deprivation, emotional and physical abuse and other environmental stressors that are also associated with mental disorders (Fergusson and Mullen 1999). The interaction between these additional stressors, CSA and adult mental disorders is not likely to be simple or linear (Mullen et al. 1996, 2000; Rutter 1999). Furthermore, it has been argued that the apparent association between CSA and mental disorders can in fact be attributed to family dysfunction rather than to CSA (Rind

et al. 1998). It is therefore important to establish that the effect of CSA on adult functioning remains after controlling for some of these co-occurring factors. The following section contains a discussion of the mediating factors that are commonly reported in studies of the effects of CSA on adult functioning.

Sociodemographic characteristics. CSA is not evenly distributed across sex and socioeconomic groups (Mullen et al. 2000), factors that have been found to be independently associated with mental disorders in adulthood (Andrews et al. 2001; Kessler et al. 1994). However, after controlling for these factors, several studies have demonstrated an independent association between CSA and mental disorders (see Table 23.7). Additionally, a recent meta-analysis found that sex or socioeconomic status did not mediate the relationship between CSA and depressive symptoms, PTSD or suicide attempts (Paolucci et al. 2001).

Family environment. Family environment is one of the most commonly reported mediating factors in the CSA literature. Aspects of family environment studied are myriad but include parental functioning and relationships, domestic violence, parental separation during childhood, growing up away from parents, poor parental health—both physical and emotional—and parental drug and alcohol use (Conte and Schuerman 1987; Fergusson and Mullen 1999; Fromuth 1986; Jumper 1995; Kendall-Tackett et al. 1993; Martin 1996; Mullen et al. 2000; Neumann et al. 1996; Rind et al. 1998; Wyatt and Newcomb 1990). Although measures of adverse family environment vary substantially, it is generally considered to be one of the most important mediators of the effect of CSA on adult functioning (Chandler and Jackson 1997; Fergusson and Mullen 1999; Rind et al. 1998). Moreover, most studies have found that although the independent effect of CSA on adult functioning is substantially reduced once family environment is controlled for CSA, particularly abuse involving penetration is significantly and independently associated with negative outcome (Mullen et al. 2000).

Other abuse. Children who have experienced CSA are at considerably greater risk of experiencing other types of abuse such as physical abuse and neglect (Bifulco et al. 1991; Briere and Runtz 1990; Fergusson and Mullen 1999; Fergusson et al. 1996a; Hibbard et al. 1990; Mullen et al. 1996, 2000; Paradise et al. 1994). There is also some evidence that psychopathology increases with the number of abuse types experienced. In a retrospective study of adult women in New Zealand, the chances of being assigned a clinical diagnosis increased to 24% for a single type of abuse (sexual, physical or emotional), 41% for two types of abuse and to 60% for three types of abuse (Mullen et al. 1996). As such it is difficult to isolate the independent contribution of each of these types of abuse to adult psychopathology. Nonetheless, of the studies included in

the present report that controlled for other types of abuse the majority supported an independent effect of CSA on outcome (Molnar et al. 2001; Mullen et al. 1993, 1996; Yama et al. 1995; Zuravin and Fontanella 1999).

Temperament. The area of CSA deals specifically with human behaviour and therefore with substantial gene environment interaction (Kendler et al. 2000; Rutter 1999). Genetic factors act to enhance vulnerability to mental disorders in general, and may also act to enhance or reduce the risk of developing mental disorders following CSA. Twin studies provide one of the best ways to examine the interplay between genetic and environmental influences. There have been three studies examining the effects of CSA on mental disorders in twins (Dinwiddie et al. 2000; Kendler et al. 2000; Nelson et al. 2002). All three studies concluded that CSA is independently associated with most mental disorders and suicide attempts; two reported that the effect was found, even in twins discordant for CSA, when genetic vulnerabilities and many family factors were controlled for. Unfortunately small numbers of CSA-discordant twins in these analyses meant that, although these studies were included in the final estimates for this report, we could not include a separate genetic adjustment factor.

Protective factors. Much of the discussion around variables that mediate the effects of CSA on adult mental disorder focuses on negative or destructive environmental influences rather than protective ones. However, a recent prospective study examined external protective factors that can modify a child's psychiatric trajectory. Lynskey and Fergusson (1997) developed a regression model that demonstrated factors that protected against the development of psychiatric disorders. These were higher levels of paternal care during childhood and having fewer affiliations with delinquent or substance abusing peers. Once the model adjusted for both of these factors, severity of the sexual abuse (ranging from none, non-contact, contact through to intercourse) was not a significant predictor of outcome. Similarly, having a warm and supportive relationship with the non-offending parent and lower levels of abuse-related stress have been shown to predict resilience in sexually abused girls (Spaccarelli and Kim 1995). In essence, while the majority of research has shown that other negative factors contribute to the risk of developing adult mental disorder having experienced sexual abuse as a child, the converse is also likely to be true. That is, certain positive mediating variables are likely to reduce the risk of negative outcomes following CSA. This concept is often referred to as resilience (Fergusson and Mullen 1999). Unfortunately very few studies have measured protective factors in a systematic way and as such they could not be quantified for the present report.

Covariation or biologic gradient

For biological risk factors such as blood pressure levels this criterion is typically established by the presence of a dose–response relationship between risk factor and outcome. In the area of child sexual abuse, it is difficult to determine the presence of a dose–response relationship because it is difficult to define a "dose". A dose most closely relates to the severity of abuse to which an individual is subjected. The literature generally defines severity of abuse in five ways: type, frequency, duration, age of onset of abuse, and relationship of victim to offender. Regardless of how it is defined, there is broadly supportive evidence relating the severity of the abuse to the degree of psychiatric or psychological disturbance.

Type of abuse. Using this definition, severity of abuse is generally taken to express the spectrum that ranges from non-contact forms of sexual abuse (e.g. verbal sexual invitations, showing pornography), to contact forms of abuse (touching), through to intercourse. In those studies that have presented risk for disorder according to exposure to different types of abuse, risk for disorder increases as exposure to more severe types of abuse occurs (Baynard 1999; Fergusson et al. 1996b; Kendler et al. 2000; Saunders et al. 1992). In general the literature supports the notion that CSA involving contact or intercourse is associated with a more negative outcome in adulthood than non-contact CSA. It is also the most widely reported definition of the severity of exposure to CSA and is therefore used to obtain estimates for the current report.

Frequency of abuse. It has been demonstrated that experiencing one episode of child sexual abuse is often associated with further sexual victimization. In one study, for example, 16.8% of children were reabused in the 61–72 months prior to follow-up, with the greatest risk period occurring in the two years immediately after the initial abuse (Levy et al. 1995). In another study of 24 507 children with substantiated abuse/neglect who were monitored up to four years after the initial maltreatment incident, 9.3% of the children experienced abuse or neglect in the follow-up period (Fryer and Miyoshi 1994). For these children, the risk of reabuse continued to be greater than the risk of abuse in the general population and was greatest immediately following the first notified abuse/neglect incident. For example, 24% of abused/neglected children were revictimized in the first month following the index event. Bentovim et al. (1987) followed up families who were referred to a treatment programme for sexual abuse and found that 16% of children had experienced revictimization; in 15% of children it was unclear whether children had been reabused or not. In a sample of children and adolescents, Boney-McCoy and Finkelhor (1995) found that 39% of children who had histories of prior sexual victimization had been sexually abused in

the last year. Similar outcomes can occur in adults. Women with a history of CSA were significantly more likely to experience rape as an adult and to be victims of domestic violence (Fleming et al. 1999), which raises the question as to whether child sexual abuse is a vulnerability factor to further sexual abuse in and of itself.

Several studies have reported that not only is reabuse common, it is also associated with poorer outcome. In their meta-analysis, Kendall-Tackett et al. (1993) observed that poor psychological and behavioural outcomes in children were related to a variety of abuse-related variables, including greater abuse frequency. Increases in frequency of abuse have also been shown to be significantly associated with greater severity of psychological disorder, such as making more numerous suicide attempts (Bagley et al. 1995). While it is generally acknowledged that frequency of abuse is associated with more negative outcome, very few studies report outcome for varying abuse frequencies.

Duration of abuse. Duration of CSA has been shown to significantly affect psychological outcome, both in meta-analyses (Kendall-Tackett et al. 1993) and other research. Peters (1988) found that the greater the duration of the abuse, the more mental disorders and suicide attempts in adulthood. This is consistent with the notion that cumulative trauma has a more substantial effect than a single or less frequent abusive event. Once again, outcome of CSA is rarely reported for varying duration of abuse.

Age at onset of the abuse. At first glance, the evidence of the effect of age at abuse onset on mental disorder appears to be conflicting in terms of its direction. Peters (1988) found that women aged 18–36 years who had been older at the time they were sexually abused were diagnosed with more mental disorders or had made more suicide attempts. In contrast, Lynskey and Fergusson (1997) found a significant relationship between being younger at the time of the sexual abuse and increasing rates of mental disorders in a sample of 18 year olds. In both of these analyses, once adjusted for confounders, the relationship between age at abuse onset and mental disorders was no longer significant. Consistent with the conclusion of Browne and Finkelhor (1986) there appears to be no solid evidence for a relationship between age at onset and mental disorders after controlling for other aspects of the abuse and relationship variables.

Relationship of the offender to the child. A meta-analysis of the child sexual abuse research found no significant association between the relationship of the offender to the child, and mental disorders (Paolucci et al. 2001). This relationship is surprising given that intrafamilial abuse may occur over a longer period of time and with greater frequency than extrafamilial abuse (Fergusson and Mullen 1999). Browne and

Finkelhor (1986) suggest two reasons why the relationship of the child to the offender may not be a consistent predictor of negative outcomes. First, that lack of a consistent association may reflect variations in the degree of betrayal, rather than whether the victim and perpetrator are related. Second, while abuse by someone who is trusted may involve betrayal, abuse by a stranger may involve more fear and therefore be more aversive to the victim.

Consistency

The literature has consistently reported that psychiatric disorders are frequently found to be more common among those subjected to CSA compared to their non-abused peers. This finding persists across a range of populations that have included college students, community samples, school students, children, adolescents and adults, and cohorts in a number of different developed countries. Few studies, however, have been conducted in developing countries. Further research is required to confirm that the deleterious consequences associated with CSA reported in the literature so far also applies to the rest of the world and is not mediated by cultural and social factors.

Plausibility

It is acknowledged that psychiatric disorder arises from an interaction between adverse environmental influences and an individual's genetic make-up (Rutter 1999). Genetic influences aside, it is accepted that childhood adversity is a potent influence on psychiatric outcome (Brown and Moran 1994; Kessler and Magee 1993). This milieu of adversity has been described as a matrix of disadvantage (Mullen et al. 2000) and includes a variety of socioeconomic, familial and other environmental factors, as outlined earlier. Child sexual abuse falls at the more severe end of the spectrum of this adversity.

In terms of the effect that child sexual abuse has on the individual, it is logical that a child exposed to a traumatic event such as sexual assault may function less well psychologically and may develop phobic responses and anxiety-related symptoms, including PTSD (Green 1988). It has been proposed that the sexual abuse, regardless of type, involves four traumagenic dynamics (Finkelhor and Browne 1988). These are betrayal, powerlessness, traumatic sexualization and stigmatization. Synthesizing the child sexual abuse literature, Polusny and Follette (1995) placed the various outcomes associated with child sexual abuse in the context of emotional avoidance, suggesting that these outcomes are the result of maladaptive coping behaviour. Within this framework, a spectrum of avoidance, anxiety, despair and attempts to control becomes evident. When that fails, it produces anxiety disorders, alcohol and substance abuse, depression and other psychopathology, and suicide at the extreme. Within this context, despite the lack of a biological link between CSA and mental disorders, a causal relationship would certainly be plausible.

3.2 Description of studies including methodological qualities

Table 23.7 presents the characteristics of the studies that contributed to the risk factor–disease relationship grouped by psychiatric outcome. Also reported in Table 23.7 are the range of odds ratios and significance for each study.

3.3 Overview of methods

The criteria for identifying relevant studies and the characteristics of excluded studies have been reported on previously. All articles that met the inclusion criteria were coded against each outcome measured. The majority of studies measured more than one of our chosen outcomes. Where possible both unadjusted and adjusted measures of risk (RRs and ORs) were coded from articles. The majority of studies did not present estimates of risk adjusted for relevant confounders. Moreover, many presented proportions only. Regardless of which estimates of risk or association were quoted in articles, 2x2 tables were coded from every article included in the analysis. Where possible 2x2 tables were coded for each exposure level and where these data were not available 2x2 tables were coded for overall exposure. Where studies presented risks or proportions for both lifetime and current levels of outcome, both were coded. Many articles presented outcome as a continuous rather than a categorical variable. In these cases and where studies had used a measure of outcome that could be mapped to diagnostic criteria via a validated cut-off point, authors were contacted and asked to supply 2x2 tables. Unadjusted RRs and ORs and corresponding 95% confidence intervals were calculated from each 2x2 table using conventional formulae (Gardner and Altman 1989; Streiner 1998).

Extrapolations across sex, diagnostic time frame and levels of exposure

Given the requirements for data presentation (relative risk by age and sex for each level of exposure for each subregion) the biggest source of error in the data arose from having a small number of studies from which to derive estimates. The following sections detail the decisions that were made for each of these extrapolations. However, several general rules applied.

- Consider any theoretical implications of the extrapolation (e.g. is there any reason to expect relative risk will vary with age or sex or that confounding factors will differ for different mental disorder outcomes? Is there a plausible hypothesis or explanation?).
- Assume that no difference exists between the groups of interest (e.g. across sex, age or diagnostic time frame) unless there is clear evidence of a consistent pattern.

Table 23.7 Characteristics of studies included in the risk factor–disease relationship analysis

	Sample				Adjusted for confounders									
Level of evidence	*Type*	*N*	*% female*	*Age (years)*	*Demo-graphics*	*Family function*	*Other abuse*	*Other disorders*	*Outcome measure*[a]	*Diagnosis time frame*	*Childhood definition*	*CSA definition*[b]	*OR*	*P <0.05*
Depression														
Level 1: Twin studies														
Dinwiddie et al. (2000)	Adult twins	5946	65	43	Yes	No	No	No	SSAGA	Lifetime	<18	Narrow	2.2–3.9	Yes
Kendler et al. (2000) (10-year follow-up)	Adult twins	1411	100	40	No	Yes	No	No	SCID	Lifetime	<16	Broad	1.1–2.8	Yes[c]
Nelson et al. (2002)	Adult twins	33892	58	30	Yes	Yes	No	Yes	SSAGA	Lifetime	<16	Narrow	1.3–1.7	Yes[c]
Level 2: Prospective studies														
Brown and Harris (1993) (8-year follow-up)	Low income women	404	100	—	No	Yes	Yes	No	PSE	12 months	<17	Broad	—	Yes
Brown et al. (1999) (13-year follow-up)	Community	639	48	—	Yes	Yes	No	Yes	DISC	—	<18	Narrow	3.2	Yes
Ernst et al. (1993) (10-year follow-up)	Community	421	47	28	No	No	No	No	SPIKE interview	—	<16	Broad	—	No
Fergusson et al. (1996b) (Follow-up U/K)	Community	1019	51	18	Yes	Yes	No	Yes	CIDI	2 years	<16	Broad	3.0–5.4	Yes
Silverman et al. (1996) (17-year follow-up)	Community	375	50	—	No	No	No	No	CDI & DIS	2 weeks & lifetime	<18	—	2.0	Yes

continued

Table 23.7 Characteristics of studies included in the risk factor–disease relationship analysis (*continued*)

	Sample				Adjusted for confounders									
Level of evidence	*Type*	*N*	*% female*	*Age (years)*	*Demo-graphics*	*Family function*	*Other abuse*	*Other disorders*	*Outcome measure*[a]	*Diagnosis time frame*	*Childhood definition*	*CSA definition*[b]	*OR*	*P <0.05*
Level 3: Cross-sectional studies														
A. Representative community samples														
Molnar et al. (2001)	Adults	5877	50	—	Yes	Yes	Yes	No	CIDI	Lifetime	<18	Broad	1.8	Yes[c]
Saunders et al. (1999)	Adults	4008	100	45	Yes	No	No	No	Clinical interview	Lifetime & 12 months	<18	Intercourse	2.5–2.6	Yes
Stein et al. (1988)	Adults	2683	51	—	Yes	No	No	No	DIS	Lifetime & 6 months	<16	Narrow	2.0–2.64	Yes
Wilsnack et al. (1997)	Community	641	100	—	Yes	No	No	No	DIS	Lifetime	<18	Broad	2.51	Yes
B. Other community samples (representativeness not known)														
Bagley and Ramsay (1985)	Adults	377	100	40	No	No	No	No	CESD	1 month	<17	Narrow	eta = 0.25	Yes
Bagley et al. (1994)	Adults	750	0	23	No	No	No	No	CESD ≥28	1 month	<17	Narrow	—	Yes
Lopez et al. (1995)	Adults	1821	47	—	—	—	—	—	—	—	<17	Broad	—	Yes
Mullen et al. (1996)	Adults	497	100	—	No	Yes	Yes	No	PSE	Lifetime	<16	Broad	1.8	Yes
Peters (1988)	Adults	119	100	—	Yes	No	No	No	SADS	Lifetime	<18	Narrow	—	Yes
Saunders et al. (1992)	Adults	391	100	42	No	No	No	No	DIS	Lifetime & 1 month	<18	Broad	1.65–1.75	Yes[c]
C. Community subgroups or convenience samples														
Arata (1999)	College	92	100	24	No	No	No	No	SCID	Lifetime & 1 month	<14	Broad	—	No

Chandler and Jackson (1997)	College	266	100	19	No	No	No	No	BDI	2 weeks	<18	Broad	—	Yes
Jackson et al. (1990)	College	40	100	23	No	No	No	No	BDI	2 weeks	<18	Narrow	—	No
Zuravin and Fontanella (1999)	Low income women	513	100	30	Yes	Yes	Yes	No	DIS	1 month	<14	Narrow	3.36	Yes
Level 4 studies: Prospective case–control studies														
Swanston et al. (1997) (5-year follow-up)	CPU & controls from community	143	100	15	Yes	Yes	No	No	BDI & CDI	2 weeks	<16	Narrow	48.1	Yes
Level 5: Single wave case–control studies														
Mullen et al. (1993)	Community	492	100	—	No	No	No	No	PSE	Lifetime	<16	Broad	2.6–5.2	Yes
Wise et al. (2001)	Community	732	100	—	Yes	Yes	No	No	SCID	Lifetime	<18	Narrow	2.2	Yes
Panic disorder														
Level 1: Twin studies														
Dinwiddie et al. (2000)	Adult twins	5946	65	43	Yes	No	No	No	SSAGA	Lifetime	<18	Narrow	3.5–5.0	Yes
Kendler et al. (2000) (10-year follow-up)	Adult twins	1411	100	40	No	Yes	No	No	SCID	Lifetime	<16	Broad	1.3–2.6	Yes[c]
Level 2: Prospective studies														
Brown and Harris (1993) (8-year follow-up)	Low income women	404	100	—	No	Yes	Yes	No	PSE	12 months	<17	Broad	—	Yes
Ernst et al. (1993) (10-year follow-up)	Community	421	47	28	No	No	No	No	SPIKE interview	—	<16	Broad	—	No

continued

Table 23.7 Characteristics of studies included in the risk factor–disease relationship analysis (*continued*)

	Sample				Adjusted for confounders									
Level of evidence	*Type*	*N*	*% female*	*Age (years)*	*Demo-graphics*	*Family function*	*Other abuse*	*Other disorders*	*Outcome measure*[a]	*Diagnosis time frame*	*Childhood definition*	*CSA definition*[b]	*OR*	*P <0.05*
Level 3: Cross-sectional studies														
A. Representative community samples														
Molnar et al. (2001)	Adults	5877	50	—	Yes	Yes	Yes	No	CIDI	Lifetime	<18	Broad	0.8–1.4	Yes[c]
Stein et al. (1988)	Adults	2683	51	—	Yes	No	No	No	DIS	Lifetime & 6 months	<16	Narrow	3.4–3.9	Yes[c]
B. Other community samples (representativeness not known)														
Saunders et al. (1992)	Adults	391	100	42	No	No	No	No	DIS	Lifetime & 1 month	<18	Broad	5.0	Yes[c]
C. Community subgroups or convenience samples														
Arata (1999)	College	92	100	24	No	No	No	No	SCID	Lifetime & 1 month	<14	Broad	—	No
Obsessive–compulsive disorder														
Level 3: Cross-sectional studies														
A. Representative community samples														
Stein et al. (1988)	Adults	2683	51	—	Yes	No	No	No	DIS	Lifetime & 6 months	<16	Narrow	—	No
B. Other community samples (representativeness not known)														
Saunders et al. (1992)	Adults	391	100	42	No	No	No	No	DIS	Lifetime & 1 month	<18	Broad	4.5– ≥6	Yes[c]
C. Community subgroups or convenience samples														
Arata (1999)	College	92	100	24	No	No	No	No	SCID	Lifetime & 1 month	<14	Broad	—	No

Post-traumatic stress disorder													
Level 2: Prospective studies													
Silverman et al. (1996) (17-year follow-up)	Community	375	50	—	No	No	No	No	DIS	Lifetime	<18	—	—	Yes
Level 3: Cross-sectional studies													
A. Representative community samples													
Davidson et al. (1991)	Adults	2985	54	—	No	No	No	No	DIS	Lifetime	<16	Intercourse	9.5	Yes
Molnar et al. (2001)	Adults	5877	50	—	Yes	Yes	Yes	No	CIDI	Lifetime	<18	Broad	5.3–10.2	Yes
Saunders et al. (1999)	Adults	4008	100	45	Yes	No	No	No	Clinical interview	Lifetime & 12 months	<18	Intercourse	3.2–2.4	Yes
C. Community subgroups or convenience samples														
Arata (1999)	College	92	100	24	No	No	No	No	SCID	Lifetime & 1 month	<14	Broad	—	Yes
Hien and Bukszpan (1999)	Obs/gyn. clinic	98	100	33	No	No	No	No	SCID	Lifetime	<18	Broad	—	Yes
Robin et al. (1997)	American Indians	375	58	37	No	No	No	No	SADS-1	Lifetime & 1 month	<16	Broad	1.6–8.7	Yes[c]
Schaaf and McCanne (1998)	College	269	100	18	No	No	No	No	Clinical	Current	<15	Broad Interview	—	Yes
Alcohol abuse or dependence														
Level 1: Twin studies														
Dinwiddie et al. (2000)	Adult twins	5946	65	43	Yes	No	No	No	SSAGA	Lifetime	<18	Narrow	1.9–2.8	Yes
Kendler et al. (2000) (10-year follow-up)	Adult twins	1411	100	40	No	Yes	No	No	SCID	Lifetime	<16	Broad	1.9–6.5	Yes[c]
Nelson et al. (2002)	Adult twins	3892	58	30	Yes	Yes	No	Yes	SSAGA	Lifetime	<16	Narrow	1.3–1.7	Yes[c]

continued

Table 23.7 Characteristics of studies included in the risk factor–disease relationship analysis (*continued*)

	Sample				Adjusted for confounders									
Level of evidence	*Type*	*N*	*% female*	*Age (years)*	*Demo-graphics*	*Family function*	*Other abuse*	*Other disorders*	*Outcome measure*[a]	*Diagnosis time frame*	*Childhood definition*	*CSA definition*[b]	*OR*	*P <0.05*
Level 2: Prospective studies														
Fergusson et al. (1996b) (Follow-up U/K)	Community	1019	51	18	Yes	Yes	No	Yes	CIDI	2 years	<16	Broad	1.9–2.7	Yes[c]
Silverman et al. (1996) (17-year follow-up)	Community	375	50	—	No	No	No	No	Various	Lifetime	<18	—	—	Yes
Widom and White (1997) (20-year follow-up)	Community	1190	49	29	No	No	No	No	DIS	Lifetime	<11	Narrow	—	No
Level 3: Cross-sectional studies														
A: Representative community samples														
Fleming et al. (1998)	Adults	710	100	40	Yes	Yes	Yes	No	AUDIT	—	<16	Narrow	0.61	No
Kilpatrick et al. (2000)	Adults	4023	49	—	Yes	Yes	Yes	No	Clinical interview	12 month	<17	Narrow	2.4	Yes
Molnar et al. (2001)	Adults	5877	50	—	Yes	Yes	Yes	No	CIDI	Lifetime	<18	Broad	1.5–1.7	Yes
Saunders et al. (1999)	Adults	4008	100	45	Yes	No	No	No	Clinical interview	Lifetime & 12 months	<18	Intercourse	2.0–2.4	Yes
Spak et al. (1998)	Adults	316	100	—	Yes	No	No	Yes	CIDI-SAM	Lifetime	<18	Broad	3.5	Yes[c]
Stein et al. (1988)	Adults	2683	51	—	Yes	No	No	No	DIS	Lifetime & 6 months	<16	Narrow	1.8	Yes[c]
B. Other community samples (representativeness not known)														
Peters (1988)	Adults	119	100	—	Yes	No	No	No	DIS	Lifetime	<18	Narrow	—	Yes

C. Community subgroups or convenience samples													
Arata (1999)	College	92	100	24	No	No	No	No	SCID	Lifetime & 1 month	<14	Broad	—	No
Robin et al. (1997)	American Indians	375	58	37	No	No	No	No	SADS-I	Lifetime & 1 month	<16	Broad	1.8–2.8	Yes[c]
Drug abuse or dependence														
Level 1: Twin studies														
Kendler et al. (2000) (10-year follow-up)	Adult twins	1411	100	40	No	Yes	No	No	SCID	Lifetime	<16	Broad	1.2–6.6	Yes[c]
Level 2: Prospective studies														
Fergusson et al. (1996b) (Follow-up U/K)		1019	51	18	Yes	Yes	No	Yes	CIDI	2 years	<16	Broad	0.1–2.9	Yes[c]
Silverman et al. (1996) (17-year follow-up)		375	50	—	No	No	No	No	DIS	Lifetime	<18	—	—	No
Widom and White (1997) (20-year follow-up)	Community	1190	49	29	No	No	No	No	DIS	Lifetime	<11	Narrow	—	No
Level 3: Cross-sectional studies														
A. Representative community samples														
Molnar et al. (2001)	Adults	5877	50	—	Yes	Yes	Yes	No	CIDI	Lifetime	<18	Broad	2.0–2.0	Yes
Stein et al. (1988)	Adults	2683	51	—	Yes	No	No	No	DIS	Lifetime & 6 months	<16	Narrow	1.8–2.1	Yes[c]
C. Community subgroups or convenience samples														
Arata (1999)	College	92	100	24	No	No	No	No	SCID	Lifetime & 1 month	<14	Broad	—	No
Robin et al. (1997)	American Indians	375	58	37	No	No	No	No	SADS-I	Lifetime & 1 month	<16	Broad	1.6–4.8	Yes[c]

continued

Table 23.7 Characteristics of studies included in the risk factor–disease relationship analysis (*continued*)

	Sample				Adjusted for confounders									
Level of evidence	*Type*	*N*	*% female*	*Age (years)*	*Demo-graphics*	*Family function*	*Other abuse*	*Other disorders*	*Outcome measure*[a]	*Diagnosis time frame*	*Childhood definition*	*CSA definition*[b]	*OR*	*P <0.05*
Suicide attempts														
Level 1: Twin studies														
Dinwiddie et al. (2000)	Adult twins	5946	65	43	Yes	No	No	No	SSAGA	Lifetime	<18	Narrow	7.1–7.7	Yes
Nelson et al. (2002)	Adult twins	3892	58	30	Yes	Yes	No	Yes	—	Lifetime	<16	Narrow	0.97–1.1	Yes[c]
Level 2: Prospective studies														
Brown et al. (1999) (13-year follow-up)	Community	639	48	—	Yes	Yes	No	Yes	DISC	—	<18	Narrow	5.7	Yes
Ernst et al. (1993) (10-year follow-up)	Community	421	47	28	No	No	No	No	SPIKE interview	—	<16	Broad	—	No
Fergusson et al. (1996b) (Follow-up U/K)	Community	1019	51	18	Yes	Yes	No	Yes	CIDI	2 years	<16	Broad	0.8–5.0	Yes[c]
Silverman et al. (1996) (17-year follow-up)	Community	375	50	—	No	No	No	No	Various	Lifetime	<18	—	10.7	Yes
Level 3: Cross-sectional studies														
A. Representative community samples														
Bagley et al. (1995)	Adolescents	2112	49	—	No	No	No	No	Study-specific questions	6 month	<18	Broad	10–37	Yes
Bensley et al. (1999)	Adults	4790	48	16	Yes	No	Yes	No	YRBS	12 month	<18	Broad	2.7–47.1	Yes
Garnefski and Arends (1998)	Adults	13894	50	15	Yes	No	No	No	Study-specific questions	Lifetime	<19	Narrow	—	Yes[c]

B. Other community samples (representativeness not known)														
Bagley and Ramsay (1985)	Community	377	100	40	No	No	No	No	Paykel (1972)	—	<17	Narrow	eta = 0.16	Yes
Bagley et al. (1994)	Adults	750	0	23	No	No	No	No	Study-specific questions	Lifetime	<17	Narrow	—	Yes
Leth (2001)	Adults	1235	54	—	No	No	No	No	—	Lifetime	<18	Broad	—	Yes
Martin (1996)	Adolescents	352	43	15	No	No	No	No	Smith and Crawford (1986)	6 month	<18	Narrow	—	Yes
Mullen et al. (1996)	Community	497	100	—	No	Yes	Yes	No	Study-specific questions	Lifetime	<16	Broad	3.6	Yes
Saunders et al. (1992)	Adults	391	100	42	No	No	No	No	DIS	Lifetime & 1 month	<18	Broad	3.0	Yes[c]
C. Community subgroups or convenience samples														
Bendixen et al. (1994)	College	996	51	23	No	No	No	No	Study-specific questions	Lifetime	—	Broad	—	No
Boudewyn and Liem (1995)	College	438	61	25	No	No	No	No	Study-specific questions	Lifetime	<14	Narrow	—	Yes
Chandy et al. (1996)	School students	2022	100	15	No	No	No	No	Study-specific question	Lifetime	<18	Narrow	—	Yes
Chandy et al. (1997)	School students	740	0	15	No	No	No	No	Study-specific question	Lifetime	<18	Narrow	—	Yes

continued

Table 23.7 Characteristics of studies included in the risk factor–disease relationship analysis (*continued*)

	Sample				Adjusted for confounders									
Level of evidence	*Type*	*N*	*% female*	*Age (years)*	*Demographics*	*Family function*	*Other abuse*	*Other disorders*	*Outcome measure*[a]	*Diagnosis time frame*	*Childhood definition*	*CSA definition*[b]	*OR*	*P <0.05*
Borowsky et al. (1999)	American Indian and Alaska Native youth	11666	52	15	Yes	Yes	Yes	Yes	Study-specific question	Lifetime	<18	Narrow	—	Yes
Lazartigues et al. (1989)	College	963	57	—	No	No	No	No	—	Lifetime	<16	Broad	—	Yes
Robin et al. (1997)	American Indians	375	58	37	No	No	No	No	Clinical	Lifetime interview	<16	Broad	3.1–6.9	Yes
Sedney and Brooks (1984)	College	102	100	19	No	No	No	No	Study-specific questions	Lifetime	—	Broad	—	No
Hibbard et al. (1988)	School students	712	50	15	No	No	No	No	Study-specific questions	Lifetime	<15	Broad	3.1	Yes
Hibbard et al. (1990)	School students	3998	51	15	No	No	No	No	Study-specific questions	Lifetime	<15	Broad	9.2	Yes
Yama et al. (1995)	College	379	100	20	No	Yes	No	No	Study-specific questions	Lifetime	<16	Narrow	—	Yes

Level 4 studies: Prospective case–control studies														
Plunkett et al. (2001) (9-year follow-up)	CPU & controls from community	259	75	19	No	No	No	No	Study-specific questions	Lifetime	<15	Narrow	1.9	Yes
Level 5: Single wave case–control studies														
Briere and Runtz (1986)	Crisis centre	195	100	27	No	No	No	No	Study-specific questions	Lifetime	<17	Narrow	—	Yes
Molnar et al. (1998)	Street youth	775	35	18	Yes	No	No	No	Study-specific questions	Lifetime	<18	Broad	3.2–4.3	Yes
Mullen et al. (1993)	Community	492	100	—	No	Yes	No	No	Study-specific questions	Lifetime	<16	Intercourse	8.6–25.6	Yes
Completed suicide														
Level 4 studies: Prospective case–control studies														
Plunkett et al. (2001)	CPU & controls from community	259	75	19	No	No	No	No	Death certificate	NA	<15	Narrow	Rate = 0.18%	No

Key: CPU, Hospital Child Protection Units; NA, not applicable; U/K, unknown.

— No data.

[a] Diagnostic instrument definitions: AUDIT = Alcohol Use Disorders Identification Test; BDI = Beck Depression Inventory (Score ≥16 unless otherwise specified); CDI = Children's Depression Inventory (Score ≥20 unless otherwise specified); CESD = Center for Epidemiologic Studies Scale (Depression); CIDI = Composite International Diagnostic Interview; DIS (C) = Diagnostic Interview Schedule (for children) ; PSE = Present State Examination; SADS = Schedule for the Affective Disorders and Schizophrenia; SCID = Structured Clinical Interview for DSM-III-R; SPIKE = Structured Interview to assess psychiatric & psychosomatic symptoms & syndromes, social relationships, coping & life events; SSAGA = Semi-structured assessment for the genetics of alcohol; YRBS = Youth Risk Behaviour Schedule.

[b] Broad: non-contact, contact or intercourse; Narrow: contact or intercourse only.

[c] Significant for some relationships but not others.

- In the absence of a large number of estimates assume that reporting data from fewer sources is likely to be a greater source of error than extrapolation of data to fit the categories required for reporting (e.g. risk by type of exposure or by sex).

Sex

Within the literature, a large proportion of the research has examined the sequelae of CSA in females, leaving males underrepresented. This trend was reflected in the data set with many estimates for females and relatively few for males. Additionally, for males, there were no estimates in the severity categories of non-contact, contact and intercourse. Those studies that did examine sequelae in males only gave estimates for the categories of narrow or broad CSA. Therefore, no data were available in the categories required for analysis for males. Given the assumption that more error would be introduced if no estimates were available for males, a decision was made to extrapolate from data for females. To examine the validity of this decision a comparison between males and females was made between those studies that measured outcome in both (restricted to the narrow and broad categories since males only had data for these).

Comparisons were available across all disorder categories and 95% CIs were compared between male and female estimates within studies to determine if male and female estimates differed significantly. Confidence intervals for the RRs overlapped for depression, agoraphobia, panic, drug and alcohol abuse/dependence for all the studies available for comparison. For PTSD there were significant differences for both of the studies, but as the relative risk was higher for males in one study, and this effect was reversed in the other, no difference between males and females was assumed. For suicide attempts two studies found differences between males and females with the relative risk for males being higher in both. However, in the other six studies available for comparison for suicide attempts no significant differences were observed. Additionally, reviewers have implied that no difference exists between males and females in terms of consequences of CSA (Urquiza and Capra 1990; Watkins and Bentovim 1992). Overall, given that no significant differences were found between the sexes and no theoretical reason presents, no difference between the sexes for the relationship between CSA and mental disorders was assumed.

In light of this decision and in order to maximize the number of estimates available for analysis a hierarchy for selection of studies based on sex was constructed. Where estimates for males and females combined were available from a study these were selected first. Female estimates were then chosen followed by male estimates. This ensured that one estimate was available for each outcome that each study reported.

Diagnostic time frame

There was considerable variation in the time frame used to measure outcome across studies. While some studies determined the presence of mental disorders over an individual's lifetime, others determined the presence of disorder over the past 12 months, six months or one month. While current or one month estimates might be considered the most accurate measure of current risk, these were only presented for a small number of studies (see Table 23.7). In order to examine the differences between lifetime and current estimates the following analysis was undertaken.

Lifetime and current estimates of risk were compared in the six studies that presented both. All six samples were in the 30–44-year age group. There were five sets of estimates for depression, two for panic disorder, two for drug dependence, three for alcohol dependence and two for PTSD. Only one study presented estimates for males. Ratios of current to lifetime relative risk ranged from 0.57 to 3.21 with a trend for current risks to be greater than lifetime (ratios of >1). However, when confidence intervals around RRs were examined very few comparisons were significant.

A second set of comparisons was also undertaken. Relative risks across all studies were grouped according to diagnostic time frame and averaged within age groups and outcomes. Again no clear pattern emerged. On the basis of these analyses and in order to maximize the number of estimates it was decided to include all estimates of relative risk regardless of whether the diagnostic time frame used was lifetime, current or 12 months. Where studies presented both, current risk was used.

Levels of exposure

Many studies presented relative risk for exposure vs non-exposure only, rather than by levels of exposure. Moreover these studies varied in terms of whether they presented relative risk for broad CSA (non-contact, contact or intercourse) or for narrow CSA (contact or intercourse only). There is strong evidence in the literature to suggest that outcome varies with level of exposure, risk being the highest for those who have experienced abuse involving intercourse (Fergusson and Mullen 1999). In order to examine the relationship between relative risks for each level of exposure the following analysis was undertaken.

Relative risks for overall exposure to CSA were calculated for all studies ($N = 5$) that presented risk by level of exposure. Relative risks for each level of exposure were then expressed as a ratio of the overall risk. Estimates and ratios are presented in Table 23.8.

The ratios in Table 23.8 were then applied to those studies that only reported risk for exposed vs non-exposed. More specifically, the ratios derived from those studies reporting risk estimates for the contact and intercourse categories of abuse were applied to those studies reporting

Table 23.8 Relative risks of each disorder for each level of exposure, as a ratio of overall relative risk[a]

	Study characteristics		Exposure level			Any CSA		Ratios		
Outcome	*Sample*	*N*	RR_1	RR_2	RR_3	RR_{any} broad	RR_{any} narrow	RR_1/RR_{any}	RR_2/RR_{any}	RR_3/RR_{any}
Depression										
Saunders et al. (1992)	Community	391	1.24	1.65	1.76	1.57	—	0.79	1.05	1.12
Fergusson et al. (1996b)	Community	1019	2.19	2.04	3.83	2.56	—	0.86	0.80	1.50
Kendler et al. (2000)	Community	1411	1.25	1.39	1.83	1.48	—	0.84	0.94	1.24
Mullen et al. (1993)	Community	492	—	—	4.38	2.62	—	—	—	1.67
Panic disorder										
Saunders et al. (1992)	Community	391	1.49	0.91	2.67	1.59	—	0.94	0.57	1.68
Kendler et al. (2000)	Community	1411	1.41	1.76	2.42	1.86	—	0.76	0.95	1.30
Alcohol dependence										
Fergusson et al. (1996b)	Community	1019	1.63	2.19	2.02	2.01	—	0.81	1.09	1.00
Kendler et al. (1996b)	Community	1411	2.32	2.29	3.43	2.61	—	0.89	0.88	1.31
Drug dependence										
Fergusson et al. (1996b)	Community	1019	0.78	1.64	3.66	2.13	—	0.37	0.77	1.72
Kendler et al. (2000)	Community	1411	2.63	2.03	5.19	3.05	—	0.86	0.67	1.70
Suicide										
Saunders et al. (1992)	Community	391	0.50	2.74	3.11	2.25	—	0.22	1.22	1.38
Fergusson et al. (1996b)	Community	1019	1.03	2.15	3.53	2.33	—	0.44	0.92	1.47
Mean								0.71	0.90	1.42
Depression										
Saunders et al. (1992)	Community	391	—	1.65	1.76	—	1.69	—	0.98	1.04
Fergusson et al. (1996b)	Community	1019	—	2.04	3.83	—	2.68	—	0.76	1.43
Kendler et al. (2000)	Community	1411	—	1.39	1.83	—	1.56	—	0.89	1.17
Banyard (1999)	Low income women	518	—	1.17	2.38	—	2.04	—	0.57	1.17
Panic disorder										
Saunders et al. (1992)	Community	391	—	0.91	2.67	—	1.63	—	0.56	1.57

Table 23.8 Relative risks of each disorder for each level of exposure, as a ratio of overall relative risk[a] (*continued*)

	Study characteristics		Exposure level			Any CSA		Ratios		
Outcome	Sample	N	RR_1	RR_2	RR_3	RR_{any} broad	RR_{any} narrow	RR_1/RR_{any}	RR_2/RR_{any}	RR_3/RR_{any}
Kendler et al. (2000)	Community	1411	—	1.76	2.42	—	2.01	—	0.88	1.20
Agoraphobia										
Saunders et al. (1992)	Community	391	—	2.03	5.19	—	3.31	—	0.61	1.57
Alcohol dependence										
Fergusson et al. (1996b)	Community	1019	—	2.19	2.02	—	2.12	—	1.03	0.95
Kendler et al. (2000)	Community	1411	—	2.29	3.43	—	2.71	—	0.85	1.27
Drug dependence										
Fergusson et al. (1996b)	Community	1019	—	1.64	3.66	—	2.53	—	0.65	1.45
Kendler et al. (2000)	Community	1411	—	2.03	5.19	—	3.20	—	0.63	1.62
Suicide										
Saunders et al. (1992)	Community	391	—	2.74	3.11	—	2.89	—	0.95	1.08
Fergusson et al. (1996b)	Community	1019	—	2.15	3.43	—	2.71	—	0.79	1.27
Mean									0.78	1.29

— No data.

[a] RR_1, RR_2, RR_3 refers to RRs for level 1 exposure (non-contact), level 2 (contact) and level 3 (intercourse).

Note: 95% CIs were calculated but are not presented here. All estimates are for females or all persons. None are for males.

risk estimates for the narrow category of abuse. Ratios from studies reporting risk estimates in the non-contact, contact and intercourse categories were applied to the risk estimates for the broad category of abuse. This process generated relative risks for the three categories of abuse required for analysis in relation to the single estimate reported by each study. In this way, the extrapolated relative risks reflect an approximation of what the risk may have been if each study had reported risks for levels of exposure, rather than only risks for exposure vs non-exposure. This ensured that data from studies reporting risk in terms of exposed vs non-exposed, rather than risk by levels of exposure, could still be included in the analysis.

The accuracy of the extrapolated relative risks relies on the premise that risk increases with level of exposure to the same degree across

studies. It was decided, however, that the error introduced by the extrapolation process was less than the error introduced by pooling relative risk estimates from a small number of studies. After the extrapolation process each study that reported an estimate only for the broad category of abuse now had relative risks for the non-contact, contact and intercourse categories of abuse. Accordingly, each study that reported an estimate for the narrow category of abuse now had a relative risk for the contact and intercourse categories of abuse.

In order to calculate confidence intervals for those relative risks that had been extrapolated, standard errors had to be estimated. In this instance, the standard errors derived for the relative risks for the broad and narrow categories of abuse were used. This may not accurately reflect—indeed may underestimate—the true variance around the extrapolated estimates, but for the purposes of this analysis it was assumed to be a reasonable approximation.

Adjustment for confounders

There is strong evidence within the literature that child sexual abuse is often comorbid with other forms of child abuse and also that child abuse occurs within the context of other family dysfunction (Fergusson and Mullen 1999). Given this, any studies that do not control for these other childhood adversities may inflate the contribution child sexual abuse makes to the onset of our chosen outcomes. Only 13 studies in the data set controlled for confounders. In order to adjust uncontrolled estimates for the potential contribution of confounders a method proposed by Rothman and Greenland (1998) was utilized.

Each of the 13 studies varied in terms of outcomes measured and confounders controlled for. For each study the confounders were recorded and grouped into four categories: sociodemographic, other psychopathology, other abuse and family dysfunction. The studies varied according to the types of confounders controlled for and the measures used. In particular, the category of family dysfunction represented a wide variety of factors. The measures used varied from questionnaires concerning parental attachment and parent/child bonding through to those measuring markers of dysfunction, such as whether the subject grew up in a nuclear family or whether there was parental psychopathology. While the measures varied greatly it was assumed that a common underlying dimension was being measured, that is, the degree to which the family environment was impoverished or dysfunctional and so they were grouped together.

At this stage five studies were excluded from the analysis, as they were not consistent with the pattern of confounders measured by the other seven studies. Saunders et al. (1999), Molnar et al. (1998) and Wilsnack et al. (1997) were excluded since they only controlled for sociodemographic variables and might dilute the adjustment factor if they contributed to the average estimate. Stein et al. (1988) was excluded since

it did not control for any childhood adversity, instead only controlling for subsequent adult sexual abuse. Spak et al. (1998) controlled for childhood behavioural difficulties and childhood psychopathology. These may be important factors confounding the relationship between CSA and alcohol dependence, but, it is unclear to what extent childhood behavioural difficulties and childhood psychopathology are additive to the effects of other confounders. It was therefore excluded.

The eight studies left in the analysis controlled for family dysfunction or other types of abuse, including physical and emotional abuse. Therefore the adjustment factor derived from these studies reflects an adjustment for childhood adversity stemming from dysfunctional home and family environments and is the confound identified as being most important in the literature (Chandler and Jackson 1997; Fergusson and Mullen 1999).

For each of the eight studies adjusted odds ratios and unadjusted odds ratios could be derived. Using the odds ratios an adjustment factor was calculated using the following formula:

$$U = OR_u / OR_a$$

OR_u represents the unadjusted odds ratio, OR_a represents the odds ratio adjusted for confounders and U is the bias produced from failure to control for the confounders. Since there were only eight studies some assumptions about the commonality of effect across sex, disorder categories and abuse categories had to be made. Table 23.9 presents the final adjustment factors by study and disorder averaged across abuse category and sex.

Extrapolation across sex

Of the eight studies, seven provided estimates for females but only two provided estimates for males. One study provided estimates for males and females combined. Of the two studies that presented data for both males and females there was no clear pattern of differences between the sexes. Additionally, there is no theoretical reason to expect that the confounders in question would differentially affect the relationship between exposure to CSA and disorder; and due to the paucity of data for males we assumed no difference.

Extrapolation across levels of exposure

With the exception of three studies, all of the studies provided estimates for only the narrow or broad categories of abuse. In the absence of any data it was assumed that the effects of confounders would be the same across abuse categories.

Table 23.9 Adjustment factors for family dysfunction according to disorder type and study

		Adjustment factor (U)						
Study	*Sample type*	*Depression*	*Panic*	*Drug*	*Alcohol*	*PTSD*	*Disorder mean*	*Suicide*
Molnar et al. (2001)[a]	Community	1.68	1.69	1.84	1.55	1.53	1.62	—
Kendler et al. (2000)[b]	Community	1.21	1.02	1.19	0.92	—	1.08	—
Fergusson et al. (1996b)[b]	Community	1.43	—	0.86	1.06	—	1.12	1.64
Mullen et al. (1996)[a]	Community	2.11	—	—	—	—	2.11	5.48
Zuravin and Fontanella (1999)[a]	Low income mothers	1.28	—	—	—	—	1.28	—
Mullen et al. (1993)[a]	Community	—	—	—	—	—	—	2.39
Yama et al. (1995)[a]	College	—	—	—	—	—	—	1.71
Borowsky et al. (1999)[b]	American Indian and Alaska Native youth	—	—	—	—	—	—	2.08
Mean		1.54	1.35	1.29	1.18	1.53	1.39[c]	2.66

— No data.

[a] Controlled for both other abuse and family factors.

[b] Controlled only for family factors.

[c] Excludes suicide.

Extrapolation across outcomes

Most of the eight studies in the analysis only provided estimates for one or two disorders. There were three studies that gave estimates for most of the disorders, excluding suicide attempts; and within these studies there was no definitive variation across disorder. This conclusion is again limited by paucity of data, but there is no theoretical reason for the confounders to act differentially according to disorder. Suicide attempts appear to be an exception since when the means across disorder and study are compared the adjustment factor for suicide is higher. This may indicate that the confounding variables are more predictive of suicide attempts than the other disorders considered and hence, to be conservative, a different adjustment factor is applied to the suicide estimates.

Generalizability of the adjustment factor

The validity of adjusting estimates from uncontrolled studies using an adjustment factor derived from studies that do control for confounders will be accurate only to the extent that the confounding effects of the covariates are similar across both the controlled and uncontrolled studies (Greenland 1987). While there is no way to assess this issue quantitatively, we can consider the samples from which our adjustment factors were derived. If the samples from controlled studies are drawn from significantly different groups within the community then generalizability to the uncontrolled studies may be limited. In the current analysis the controlled samples included four community and one college sample that were representative of the samples from uncontrolled studies. Moreover, the two samples from community subgroups, low-income mothers, and American Indian and Alaska Native youth, provided estimates that are comparable to the community estimates.

Application of adjustment factors

Adjustment factors were applied differentially across the risk estimates according to several criteria. For those eight studies reporting both adjusted and unadjusted risk estimates from which an adjustment factor could be calculated, its own individual adjustment factor was applied to each. For those studies that reported no adjusted estimates and from which an adjustment factor could not be derived, the average of the adjustment factors was applied. The exception was where the unadjusted relative risks were non-significant, and in this instance the relative risks were not adjusted for the presence of confounders. This ensured that a significant protective relationship between CSA and psychiatric outcome was not created artificially.

Meta-analysis

The relative risks were combined using meta-analysis with STATA Intercooled 7. For ease of calculation the macro "meta" was utilized (Sharp and Sterne 1997). Estimates were grouped according to psychiatric outcome and then combined. In most cases the studies combined within each group were significantly heterogeneous according to Cochrane's Q statistic, indicating that moderator variables other than psychiatric outcome were still accounting for significant variation. However the small number of studies prevented further partitioning according to other hypothesized moderator variables.

The presence of significant, unexplained heterogeneity generally indicates preference for a random-effects model to take into account the between-study heterogeneity (Cooper and Hedges 1994; Rothman and Greenland 1998). However, when only two or three studies are available for combination, the between-study variance is estimated with poor precision (Cooper and Hedges 1994). In this instance it was decided that

Table 23.10 Number of studies contributing to each estimate within disorder category

	No. of studies		
Outcome	*Non-contact*	*Contact*	*Intercourse*
Depression	14	23	25
Panic disorder	5	8	8
Alcohol abuse/dependence	7	13	15
Drug abuse/dependence	4	7	7
PTSD	5	6	8
Suicide attempts	13	29	29

groups with five or more studies would be combined using a random-effects model and those with less than five would use a fixed-effects model.

3.4 Assessment of causality for each outcome

The following summarizes the evidence of a causal relationship between CSA and each of the seven outcomes examined. A schema or hierarchy was developed to organize this evidence and is outlined below. All of the studies that reported relevant data on proportions of persons in the clinical ranges for each outcome have been tabulated by level of evidence. For some, the χ^2 statistic was calculated to test for significance of the odds ratios where these were not available from the studies themselves. Where research on a given psychiatric outcome focused on a subsample of a study group that has been described previously, the findings of the main sample are reported.

Level 1: Studies controlling for both genetic background and family environment.

Level 2: Prospective studies where family environment measured prospectively was used to control confounding of deprivation and CSA.

Level 3: Retrospective cross-sectional studies in which the occurrence of CSA was determined at the time illness was ascertained. Family environment measured reliably was used to control confounding of deprivation and CSA.

Level 3a: Representative community samples: samples of adolescents or adults where either sampling strategy or weighting procedures ensured representativeness of sample.

Level 3b: Non-representative community samples: samples of adolescents or adults where methodology did not necessarily ensure representativeness of sample.

Level 3c: Community subgroup samples: samples of college students, general practice attendees or other community subgroups.

Level 4: Prospective case–control studies in which a CSA group was compared with controls matched for family environment (actually or statistically) and followed over time to measure the onset of mental disorders.

Level 5: Single wave case–control studies in which a CSA group was compared with controls matched for family environment (actually or statistically).

Level 6: Studies of special groups such as foster care children, street youth and juvenile detainees.

Depression

The most convincing evidence for a relationship between child sexual abuse and adult depression is from three level 1 studies of adult twins. Dinwiddie et al. (2000) adjusted for demographic factors and found significant relationships between contact or intercourse CSA and depression for female (OR = 2.20) and male twins (OR = 3.93). Restricting the sample to twin pairs discordant for CSA (and thereby substantially reducing the power of the analysis), the relationship between CSA and depression was no longer statistically significant for either males or females even though both were at increased risk for depression.

Kendler et al. (2000) adjusted for family functioning and parental psychopathology in a sample of female twins. They found that odds ratios for the risk of depression in abused twins compared to non-abused twins were modest but not significant for non-genital abuse (OR = 1.08), but increased with the severity of the abuse. For abuse involving genital contact (OR = 1.58) and intercourse (OR = 2.79), odds ratios were significant. When analyses were restricted to twins discordant for CSA, odds ratios were of similar magnitude to the previous analysis but only risk in the intercourse category of abuse remained statistically significant (Kendler et al. 2000). It was noted by the author that the discordant twin analyses were limited by small sample size.

In a sample consisting of monozygotic and dizygotic twins who were discordant for CSA, the risk of developing major depression was 1.68 for women and 1.25 in men; however, this relationship was significant only for women (Nelson et al. 2002). Of note was the non-significant trend for non-abused co-twins to also be at greater risk of having a history of depression in comparison to twin pairs with no history of abuse, providing evidence for the contribution of familial factors to the onset of disorder. Therefore, the finding that abused co-twins have a higher risk of depression than their non-abused co-twins demonstrates the increased risk that CSA contributes over and above family background.

Across the three twin studies a significant relationship between CSA and depression has been found. Particularly, abuse involving contact and penetration has been found to significantly increase the risk of a depressive disorder. Discordant twin analyses were conducted for three studies and have the advantage of controlling more tightly for familial and genetic factors. These analyses also found CSA to contribute significantly to onset of depression, but in the case of Dinwiddie et al. (2000) and Kendler et al. (2000) only when the abuse involved intercourse. While the small sample sizes of the discordant twins restricted the power of the analyses they do provide strong support for a causal relationship between depression and CSA involving intercourse.

To conclude, there have been three level 1, and 19 level 2 and 3 studies examining the relationship between CSA and depression; 19 of them supported a significant relationship. There is strong, consistent evidence that, after adjustment for confounders, there is a significant relationship between CSA and depression in adults, particularly for women who experienced more severe forms of abuse.

Panic disorder

There is evidence from level 1 twin studies for a relationship between CSA and panic disorder. Defining sexual abuse as contact or intercourse, Dinwiddie et al. (2000) adjusted for demographic factors and found significant relationships between CSA and panic disorder for female (OR = 3.54) and male twins (OR = 5.02). In the twin pairs discordant for CSA, the relationship between CSA and panic disorder was no longer significant for women even though they were at increased risk for the disorder (OR = 2.00). Similar analyses were unable to be conducted for men because of sample size.

In their analyses of panic disorder, Kendler et al. (2000) adjusted for family functioning (but not parental psychopathology) and found that odds ratios in abused twins compared to non-abused twins were modest but not significant for either non-genital abuse (OR = 1.25) or for abuse involving genital contact (OR = 1.92). Odds ratios for intercourse (OR = 2.62) were significant. Discordant co-twin analyses were not performed due to small sample sizes. Prospective studies have found equivocal evidence of a relationship (Brown and Harris 1993; Ernst et al. 1993) and studies using community samples have found significant associations between some forms of CSA but not others (Molnar et al. 2001; Saunders et al. 1992; Stein et al. 1988). Although few studies have examined panic disorder as an outcome of CSA, there is evidence from two level 1 studies, one level 2 study and three level 3 studies that the rates of panic disorder are increased in sexually abused adults and are more strongly predicted by abuse involving penetration.

Obsessive–compulsive disorder (OCD)

Only three studies explored the relationship between CSA and OCD and only one of these controlled for confounders. This representative community sample (level 3a) adjusted for demographics and other abuse but found no significant relationships between CSA and lifetime or 6-month history of OCD (Stein et al. 1988). Results from the two samples that did not adjust for confounders were mixed. In a sample of female college students (level 3c) (Arata 1999), no significant relationships were reported between CSA and lifetime or 1-month history of OCD. However, childhood was defined as aged <14 years and was therefore quite restrictive. Saunders et al. (1992) (level 3b) defined childhood more broadly, as aged <18 years. While results for non-contact abuse were not significant, women were 4.5 times more likely to have a lifetime history of OCD if they had experienced CSA in the form of contact abuse and over six times more likely if they had experienced intercourse; and these findings were significant. This pattern of significant results was also the case for the relationship between CSA and women who met the diagnostic criteria for OCD at interview. In summary, only one study supported a relationship between CSA and OCD. Further research is required in order to confirm this association. For this reason the risk for OCD will not be calculated in this report.

Post-traumatic stress disorder (PTSD)

There was one study of PTSD that provided level 2 evidence (Silverman et al. 1996). This was a 17-year follow-up of a community sample of children in a working class area. There was no adjustment for confounders; however, a significant relationship was found between CSA and lifetime history of PTSD for females at age 21 years.

Of the remaining studies of PTSD, all were from level 3 evidence and only two of these adjusted for confounders. These studies were representative community samples (level 3a). One study restricted CSA to intercourse only, and only adjusted for age, but found that CSA significantly increased the risk of lifetime (OR = 3.42) and recent (OR = 3.17) PTSD (Saunders et al. 1999). The other study adjusted for demographic and family variables such as parental substance abuse and psychopathology, and the presence of physical abuse (Molnar et al. 2001). Child sexual abuse was defined as contact sexual abuse or intercourse and was found to be significantly related to lifetime history of PTSD in women (OR = 10.2) and men (OR = 5.3). A notable finding was that the risk of PTSD was significantly higher for penetrative abuse than contact abuse.

In summary, there were eight level 2 and 3 studies; all showed a significant relationship between child sexual abuse and adult PTSD. There is strong, consistent evidence that, after adjustment for confounders, there is a significant relationship between CSA and PTSD in adults, particularly for those who have experienced more severe forms of abuse.

Alcohol abuse or dependence

The evidence for a relationship between childhood sexual abuse and adult alcohol abuse/dependence is from the three level 1 studies of adult twins. Dinwiddie et al. (2000) adjusted for demographic factors and found significant relationships between contact or intercourse CSA and alcohol abuse/dependence for female (OR = 2.81) and male twins (OR = 1.91). Restricting the sample to twin pairs discordant for CSA, the relationship between CSA and alcohol abuse/dependence was no longer significant for either males or females; however females remained at increased risk (OR = 2.50).

Kendler et al. (2000) adjusted for family functioning and parental psychopathology in a sample of female twins. The odds ratios for the risk of alcohol abuse/dependence in abused twins compared to non-abused twins were modest but not significant for genital contact abuse (OR = 1.91). For non-genital abuse (OR = 3.20) and intercourse (OR = 6.48), odds ratios were significant. Analyses for twins discordant for CSA, or where the co-twin had experienced a less severe form of abuse, showed that intercourse significantly increased the risk of alcohol abuse/dependence. In another sample that also presented results for CSA among discordant twins the risk of developing alcohol abuse/dependence was 1.73 for women and 1.25 in men; however this relationship was significant only for women (Nelson et al. 2002). In particular, the findings from the discordant twin analyses suggest a causal relationship between CSA and alcohol abuse/dependence, and indicate that CSA increases the risk over and above that arising from family background. Low sample size of the discordant twin analyses may have produced the non-significant results for contact forms of abuse but the significant finding for intercourse in spite of low power makes this finding notable. Other prospective and community studies also provide evidence for a relationship between CSA and alcohol abuse/dependence with two out of three level 2 studies and seven out of nine level 3 studies presenting significant odds ratios.

Drug abuse or dependence

There was only one level 1 study that explored drug abuse or dependence. In their study of female twins, Kendler et al. (2000) adjusted for family functioning and parental psychopathology. They found that odds ratios for the risk of drug abuse/dependence in abused twins compared to non-abused twins were modest but not significant for genital contact abuse (OR = 1.21). For non-genital abuse (OR = 3.57) and intercourse (OR = 6.55), odds ratios were significant. Analyses of twins discordant for CSA, or where the co-twin had experienced a less severe form of abuse, showed a similar pattern of results with non-genital CSA and CSA involving intercourse placing subjects at increased risk (ORs 4.29 and 2.85, respectively). However, these results were not significant, and this is likely to be a function of small sample sizes.

Of the three level 2 studies, only one showed a significant association and was significant only for the intercourse category of abuse (OR = 5.1) (Fergusson et al. 1996b). The two other prospective studies failed to show a relationship (Silverman et al. 1996; Widom and White 1997). Of the four level 3 studies using community samples, three demonstrated a significant association but not across all three levels of abuse. In summary, there is evidence to suggest a relationship between CSA and drug dependence but only for the more severe forms of abuse. However, the evidence is more equivocal compared to other outcomes and more research is required to confirm this relationship.

Suicide attempts

There were two level 1 studies of adult twins that addressed suicide attempts. In the first, Dinwiddie et al. (2000) adjusted for demographic factors and found significant relationships between contact or intercourse CSA and serious suicide attempts for female (OR = 7.74) and male twins (OR = 7.07). Restricting the sample to twin pairs discordant for CSA, the relationship between CSA and suicide attempts was no longer significant for females; however they were still at increased risk (OR = 2.33). Risk estimates could not be computed for males in the discordant twin analysis due to small numbers. In the second study of twins who were discordant for CSA (Nelson et al. 2002) the risk of suicide attempts was 2.33 for women and 4.50 in men. This relationship was significant for women, and almost reached significance in men (95% CI 0.97–20.83), even after adjustment for demographic and family factors. Furthermore, three of four prospective studies (level 2) and 16 of 18 level 3 studies also found significant associations between CSA and suicide attempts. There is strong evidence for a relationship between CSA and suicide attempts.

Completed suicide

Completed suicide, by definition, can only be studied prospectively. Only one study to date has examined the relationship between completed suicide and child sexual abuse. This Australian study was based on a relatively small sample of sexually abused young people, the majority of them female, who presented to hospitals for the abuse, and a control group of non-abused young people from the community (Plunkett et al. 2001). Without controlling for confounders, CSA was found to increase the risk of completed suicide; and the rate was found to be very high, 179.5 per 100 000 person-years, or 1.8%, but this relationship was not significant. It should be noted that the number of completed suicides was 3 out of a sample of 259, and none of the controls had committed suicide, so analyses were somewhat limited. The national suicide death rates for 15–24 year olds during the same time period ranged from 13.8 to 16.7, so the observed rate in the study was 10.7 to 13.0 times that of the Australian national suicide death rate (Dudley et al. 1998).

Relationship between attempted and completed suicide

Attempted suicide has consistently been shown to be a strong predictor of completed suicide (see Graham et al. 2000 for review). Estimates of the magnitude of this risk however, vary considerably. The prevalence of completed suicides among those who have attempted has been estimated at 1% to 19% in the 12 months after the attempt (Diekstra 1992; Graham et al. 2000), 2.8% after 8 years (Hawton and Fagg 1988) and 10% after 10 years (Tejedor et al. 1999). Lifetime prevalence of completed suicide in those who have ever been hospitalized for suicidality has been estimated at 8.6% (Bostwick and Pankratz 2000). Although difficult to quantify on the basis of available data, the evidence suggests that approximately 1 in 10 individuals who attempt suicide will die by suicide at some point following the attempt. These reviews also pointed out, however, that the majority of completed suicides are not preceded by a suicide attempt (Graham et al. 2000). Estimates of risk for completed suicide that are derived only from estimates of previous attempts are therefore likely to underestimate the risk of completed suicide.

The issue is further complicated in that mental disorders have also been shown to be strong and consistent predictors of both suicide attempts and completed suicide (Brent et al. 1999; Graham et al. 2000; Harris and Barraclough 1997; Hawton and Fagg 1988; Kessler et al. 1999). Within this context, the exact nature of the relationship between CSA, mental disorder, suicide attempts and completed suicide is likely to be complex (Beautrais 2000). Unfortunately, none of the studies in the present report that examined the relationship between CSA and suicide attempts controlled for concurrent psychopathology.

From the above it is concluded that there is evidence of a relationship between attempted suicide and completed suicide. However, for the purposes of the current analysis it is necessary to determine whether a person who has been subject to CSA has any higher or lower chance of completing suicide conditional on having attempted it. To our knowledge these data do not exist. In the absence of these data the relative risk for suicide attempts will be used as proxies for the relative risk for completed suicide. This assumes a constant relationship between attempted and completed suicide given exposure to CSA. The extent to which this underestimates or overestimates the relative risk is unknown.

3.5 Estimates of risk factor–disease relationships by age, sex and subregion

Estimates of risk

Table 23.11 summarizes the results of the meta-analysis and reports the relative risks for the psychiatric outcomes examined. No estimates are available for different age and sex groups. All studies were from AMR-A, WPR-A or EUR-A. No studies were available from other subregions that may have different cultural norms and socioeconomic

Table 23.11 Unadjusted and adjusted relative risks across disorder and abuse categories

	Non-contact abuse				*Contact abuse*				*Intercourse*			
	Adjusted[a]		*Unadjusted*		*Adjusted*[a]		*Unadjusted*		*Adjusted*[a]		*Unadjusted*	
	Pooled RR	*95% CI*	*Pooled RR*	*95% CI*	*Pooled RR*	*95% CI*	*Pooled RR*	*95% CI*	*Pooled RR*	*95% CI*	*Pooled RR*	*95% CI*
Depression	1.06	0.91–1.24	1.37	1.16–1.61	1.32	1.16–1.51	1.80	1.61–2.02	2.04	1.78–2.35	2.80	2.44–3.22
Panic disorder	1.01	0.76–1.35	1.52	1.13–2.04	1.64	1.12–2.42	2.27	1.56–3.32	2.60	1.70–3.97	3.58	2.34–5.48
Alcohol abuse/dependence	1.19	0.85–1.67	1.35	0.91–2.02	1.32	1.07–1.63	1.61	1.24–2.09	1.87	1.47–2.39	2.58	2.04–3.24
Drug abuse/dependence	1.03[b]	0.84–1.26	1.57[b]	1.28–1.93	1.31	0.90–1.91	1.68	1.03–2.72	2.40	1.46–3.96	3.04	1.83–5.04
PTSD	1.95	0.95–3.98	2.70	1.32–5.54	2.95	1.53–5.68	4.10	2.12–7.90	4.48	2.33–8.65	6.23	3.23–12.02
Suicide attempts	1.02	0.71–1.45	2.80	1.89–4.15	1.32	1.08–1.60	3.25	2.53–4.18	2.21	1.77–2.76	5.56	4.32–7.16

[a] These relative risks have been adjusted for family dysfunction and other types of abuse. The RRs for suicide attempts have been divided by 2.66 and the RRs for the mental disorders have been adjusted by 1.39.

[b] Fixed-effects model used to combine estimates due to small number of studies available.

circumstances. No differences in risk were assumed across sex and age breakdowns since numbers were few and many studies used lifetime diagnoses.

On the whole several conclusions can be drawn from these results. First, the relative risks were not significantly different across types of mental disorder, suggesting that CSA is not particularly associated with any one disorder. Rather the risk appears to be pervasive across the whole spectrum of mental disorders examined. This lack of specificity makes CSA particularly damaging, putting individuals at risk for a wide range of mental disorders.

Second, with the exception of suicide attempts, risks did not vary significantly across categories of abuse. This may reflect the small number of studies available for analysis, as there is a general trend for increased risk to be associated with "increased" exposure to CSA. That is, as more severe forms of CSA are experienced the risks for developing a mental disorder increase. This may indicate that those exposed to CSA do not represent a homogeneous group but instead reflect a group that varies in terms of exposure and subsequent risk for psychiatric disorder (Fergusson and Mullen 1999). However, this finding is largely an artefact of the extrapolation process. Further research will enable this "dose–response relationship" to be confirmed.

Non-contact abuse was not a significant predictor of risk after external adjustment for confounders. Non-contact abuse may constitute a more heterogeneous category of abuse compared to the contact and intercourse categories. Certainly, in the studies that contributed to this analysis the category of non-contact abuse encompassed a variety of acts. Such heterogeneity may make the results of the analysis hard to interpret. The question is whether non-contact does not place individuals at increased risk for disorders or whether some non-contact forms of abuse are more or less harmful than others. Cultural factors are likely to play a role in this and further investigation is required. Across the majority of disorders (excluding drug abuse/dependence) the relative risks for contact and intercourse forms of abuse remained significant after external adjustment. These results are more easily interpreted as the two categories are a more homogeneous group of acts and can be more tightly defined.

Results of the current meta-analysis were consistent with the only other review to look systematically at psychiatric diagnosis as an outcome of CSA. Fergusson and Mullen (1999) collated and reanalysed data from 12 studies reporting on the relationship between CSA and psychiatric dysfunction. While Fergusson and Mullen (1999) did not statistically combine the estimates, consistent and pervasive relationships between CSA and adult psychopathology were apparent. Other meta-analyses have been conducted in this area (Jumper 1995; Paolucci et al. 2001; Rind 1997; Rind et al. 1998) but have focused on continuous measures of adjustment. Jumper (1995) and Paolucci et al. (2001) have com-

mented on the difficulty in quantifying the impact that confounders have since many large-scale community studies do not measure them. Rind et al. (1998) was the only meta-analysis to control for the confounding effect of family environment using college samples since confounds are more likely to be measured in studies using these samples. Results of their analysis showed that the relationship between CSA and adjustment in adulthood disappeared after controlling for family environment. Adjustment in their analysis refers to the various psychological correlates of CSA measured in the studies that contributed to their analysis. Eighteen categories of psychological correlates were coded from the studies and included anxiety and depressive symptoms, plus broader areas of adjustment such as sexual adjustment and social adjustment. When commenting on Rind et al. (1998) results, Kendler et al. (2000) stated that CSA appears to be more related to lifetime psychiatric disorder rather than cross-sectional measures of adjustment which focus solely on current well-being. Certainly, the results of the current meta-analysis do suggest a significant relationship between CSA and psychiatric disorder after adjustment for important confounders.

Several caveats should be mentioned in regard to the current analysis. The external method of adjustment used was limited by the methodology of the studies from which the adjustment factors were derived. Across the studies family dysfunction was not defined or measured consistently and the measures that were used may not be reliable. It should also be noted that current research methodologies make it impossible to determine whether any antecedent signs or symptoms of psychiatric disorders mean that the child is more vulnerable to abuse, thus inflating the risk factor–disease relationship. Additionally, the majority of studies contributing to this analysis were conducted during a time when CSA was ignored and the victims were stigmatized. While no cohort effects were found for reporting episodes of CSA the stigmatization could possibly have had an impact on the degree to which people were affected.

To conclude, it appears that CSA is particularly damaging with effects evident over and above other forms of childhood adversity. Contact and intercourse categories of abuse remained significant after controlling for confounders but the category of non-contact was non-significant. Further research is required to understand the effects of various forms of non-contact abuse.

Risk reversibility

By definition, exposure to CSA stops at the age of 17 years. The reversibility of risk therefore refers simply to decline in the risk of developing mental disorder given exposure to CSA over the lifespan. Problems in quantifying the decline arise from both paucity of data and competing theoretical arguments. There are two arguments. Either assume that risk remains constant throughout the lifespan or assume that

risk subsides over time and eventually becomes that of people who were never abused. One can argue that risk remains constant over time as there is evidence to suggest that CSA alters your life trajectory such that you are more likely to experience problems with relationships, self-esteem and sexual adjustment (Mullen et al. 2000). These problems in themselves are associated with mental disorder and so potentially mediate the relationship between CSA and disorder in later life. Therefore, those exposed to CSA remain at increased risk for mental disorder compared to those who have not been exposed. Alternatively one can argue that risk decreases over the lifespan. As a person moves away from a traumatic life event, its power to inflict psychological harm is lessened and so those exposed to CSA eventually have the same risk for mental disorders as those not exposed.

The data collected for this report do not inform either argument further. Preliminary analyses divided the relative risks into two age groups, 15–29 and 30–44 years. Confidence intervals overlapped suggesting no significant difference between the two age groups. Two explanations can be given. First, the numbers in the groups were small limiting the robustness of the estimates. Second, most studies reported estimates of risk for lifetime mental disorders thereby preventing any clear distinction in onset of mental disorders between the two age groups from being made. In the absence of data we have assumed that risk remains constant over the lifespan, as it becomes mere speculation if one tries to estimate the amount risk reduces over time. In order to better inform this decision, data on risk for current disorder are required, broken down into appropriate age groups. Further analyses of the large community and prospective samples are required to address this.

3.6 Reasons and implications for extrapolation of risk factor–disease relationships from one subregion to another

This represents perhaps the biggest threat to validity to the present study. The studies in the risk analysis were overrepresented by samples from Australia, Canada, New Zealand, some European countries and the United States of America. The prevalence of psychiatric disorders varies from country to country and this variability represents the myriad social, economic and cultural factors that interplay with the development of disorder. The degree to which these same factors mediate the relationship between CSA and mental disorder cannot be quantified and is difficult to even speculate on. The answer to this is not likely to be reached in the near future as extensive research is required before sound conclusions can be drawn. The paucity of data and theoretical complexity necessitated the assumption that the relative risks remain constant across subregions.

3.7 Quantitative and qualitative sources of uncertainty

Uncertainty in the current analysis came from several sources. Meta-analysis was used as a method of quantifying the quantitative uncertainty around the final prevalence estimates taking into account sample size or variability between studies, whichever is appropriate given the homogeneity of the estimates being combined.

The methods of extrapolation also introduce uncertainty into the analysis. In particular, the extrapolation conducted to get relative risks for the three levels of exposure will have introduced error. The ratios used for extrapolation came from only five studies and the relationship between the relative risks for each level of exposure may vary according to the population studied.

The external method employed to adjust for confounders introduces three sources of uncertainty. First, the instruments used to measure the confounders may have variable reliability. Second, the 12 studies that contributed to the adjustment factor all measured family dysfunction differently thereby creating another source of uncertainty. Third, the ability of the adjustment factor to be generalized to other studies conducted on different populations can be questioned. It is possible that confounders may mediate the relationship between CSA and outcomes differentially depending upon the population studied. Not enough studies that controlled for confounds were available to see if the adjustment factor varied in any systematic way.

Additionally, while all estimates came from AMR-A, WPR-A and EUR-A not all countries from those subregions were represented. Inter-country variations within subregions are possible and may affect the generalizability of our relative risk estimates. Even greater uncertainty is introduced when estimates from one subregion are extrapolated to another, a decision necessitated by the paucity of data from other subregions.

4. Results and discussion

The discussion of the estimates of the burden in mental disorder outcome that is attributable to CSA will focus on differences between mental disorders, followed by an examination of sex, age and subregional differences. Across the world, CSA contributed to between 4% and 5% of the disability-adjusted life years (DALYs) in males and between 7% and 8% of the DALYs in females for each of depression, alcohol abuse/dependence and drug abuse/dependence. The attributable fractions were higher for panic disorder (7% for males and 13% for females) and higher still for PTSD (21% for males and 33% for females). For suicide attempts the attributable fractions were 6% for males and 11% for females. As discussed above, the confidence intervals around the relative risks of each of the mental disorders overlap and thus the apparent differences may not be real. The same applies to the attributable fractions.

On the whole CSA contributes to a higher percentage of DALYs for females than for males. This difference is driven by the difference in the prevalence of CSA for females and males. There are slight subregional variations in the attributable fractions. In particular, AFR-E and SEAR-D have higher attributable fractions than the other subregions. This is a function of the higher prevalence rates for these subregions. However, data for these subregions came from a few studies that were poor methodologically.

The number of DALYs attributable to CSA varies as this is a function of both the attributable fractions and the amount of burden of disease accounted for by psychiatric disorders in the various subregional groups. One pattern is evident, however: the number of DALYs are greater in the younger age groups and decline in the older age groups. Since risk was assumed to be constant across age, this merely reflected the distribution of DALYs for mental disorders, which impact largely in the younger age groups due to their early onset and chronic nature.

5. Methods for projection of exposure forward

Estimates of avoidable burden, the amount of burden that could be prevented if exposure to CSA was curtailed, are dependent upon the expected prevalence of CSA in the future. As outlined above, we have assumed that CSA will not vary over time. The reviews conducted on cohort effects have provided equivocal evidence (Bagley 1990, 1995; Bagley and Ramsay 1985; Bickerton et al. 1991; Feldman et al. 1991; Fergusson et al. 2000). Three reviews concluded that the prevalence of CSA could increase over time while three reviews also conclude that there is no evidence to support a change in prevalence over time. Analysis of this phenomenon in our own data set also provided no evidence of a change in prevalence over time. Additionally, it is difficult to speculate on what future factors may arise to influence prevalence. Therefore, the estimates of avoidable burden should be the same as those for attributable burden.

Acknowledgements

Steering Committee: Majid Ezzati, David Fergusson, Bronwyn Gould, Beth Kotze, Ron Kessler, Alan Lopez, Michael Lynskey, Judy Martin, Paul Mullen, Anthony Rodgers, Bedirhan Üstün.

Note

1 See preface for an explanation of this term.

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