Childhood adversity and adult psychiatric disorder in the US National Comorbidity Survey

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ABSTRACT

Background. Survey data are presented on the associations between retrospectively reported childhood adversities and subsequent onset and persistence of DSM-III-R disorders.

Methods. Data come from the US National Comorbidity Survey, a large survey of the US household population.

Results. Twenty-six adversities were considered, including loss events (e.g. parental divorce), parental psychopathologies (e.g. maternal depression), interpersonal traumas (e.g. rape) and other adversities (e.g. natural disaster). These adversities were consistently associated with onset, but not persistence, of DSM-III-R mood disorders, anxiety disorders, addictive disorders and acting out disorders. Most bivariate associations with onset attenuated in models that controlled for clustering of adversities and for lifetime co-morbidities among psychiatric disorders. Multivariate effects of adversities in logistic models were additive, which means that they have multiplicative effects on probability of disorder onset. Adversities showed little specificity. An analysis of time decay showed that the effects of childhood adversities on disorder onset persist beyond childhood.

Conclusions. The existence of strong clustering among childhood adversities and lifetime co-morbidity among adult disorders means that caution is needed in interpreting the results of previous single-adversity single-disorder studies as documenting unique effects of specific childhood adversities on specific adult disorders. Future studies need to assess a broader range of adversities and disorders and to explore the existence and effects of commonly occurring adversity clusters. Replication is needed to verify that the effects of childhood adversities are mostly on first onset rather than on the creation of vulnerabilities that lead to increased risk of persistence.

INTRODUCTION

Retrospective studies in both treatment samples (e.g. Paris *et al*. 1994; Buist & Barnett, 1995; Mancini *et al*. 1995; Sullivan *et al*. 1995; Portegijs *et al*. 1996) and community samples (e.g. Rodgers, 1994; Oakley-Browne *et al*. 1995*a*; Romans *et al*. 1995; Mullen *et al*. 1996; Stein *et al*. 1996) consistently find that adults who suffer from current psychiatric disorders are significantly more likely than others to report exposure to childhood adversities. These studies are limited by the fact that the retrospective reports on which they are based are subject to recall bias. However, these studies can nonetheless be useful in providing preliminary information to target modifiable risk factors for experimental interventions and to narrow the range of issues examined in subsequent naturalistic prospective studies.

The usefulness of retrospective studies has been compromised, however, by three limitations. First, many of these studies focus on only a single adversity. For example, there have been separate studies of the effects of poor maternal care (e.g. Plantes *et al*. 1988), parental divorce

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(e.g. Rodgers, 1994), childhood sexual abuse (e.g. Romans *et al.* 1995) and parental substance abuse (e.g. Velleman & Orford, 1993). We know very little about how the effects of these adversities combine to promote adult psycho-pathology or whether some aspects of adversity explain others in multivariate analyses. The few studies that have investigated these issues conclude that childhood adversities often occur in clusters, making it difficult to pinpoint any one particular adversity as the critical determinant of subsequent adult disorders (Romans *et al.* 1993; Mullen *et al.* 1996; Portegijs *et al.* 1996).

Secondly, many retrospective studies focus on only a single adult psychiatric disorder, usually depression (e.g. Brown & Moran, 1994; Rodgers, 1994; Kunugi *et al.* 1995; Oakley-Browne *et al.* 1995*a*; Zlotnick *et al.* 1995), even though the evidence is clear that there is considerable lifetime co-morbidity among adult disorders (Kessler, 1995) and make no attempt to determine whether different adversities predispose to different disorders. What little work has been done on this issue suggests that there may, in fact, be specificity in the effects of some childhood adversities (Rutter, 1989; Briere & Runtz, 1990; Mullen *et al.* 1996; Portegijs *et al.* 1996), although other studies suggest that the adult consequences of difference childhood adversities are not very distinct (Bushnell *et al.* 1992; Mullen *et al.* 1993). Resolution of these inconsistent results requires that analyses be carried out that adjust for lifetime co-morbidities among disorders.

Thirdly, the effects of childhood adversity on initial disorder onset and subsequent course have not been distinguished in most previous studies. This has created uncertainty regarding causal pathways. One widely held notion is that childhood adversities create enduring intra-psychological vulnerabilities that create heightened emotional reactivity to adult stress (Harris *et al.* 1990). Loss events early in life, for example, might lead not only to early-onset depression but also to disturbed attachment styles that create a depressogenic reaction to loss events throughout life. If this is so, though, we would expect that childhood adversities are associated not only with risk of lifetime psychiatric disorder but also with illness course. Some clinical studies have reported results consistent with this expectation (Brown *et al.*

1994; Zlotnick *et al.* 1995). However, others have found that most childhood adversities are related to first onset but not to course of adult disorders (Kessler & Magee, 1993; Faravelli *et al.* 1995; Pollack *et al.* 1996). Resolution of these inconsistent results requires this issue to be examined in large and representative samples.

The aims of this paper are to address the above three limitations empirically using data from a large general population survey carried out in the US. We examine the overlap among adversities by determining whether the presumed effects of particular adversities are attenuated or specified in causal models that include other adversities as covariates. We examine the specificity of effects by determining whether these presumed effects change when controls are introduced for lifetime co-morbidity among psychiatric disorders and whether adjusted effects vary meaningfully across different disorder outcomes. And we examine differential effects on onset *versus* course by distinguishing between the predictors of first onset and the predictors of persistence of the disorders assessed in the survey.

METHOD

Sample

The data reported here come from the US National Comorbidity Survey (NCS; Kessler *et al.* 1994), a survey of the household population of the coterminous United States designed to study the distribution and correlates of DSM-III-R (American Psychiatric Association, 1987) psychiatric disorders. The survey was fielded between September, 1990 and March, 1992. Respondents were selected from a nationally representative, stratified, multi-stage, area probability sample of persons in the age range 15–54 in the non-institutionalized civilian population, including a supplemental sample of students living in campus group housing. A special non-response survey was used to adjust statistically for non-response bias (Kessler *et al.* 1995*a*). A total of 8098 respondents participated in the survey. The response rate was 82·4%.

The NCS interview was administered in two parts, each of which took somewhat more than 1 hour to complete. Part I contained the core diagnostic interview, a brief risk factor battery,

and an inventory of sociodemographic background information. Part II contained a much more detailed risk factor battery, one part of which included a series of questions about a variety of childhood adversities. Part I was administered to all 8098 respondents, while Part II was administered to a subsample of respondents consisting of all those in the age range 15–24 (99·4% of whom completed Part II), all others who screened positive for any lifetime diagnosis in Part I (98·1% of whom completed Part II), and a random subsample of other respondents (99·0% of whom completed Part II). The current report is based on the 5877 respondents in the Part II subsample. More detailed descriptions of the NCS sampling design and field procedures have are reported elsewhere (Kessler *et al*. 1994, 1995*a*).

Measures

Childhood adversities

Based on a review of the literature, 26 childhood adversities were included in the NCS, consisting of five interpersonal loss events, eight interpersonal traumas, eight measures of parental psychopathology and five miscellaneous other adversities. Each of these adversities represents an experience that occurred by the time the respondent was 16 years of age. The loss events include death of mother, death of father, parental separation or divorce, parental absence of 6 months or longer other than due to separation or divorce (e.g. due to overseas service in the armed forces, imprisonment, or lengthy hospitalization), and respondent absence of 6 months or longer (e.g. due to hospitalization, living with other relatives, or attending boarding school). The questions used to assess these events were standard questions about childhood living arrangements used in our previous surveys, with information obtained on the respondent's age at the occurrence of each of these events.

The measures of parental psychopathology include separate assessments of mother and father major depression, generalized anxiety disorder (GAD), alcohol or drug abuse and antisocial personality disorder when the respondent was a child. These were assessed by the Family History Research Diagnostic Criteria (FHRDC) Interview (Endicott *et al*. 1978), except for GAD, which was measured by a previously validated FHRDC-like instrument developed by Kendler and associates (1991). Based on the finding in pilot studies that respondents generally are unable to recall the age when their parents first developed these disorders, no information about respondent's age at onset of exposure was obtained.

The measures of interpersonal trauma included assessments of maternal and paternal verbal aggression towards the respondent, isolated rape, repeated rape, isolated sexual molestation, repeated sexual molestation, being mugged or held captive and being seriously physically attacked. The respondent's age at the onset of parental aggression was not assessed based on the finding in pilot studies that 'as long as I can remember' was the typical response to such a question. However, age at first exposure was recorded for each of the other interpersonal traumas. The parental aggression measures are based on the verbal abuse items in the Conflict Tactics Scales (Straus, 1979), a widely used survey measure of family violence, while the other trauma measures were assessed in a structured trauma exposure checklist developed as part of the PTSD module in the diagnostic interview. This checklist asked respondents to provide yes/no reports to structured questions about a variety of adverse situations. This checklist has been described in more detail elsewhere (Kessler *et al*. 1995*b*).

The final five adversities include being in a life-threatening accident, being in a natural (e.g. hurricane) or man-made (e.g. fire) disaster, exposure to any other trauma that is outside the range of normal human experience as defined in DSM-III-R for qualifying triggers of post-traumatic stress disorder, learning about a traumatic experience that occurred to a close loved one and witnessing a traumatic event. These experiences were assessed in the trauma exposure checklist, which included information on age at first exposure to each of these experiences.

Psychiatric disorders

The diagnostic interview in the NCS was a modified version of the World Health Organization's Composite International Diagnostic Interview (CIDI 1.0; World Health Organization, 1990). The CIDI is a fully structured diagnostic interview designed to be

used by trained interviewers who are not clinicians. The 14 DSM-III-R diagnoses used in this report were made without diagnostic hierarchy rules and include mood disorders (major depression, mania, dysthymia), anxiety disorders (panic disorder, generalized anxiety disorder, simple phobia, social phobia agoraphobia), addictive disorders (alcohol and drug abuse and dependence), conduct disorder (CD) and adult antisocial behaviour (AAB). The NCS assessed CD and AAB (the adult component of the diagnosis of antisocial personality disorder) with questions from the Diagnostic Interview Schedule (DIS; Robins *et al.* 1988) due to the fact that the CIDI does not include an assessment of CD or AAB. The reliability and validity of the CIDI diagnoses used here have been shown to be adequate in World Health Organization CIDI Field Trials (Wittchen, 1994).

Analysis procedures

Weighting

The NCS data were weighted to adjust for variation in probabilities of selection and for differential non-response. Respondents in the Part II subsample, which in the basis for the analyses reported here, were also weighted by the inverse of their probabilities of selection into Part II in order to make the subsample representative of the total population. Finally, a post-stratification weight was applied to the Part II data by means of an iterative procedure that approximated the national population distributions of the cross-classification of age, sex, race/ethnicity, marital status, education, living arrangements, region and urbanicity as defined by the 1989 US National Health Interview Survey (US Department of Health and Human Services, 1992). The weighted Part II sample is representative of the total US population ages 15–54 on a variety of sociodemographic characteristics (Kessler *et al.* 1995*b*).

Model estimation

The results reported below are presented in the form of prevalences and odds ratios (ORs). The associations of adversities with lifetime disorders were estimated in discrete-time survival models (Willett & Singer, 1993) with person-years as the unit of analysis for the time-lagged relationships between childhood adversities and the subse-

quent first onset of each DSM-III-R disorder in the total sample. The predictors in these equations included one or more dummy variables to discriminate between respondents who did and did not report the occurrence of each childhood adversity. In the case of adversities that occurred throughout the childhood of respondents (e.g. parental violence), the dummy variable was coded 1 for each year of the respondent's life. In the case of adversities that had discrete ages of occurrence (e.g. life-threatening accidents), the dummy variable was coded 0 for person-years prior to that age and 1 thereafter. The equations also included control variables for the ages represented by each person-year, age at the time of interview, sex, race, and childhood family socioeconomic status. For ease of interpretation, coefficients were exponentiated and are presented here in the form of odds ratios (OR)s. Some models also controlled for lifetime co-morbidities of outcome disorders with prior disorders that might have been influenced by the adversities. In these latter models, time-varying coding was used to control for the prior disorders as of their age of onset. That is, each respondent was coded in a series of dummy predictor variables for whether he, or she, had a lifetime history as of a particular person-year of each other NCS/DSM-III-R disorder prior to the onset of the outcome disorder.

The associations of adversities with persistence of each disorder were obtained by estimating logistic regression models for the relationships between childhood adversities and 12-month prevalence in the subsample of respondents with a history of the disorder at least 1 year prior to the interview. The same control variables were used as in the analysis of first onset. In order to adjust for possible confounding of exposure with cohort and cohort with persistence, controls were included for age of onset of the disorder and for number of years since onset of the disorder.

Initial analyses of both lifetime onset and persistence examined time-lagged associations between childhood adversities that occurred prior to the onset of the disorder and each the 14 DSM-III-R disorders and for a composite measure of any disorder. The latter was defined as occurring at the age of onset of the first

disorder reported by the respondent. Models of several sorts were considered. We began with a model in which only a single adversity was considered at a time. We then estimated a model that controlled for lifetime co-morbidities among the outcome disorders (defined as prior onset of other disorders, with separate dummy variables used to define each of the other 13 disorders). A third additive model also controlled for prior or concurrent onset of other adversities. A series of multivariate models was then estimated that evaluated the joint effects of all adversities and included terms for a variety of non-additive combinations of adversities.

Significance testing

Because of the complex sample design, significance tests in most NCS analyses are based on empirical pseudo-replication methods. However, in the present report the number of models estimated was so large that standard likelihood-ratio methods were used to evaluate the comparative fit of nested models. The 0·01 level of significance was used rather than the 0·05 level in this model-fitting phase in order to take into consideration the fact that these tests do not adjust for the effects of clustering or weighting. Once best-fitting models were selected, standard errors of parameter estimates were evaluated at the 0·05 level of significance using two-tailed tests.

RESULTS

The prevalences of childhood adversities

Prevalences of the retrospectively reported childhood adversities are shown in Table 1. The 5877 respondents reported a total of 12 352 adversities. Only 25·6 % of respondents reported that they experienced none of these adversities, while 23·2 % reported one, 16·1 % two and 35·0 % three or more adversities. Prevalence estimates of individual adversities range from a high of 22·7 % for maternal depression to a low of 1·0 % for repeated rape.

As shown in the fourth column of Table 1, the vast majority of those who reported any single adversity reported the occurrence of at least one other adversity. Nonetheless, the condition number of the Pearson correlation matrix among the adversities is only 2·5, which is in the range

of values indicating no serious problem of multicollinearity (Belsley *et al.* 1980). Furthermore, the Pearson correlations among adversities are fairly modest in magnitude, with the highest correlations found among the measures of maternal psychopathology (bivariate correlations averaging 0·28) and paternal psychopathology (bivariate correlations averaging 0·26). The other bivariate correlations average only 0·08. (A complete matrix of correlations and odds ratios among the 26 adversities as well as other detailed results of analyses mentioned in the text but not presented in tables are available as a technical appendix to this paper archived on the NCS WWW home page described in the acknowledgements section.)

Bivariate associations between specific adversities and disorder onset

Likelihood-ratio chi-square tests were compared for an additive model and a series of substantively plausible non-additive models of the associations between adversities and the subsequent first onset of each of the 14 NCS/DSM-III-R disorders. The additive model was a significant improvement over the control model for all outcomes other than mania and AAB. The non-additive models, however, generally did not lead to significant improvements in fit compared to the additive model.

The bivariate associations (odds-ratios) between childhood adversities and first onset of the 14 NCS/DSM-III-R disorders are presented in Table 2. These ORs are reported in the form in which they have typically been reported in previous studies; that is, with controls for sociodemographic variables but without controls either for exogenous correlations among the adversities or for lifetime co-morbidities among the outcome disorders.

Five broad patterns can be observed in the table. First, there is a generally positive pattern of associations between prior adversities and subsequent disorders, with 89·9 % of the ORs greater than 1·0 and 66·8 % statistically significant at the 0·05 level. The proportion of statistically significant ORs less than 1·0, in comparison, is no more than we would expect by chance (1·1 %).

Secondly, there is considerable variation in the consistency of the ORs across types of

Table 1. *Prevalences of retrospectively reported childhood adversities*

Adversity	Males %	Females %	Total %	% Only event %	Mean number of other events
I Loss events					
Father died	5·7	6·2	6.0	30·9	1·9
Mother died	3.0	3·6	3·3	23·8	2·3
Parents sep./div.	17·6	18·8	18·2	20·7	2·8
Parents leave other	7·8	10·6	9·3	27·2	2·6
R. temp. leave	2·1	2·8	2·5	30·9	2·5
II Parental psychopathology					
Father depressed	13·5	14·7	14·1	5·7	3·6
Father GAD	12·1	11·5	11·8	5·2	3·8
Father ASP	3·9	5.0	4·5	0.0	5·2
Father drugs/alc	18·1	18·5	18·3	7·0	3·5
Mother depressed	19·7	25·4	22·7	8·2	3·4
Mother GAD	15·8	18·6	17·2	6·7	3·8
Mother ASP	1·0	2·0	1·5	0·6	6·5
Mother drugs/alc	5·1	7·8	6·6	2·0	4·3
III Interpersonal traumas					
Molested (isolated)	1·3	5·1	3·3	6·8	4·0
Molested (repeated)	1·2	6·1	3·8	15·8	3·9
Rape (isolated)	0·3	3·4	2·0	2·2	4·2
Rape (repeated)	0·2	1·8	1·0	0·0	4·1
Mugged/Kidnapped	5·6	2·1	3·8	4·9	4·5
Phys. attacked	4·4	2·2	3·3	5·4	4·8
Father aggressed*	24·3	16·2	20·0	9·0	3·2
Mother aggressed*	15·1	17·6	16·4	5·4	3·3
IV Other adversities					
Accident	8·2	5·2	6·6	9·4	3·7
Natural/Man-made disasters	10·0	8·6	9·3	15·4	3·1
Witnessed trauma	13·0	6·0	9·3	13·1	3·4
Shocked	2·9	4·5	3·7	9·2	4·0
Other PTSD event	1·2	0·8	0·9	22·8	3·7
V Total number of adversities					
None	26·5	24·9	25·6	—	—
One	23·1	23·4	23·2	—	—
Two	15·9	16·3	16·1	—	—
Three or more	34·6	35·6	35·0	—	—

* Father and mother aggression are the only adversities that are not natural dichotomies. They are reported here as the percentage of respondents who reported that the parent was often or sometimes verbally aggressive. Rarely or never were coded as these adversities being absent.

adversity. Parental psychopathologies are the most consistent predictors (92·9% of ORs are statistically significant), followed by interpersonal traumas (78·6% statistically significant), with much less consistent associations for loss events (31·4% statistically significant) and other adversities (44·3% statistically significant).

Thirdly, there is also important variation among the ORs within categories of adversity. Parent marital break-up is a more consistent predictor than other loss events. Maternal psychopathologies, especially ASP, are somewhat stronger predictors than paternal psychopathologies in the majority of contrasts. Repeated rape and kidnap are more consistent and

generally stronger predictors than other interpersonal traumas. Witnessing and learning of a trauma that occurred to a loved one, finally, are more consistent predictors than the remaining events in the residual category of other traumas.

Fourthly, there is some variation in the ORs across outcomes, suggesting that childhood adversities more powerfully influence some disorders than others. Given the substantial difference in the prevalences of individual disorders and difference in statistical power associated with it, an examination of substantive importance is more useful here than an examination of statistical significance. As a group, anxiety disorders have the lowest proportion of substantively important ORs (55·4%), which we

Table 2. *Bivariate associations (odds ratios) between childhood adversities and the subsequent onset of NCS/DSM-III-R disorders, controlling for sociodemographic variables†*

Adversity	Mood disorders			Anxiety disorders					Addictive disorders				Other		
	MD	MA	DYS	PD	AG	SoP	SiP	GAD	AA	AD	DA	DD	CD	AAB	Any
I Loss events															
Father died	0·81	0·50	1·19	0·17*	0·25*	0·63*	0·73	0·57	1·40*	1·62*	1·35	1·20	1·11	1·41	0·67*
Mother died	1·02	2·34	1·35	1·13	1·20	0·61	1·00	1·43	0·65	1·29	1·94*	0·99	0·97	1·60	0·83
Parents sep./div.	1·51*	2·61*	2·12*	1·46*	1·60*	1·40*	1·39*	1·57*	1·46*	1·48*	1·79*	2·38*	1·85*	1·54*	1·31*
Parents leave other	1·19	1·45	1·41*	0·71	0·79	1·33*	1·12	1·30	1·22	1·03	1·06	0·88	1·46*	1·42	1·44*
R. temp. leave	1·03	3·30*	1·25	2·57*	1·08	0·71	1·33	1·19	0·77	0·91	0·37*	1·31	1·10	1·27	1·19
II Parental psychopathology															
Father depressed	2·23*	4·12*	1·91*	2·52*	1·60*	2·07*	1·67*	2·15*	1·34*	1·82*	1·51*	2·01*	1·84*	1·57*	1·84*
Father GAD	2·26*	3·40*	2·33*	3·20*	1·93*	2·08*	1·63*	2·72*	1·16	1·54*	1·67*	2·27*	2·35*	1·70*	1·97*
Father ASP	1·80*	2·99*	1·18	1·93*	1·39	1·59*	1·73*	2·54*	1·60*	1·58*	1·57*	2·85*	2·94*	2·20*	1·77*
Father drugs/alc	2·00*	2·01*	1·50*	2·13*	1·77*	1·74*	1·42*	2·47*	1·59*	2·32*	2·10*	2·27*	1·76*	2·04*	1·72*
Mother depressed	2·98*	4·02*	2·16*	3·05*	1·62*	1·98*	2·26*	2·45*	1·56*	1·79*	1·89*	2·01*	1·99*	1·80*	1·90*
Mother GAD	2·93*	4·93*	2·88*	3·95*	2·01*	2·76*	2·61*	3·25*	1·49*	2·15*	2·33*	2·78*	2·42*	2·23*	2·21*
Mother ASP	2·63*	10·45*	2·62*	4·68*	3·17*	1·38	1·41	6·04*	2·22*	4·05*	5·56*	3·92*	5·66*	4·07*	1·53*
Mother drugs/alc	1·79*	4·64*	1·95*	1·67*	1·10	1·27	1·20	2·39*	1·83*	2·48*	2·68*	2·59*	2·54*	2·41*	1·37*
III Interpersonal traumas															
Molested (isolated)	1·79*	2·51	1·63	1·46	0·93	1·68*	1·02	1·48	1·65*	1·76*	3·24*	1·48	2·11*	1·49	1·21
Molested (repeated)	2·62*	4·70*	2·81*	3·19*	1·52	1·67*	1·63*	2·18*	2·03*	1·71*	2·66*	2·49*	3·16*	2·29*	2·58*
Rape (isolated)	2·11*	6·19*	1·82	2·06	1·92*	1·77*	1·91*	0·53	2·45*	2·05*	4·02*	5·14*	4·77*	1·95	1·52*
Rape (repeated)	3·01*	1·85	4·36*	2·90	3·11*	1·40	1·19	4·86*	0·84	1·22	1·57	2·76*	2·85*	5·28*	1·26
Mugged/Kidnapped	2·27*	7·08*	3·41*	5·08*	2·40*	1·51	1·64*	1·38	1·64*	2·51*	1·88*	3·28*	4·83*	4·05*	1·90*
Phys. attacked	2·50*	6·25*	2·76*	3·59*	2·18*	1·71*	1·22	1·30	2·22*	2·81*	1·95*	3·70*	8·18*	4·64*	1·79*
Father aggressed	1·88*	1·88*	1·98*	1·76*	1·47*	1·77*	1·84*	1·94*	1·29*	1·46*	1·62*	1·79*	2·00*	2·00*	1·60*
Mother aggressed	1·66*	2·80*	2·28*	2·82*	1·28*	1·34*	1·48*	2·77*	1·31*	1·58*	1·77*	2·20*	1·61*	1·64*	1·54*
IV Other adversities															
Accident	2·07*	4·78*	1·75*	1·61	1·57*	1·22	1·13	1·62	1·04	2·24*	1·36	1·79*	1·15	1·71*	0·87
Nat./Man-made dis.	1·40*	1·48	0·72	1·77*	1·07	0·99	0·73	0·83	1·52*	1·28	1·51*	1·39*	1·41*	1·47*	0·84*
Witnessed trauma	2·53*	2·50*	1·31	2·92*	1·38	0·97	1·02	2·50*	0·99	1·47*	1·37*	1·88*	1·85*	2·25*	1·18
Other PTSD event	1·10	0·61	2·35*	1·73	1·45	0·35	0·42	2·06	1·30	0·63	0·86	0·15	1·42	0·63	1·03
Shocked	2·15*	2·85*	0·78	1·74	1·41	1·67*	1·20	1·49	1·53*	1·70*	2·20*	2·05*	0·95	1·42	1·21

† Models were estimated in a discrete-time survival framework with one adversity and controls used to predict the subsequent first onset of the outcome disorders.

The sociodemographic control variables include age cohort, sex, race, childhood family socioeconomic status and each person-year.

* Significant at the 0·05 level, two-tailed.

define arbitrarily as ORs greater than or equal to 1·5. There are especially low proportions for phobias (34·6% for simple phobia 50·0% for social phobia and 50·0% for agoraphobia). Larger proportions of the ORs are substantively important for addictive disorders (65·4%), CD/ AAB (71·4%) and mood disorders (76·9%).

Fifthly, while there is some evidence of specificity in the associations between particular adversities and particular outcomes (e.g. parental depression is a stronger predictor of respondent MD than of any other outcome), the main impression one gets from the total data array is that each of the adversities is meaningfully related to each broadly defined class of disorders.

The effects of controlling for co-morbidity and clustering

A second model was estimated to adjust for co-morbidities among the outcome disorders. The ORs based on this model are reported in Table 3. A third model was then estimated that added controls for the prior or joint occurrence of other adversities. The ORs based on this model are reported in Table 4.

As there are more than 1000 ORs in Tables 3 and 4, aggregate comparison, which is our main goal in this first paper on adversity and disorder in the NCS, is facilitated by examining overall patterns of association. Subsequent papers will examine particular adversities and adversity

Table 3. *Multivariate associations (odds ratios) between childhood adversities and the subsequent on-set of NCS/DSM-III-R disorders, controlling for sociodemographic variables and prior disorders†*

Adversity	Mood disorders			Anxiety disorders					Addictive disorders				Other		
	MD	MA	DYS	PD	AG	SoP	SiP	GAD	AA	AD	DA	DD	CD	AAB	Any
I Loss events															
Father died	0·81	0·21*	1·21	0·28*	0·30*	0·67	0·91	0·51	1·42*	1·65*	1·11	1·04	1·11	1·28	0·67*
Mother died	0·86	1·41	1·19	0·62	1·09	0·61	1·08	0·98	0·66	1·52	1·86	1·10	1·01	1·92*	0·83
Parents sep./div.	1·22*	2·53*	1·95*	1·37	1·33	1·14	1·09	1·25	1·27*	1·19	1·43*	1·88*	1·75*	0·89	1·31*
Parents leave other	1·03	0·68	1·24	0·64	0·76	1·24	1·05	1·31	1·11	0·71	0·81	0·86	1·38*	1·06	1·44*
R. temp. leave	1·06	1·54	1·39	2·17	1·10	0·70	1·52	1·05	0·79	0·80	0·64	1·35	1·14	1·22	1·19
II Parental psychopathology															
Father depressed	1·69*	2·21*	1·41*	1·45	1·15	1·64*	1·28*	1·44*	1·18	1·14	0·99	1·49*	1·76*	0·79	1·84*
Father GAD	1·56*	2·50*	1·77*	2·16*	1·36*	1·60*	1·21	1·64*	0·91	1·02	1·12	1·67*	2·20*	0·90	1·97*
Father ASP	1·49*	2·25	0·91	1·21	1·15	1·09	1·60*	2·72*	1·41*	1·14	1·09	2·39*	2·83*	1·35	1·77*
Father drugs/alc	1·57*	1·09	1·04	1·37	1·22	1·59*	1·13	2·10*	1·39*	1·76*	1·46*	1·39*	1·71*	1·29	1·72*
Mother depressed	2·44*	1·89*	1·12	2·02*	0·99	1·57*	1·87*	1·56*	1·33*	1·12	1·26	1·22	1·88*	1·01	1·90*
Mother GAD	2·05*	2·12*	1·35*	1·94*	1·23	1·98*	1·94*	1·77*	1·24*	1·36*	1·65*	1·59*	2·19*	1·11	2·21*
Mother ASP	1·35	4·76*	0·61	2·23	1·51	0·88	0·78	3·90*	1·22	1·98*	3·15*	0·73	4·61*	0·78	1·53*
Mother drugs/alc	1·68*	3·22*	0·81	1·10	0·70	1·06	0·97	1·55	1·31*	1·75*	2·07*	1·14	2·33*	1·05	1·37*
III Interpersonal traumas															
Molested (isolated)	1·54*	2·53	1·08	0·88	0·58	1·46	0·90	1·33	1·17	0·80	2·97*	0·74	2·05*	0·61	1·21
Molested (repeated)	1·80*	2·92	1·84*	1·45	1·00	1·23	1·17	1·10	1·50*	0·92	1·76*	1·17	2·53*	0·95	2·58*
Rape (isolated)	1·32	10·42*	1·04	1·58	1·23	0·63	1·33	0·41	1·24	0·61	2·64*	2·63*	4·59*	1·35	1·52*
Rape (repeated)	2·52*	0·78	0·91	2·20	2·12	1·00	0·70	2·08	0·66	0·52	1·22	1·58	2·42*	2·73	1·26
Mugged/Kidnapped	1·42	1·72	1·71	2·14	1·75*	0·79	0·87	0·41*	1·04	1·02	0·93	1·80*	4·48*	1·33	1·90*
Phys. attacked	1·20	1·97	1·65	1·63	1·43	0·95	0·71	0·60	1·32	0·90	1·04	1·26	6·95*	1·29	1·79*
Father aggressed	1·54*	1·02	1·20	1·24	1·00	1·52*	1·56*	1·20	1·11	1·04	1·18	1·18	1·73*	1·34*	1·60*
Mother aggressed	1·37*	1·58	1·55*	1·88*	0·91	1·20*	1·28*	1·81*	1·28*	1·34	1·34*	1·43*	1·49*	1·00	1·54*
IV Other adversities															
Accident	1·82*	1·83	0·98	0·94	1·37	0·99	0·66	0·59	0·82	1·54*	0·91	1·45	1·07	0·88	0·87
Nat./Man-made dis.	1·43*	1·58	0·56*	1·22	0·91	0·94	0·60*	0·54*	1·31*	1·03	1·16	1·49*	1·40*	1·07	0·84*
Witnessed trauma	1·95*	1·26	1·00	1·90*	0·60*	0·76	0·69	1·29	0·77*	1·21	0·83	1·39	1·70*	1·20	1·18
Other PTSD event	0·59	0·64	3·93*	2·23	1·04	0·32	0·34	2·28	1·23	0·54	0·62	0·21	1·45	0·86	1·03
Shocked	1·62*	2·69	0·64	1·73	1·05	1·28	0·87	0·92	1·24	0·86	1·67	0·91	0·65	0·67	1·21

† Models were estimated in a discrete-time survival framework with one adversity and controls used to predict the subsequent first onset of the outcome disorder. The sociodemographic variables are listed in the note to Table 2. The prior disorder controls included time-varying covariates for the onset of other NCS/DSM-III-R disorders at earlier ages than the outcome disorder. Included were both disorders that occurred prior to the adversity, which could be risk factors for both the adversity and the outcome disorder (e.g. conduct disorder predicting subsequently being in a life-threatening automobile accident and independently predicting the subsequent onset of alcoholism) and disorders that intervened between the onset of the adversity and the outcome disorder, which could mediate one of the causal pathways linking the adversity to the outcome (e.g. the adversity predicting the subsequent onset of primary depression, which, in turn, predicts the subsequent onset of secondary alcoholism).

* Significant at the 0·05 level, two-tailed.

clusters in greater detail. A summary of aggregate comparisons is presented in Table 5, where we see that the numbers of substantively important and statistically significant ORs are halved when we adjust for co-morbidities among the outcome disorders – from 65·1 % substantively important and 66·8 % statistically significant in Table 2 to 31·9 % substantively important and 31·9 % statistically significant in Table 3 – and reduced even more when we also adjust for overlap among the adversities – to 22·1 % substantively important and 14·8 % statistically significant in Table 4.

Inspection of the Total rows in Parts I and II

of Table 5 shows that mood disorders have the highest proportion of substantively important ORs in the model that controls for co-morbidities (48·7 % compared to between 21·2 % and 36·5 % for other disorders) as well as the smallest proportional reduction in substantively important ORs compared with the model that does not control for co-morbidities (36·7 % compared to between 48·6 % and 67·6 %). This means that the adversities considered here have a larger proportion of direct associations with mood disorders than with the other disorders. Addictive disorders, in comparison, have the lowest proportion of substantively important

Table 4. *Multivariate associations (odds ratios) between childhood adversities and the subsequent onset of NCS/DSM-III-R disorders, controlling for sociodemographic variables, prior disorders, and prior adversities*†

Adversity	Mood disorders			Anxiety disorders					Addictive disorders				Other		Any
	MD	MA	DYS	PD	AG	SoP	SiP	GAD	AA	AD	DA	DD	CD	AAB	
I Loss events															
Father died	0·87	0·32	1·43	0·28*	0·32*	0·75	0·92	0·47*	1·47*	1·65*	1·01	1·18	1·43	1·24	0·78*
Mother died	0·96	2·27	1·33	0·84	1·28	0·72	1·40	1·19	0·62	1·60*	2·10*	1·33	1·22	1·90	0·90
Parents sep./div.	1·06	1·83	2·31*	1·19	1·31	1·15	1·18	1·02	1·14	1·06	1·17	1·73*	1·45*	0·86	1·19*
Parents leave other	0·99	0·63	1·33	0·57	0·80	1·20	1·05	1·18	1·13	0·73*	0·77	0·80	1·21	1·00	1·28*
R. temp. leave	0·97	2·39	1·56	2·41	1·08	0·67	1·78*	1·16	0·85	0·80	0·72	1·47	1·20	1·25	1·13
II Parental psychopathology															
Father depressed	1·27*	1·70	1·06	0·88	1·03	1·24	1·11	0·97	1·14	1·03	0·81	1·16	1·11	0·72	1·25*
Father GAD	1·07	2·28	1·71*	1·76*	1·34	1·07	0·83	1·37	0·73*	0·93	1·10	1·42	1·53*	0·86	1·22*
Father ASP	0·87	1·41	0·53	0·65	0·88	0·84	1·22	1·72	1·10	0·87	0·70	1·83*	1·51*	1·23	1·02
Father drugs/alc	1·18	0·64	0·96	1·12	1·19	1·33*	0·90	1·73*	1·33*	1·79*	1·38*	1·08	1·04	1·27	1·25*
Mother depressed	1·89*	0·97	0·94	1·61*	0·86	1·13	1·64*	1·18	1·21	0·82	0·88	0·79	1·19	0·90	1·32*
Mother GAD	1·28*	1·65	1·25	1·31	1·32	1·75*	1·60*	1·38	1·04	1·38*	1·43*	1·54*	1·49*	1·26	1·61*
Mother ASP	0·75	2·03	0·54	2·04	2·04	0·83	0·63	3·16*	0·81	1·40	1·57	0·39*	2·02*	0·77	0·95
Mother drugs/alc	1·15	1·58	0·73	0·59	0·55*	0·83	0·67*	0·80	1·07	1·46*	1·53*	0·97	1·31	1·10	0·83
III Interpersonal traumas															
Molested (isolated)	1·25	2·04	0·93	0·74	0·46	1·44	0·69	1·27	1·00	0·81	2·39*	0·60	1·19	0·58	0·83
Molested (repeated)	1·38	1·82	2·05*	0·89	0·79	1·25	1·05	0·86	1·44	0·90	1·72*	0·93	1·77*	0·86	1·93*
Rape (isolated)	1·09	6·41*	0·66	1·95	1·27	0·67	1·11	0·40	0·99	0·63	1·87	3·16*	0·89	0·45	1·14
Rape (repeated)	1·63	0·30	0·73	2·01	2·11	0·74	0·68	2·59	0·54	0·49	0·80	1·60	1·38	2·33	1·02
Mugged/Kidnapped	1·08	0·95	1·66	1·68	1·74	0·81	1·07	0·38*	0·95	1·07	0·82	1·37	1·97	1·24	1·47*
Phys. attacked	0·84	0·99	1·28	0·99	0·96	1·00	0·66	0·74	1·44	0·88	1·08	0·85	4·30*	1·31	1·29
Father aggressed	1·29*	0·89	0·91	0·96	0·96	1·34*	1·54*	0·90	1·02	0·96	1·00	0·96	1·60*	1·39*	1·32*
Mother aggressed	1·02	1·26	1·46*	1·55*	0·91	0·92	1·02	1·56*	1·18	1·02	1·20	1·31	1·02	0·95	1·20*
IV Other adversities															
Accident	1·33	2·47	1·06	0·67	1·54	0·96	0·72	0·51	0·78	1·50*	0·91	1·18	0·72	0·78	0·71*
Nat./Man-made dis.	1·09	1·10	0·48	1·19	0·86	0·85	0·57*	0·50	1·30*	0·92	1·02	1·25	1·09	1·03	0·79*
Witnessed trauma	1·63*	0·78	0·93	1·86*	0·46*	0·67*	0·82	1·71*	0·67*	1·10	0·79	1·27	1·45	1·17	1·07
Other PTSD event	0·40*	0·91	2·91*	1·50	0·83	0·16*	0·16*	2·74	1·10	0·60	0·62	0·09	0·41	0·92	0·54
Shocked	1·15	1·89	0·60	1·16	1·23	1·48	1·08	0·68	1·17	0·82	1·52	0·85	0·41*	0·60	1·22

† Models were estimated in a discrete-time survival framework with all adversities and controls used to predict subsequent first onset of the outcome disorder. The sociodemographic control variables are listed in the note to Table 2. The prior disorder controls included time-varying covariates for the first onset of other NCS/DSM-III-R disorders at earlier ages than the outcome disorder. The prior adversity controls included time-varying covariates for each adversity that occurred prior to the outcome disorder.

* Significant at the 0·05 level, two-tailed.

ORs in Part II (21·2 %) as well as the largest proportional reduction in substantively important ORs (67·6 %). This means that the relationships between the adversities and addictive disorders are more likely than those between the adversities and other disorders to be indirect through prior lifetime co-morbid disorders.

Inspection of the Total column in Parts II and III of Table 5 shows that, unlike the results in earlier models, parental psychopathology, and interpersonal traumas have comparable proportions of substantively important ORs (22·3 %) when we adjust for overlap among these adversities and that these proportions are higher than the comparable proportion, for loss events (17·1 %) and other adversities (15·7 %).

Parental psychopathologies have the largest proportional reduction in the number of important adversities (51·0 %) from Part II to Part III, which means that it is not so much any one of these parental disorders as the larger set of adversities in which they are embedded that predicts the subsequent onset of the outcomes considered here.

A comparison of the individual coefficients in Tables 3 and 4 shows that this reduction is especially pronounced for paternal depression and paternal addictive disorders. Furthermore, contrary to the results in Table 2, parental marital break-up is no longer a more consistent predictor than other loss events when we adjust for adversity clustering. However, maternal psychopathologies continue to be somewhat

Table 5. *Percentage of parameter estimates in Tables 5–7 that are substantively important (SI)**
and statistically significant (SS)† by type of adversity and class of disorder‡

	Outcome disorders				
	Mood	Anxiety	Addictive	Other	Total
I Sociodemographic controls					
Loss events					
SI	33·3	12·0	20·0	30·0	21·4
SS	33·3	28·0	35·0	30·0	31·4
Parental psychopathology					
SI	95·8	82·5	90·6	100·0	90·2
SS	95·8	85·0	96·9	100·0	92·9
Interpersonal trauma					
SI	100·0	65·0	81·3	93·8	81·3
SS	83·3	65·0	87·5	87·5	78·6
Other adversities					
SI	53·3	40·0	45·0	30·0	42·9
SS	60·0	20·0	60·0	50·0	44·3
Total					
SI	76·9	55·4	65·4	71·2	65·1
SS	73·1	55·4	73·1	73·1	66·8
II Sociodemographic and other disorder controls					
Loss events					
SI	20·0	8·0	20·0	20·0	15·7
SS	20·0	0·0	25·0	30·0	15·7
Parental psychopathology					
SI	58·3	50·0	28·1	50·0	45·5
SS	66·7	50·0	56·3	50·0	55·4
Interpersonal trauma					
SI	58·3	27·5	21·9	50·0	35·7
SS	33·3	17·5	28·1	56·3	29·5
Other adversities					
SI	46·7	16·0	10·0	10·0	20·0
SS	40·0	4·0	15·0	20·0	17·1
Total					
SI	48·7	28·5	21·2	35·5	31·9
SS	42·3	21·5	33·7	42·3	32·4
III Sociodemographic, other disorders, and other adversity controls					
Loss events					
SI	33·3	8·0	20·0	10·0	17·1
SS	6·7	4·0	25·0	10·0	11·4
Parental psychopathology					
SI	29·2	25·0	15·6	18·8	22·3
SS	16·7	20·0	28·1	25·0	22·3
Interpersonal trauma					
SI	25·0	22·5	15·6	31·3	22·3
SS	16·7	10·0	9·4	25·0	13·4
Other adversities					
SI	26·7	20·0	10·0	0·0	15·7
SS	13·3	8·0	10·0	0·0	8·6
Total					
SI	28·2	20·0	15·4	17·3	20·1
SS	14·1	11·5	18·3	17·3	14·8

* Substantively important (SI) effects were defined as those with odds-ratios greater than or equal to 1·5.
† Statistical significance (SS) was evaluated at the 0·025 level, one-tailed tests.
‡ See Table 1 for the classification of adversities and Tables 2–4 for the classification of the outcome disorders.

more consistent predictors than paternal psycho-pathologies, while repeated rape and kidnap continue to be more consistent predictors than other interpersonal traumas.

The results in Part III also contain two patterns that provide more evidence for specificity of effects than we saw in the bivariate associations. First, loss events clearly have much more consistently powerful associations with the subsequent onset of mood disorders than with the subsequent onset of anxiety disorders. As shown in Table 4, these effects are confined to

Table 6. *Multivariate associations (odds ratio) between pure childhood adversities and the subsequent onset of NCS/DSM-III-R disorders, controlling for sociodemographic variables and prior disorders†*

	Mood disorders		Anxiety disorders				Addictive disorders				Other	
	MD	DYS	AG	SoP	SiP	GAD	AA	AD	DA	DD	CD	AAB
I Loss events												
Father died	1·01	3·44*	0·29	0·85	0·72	0·50	1·46	1·36	0·24	0·64	0·86	0·64
Mother died	0·48	0·42	1·88	0·73	1·45		0·53	2·27*	3·49*	0·47	0·73	1·38
Parents sep./div.	0·84	2·60*	1·86	3·12*	0·41*	3·44	1·25	1·15	0·93	0·85	3·12*	1·86
II Parental psychopathology												
Father depressed	2·79*		4·29*		0·40*		2·37*	0·36	1·25			
Father GAD	1·79		14·58*				0·56	0·84	0·67	4·70*		
Father drugs/alc	2·14*	2·29	0·20	2·84*	0·20*	3·50	1·24	2·27*	4·79*	0·87	2·84*	
Mother depressed	2·22*	0·48*	0·39	1·06*	0·68	2·46	1·21	0·48	0·97	0·56	1·06	
Mother GAD	2·18*		2·99	5·15*	0·33*	0·93	0·97	0·88	0·99	0·81	5·15*	
III Interpersonal traumas												
Molested (repeated)	1·81	16·56*					1·83		2·61			
Father aggressed	2·31*	2·29	1·57	2·54*	0·37*		0·86	0·92	0·67	0·54	2·54*	4·43*
Mother aggressed	0·37	1·08	2·80	2·25	0·18*	3·18	1·25	0·97			2·25	
IV Other adversities												
Accident	1·02						2·26*	1·06	1·98	1·78		4·07*
Nat./Man-made dis.	0·63					2·52	1·49	0·57				
Witnessed trauma	1·74				0·39	8·89*	1·83*	1·19	0·51	0·53		1·32

† Models were estimated in a discrete-time survival framework in the subsample of respondents who reported either no adversities or one and only one adversity. The sociodemographic controls are listed in the note to Table 2. The prior disorder controls included time-varying covariates for the first onset of other NCS/DSM-III-R disorders at earlier ages than the outcome disorder.

* Significant at the 0·05 level, two-tailed.

mania and dysthymia. None of the loss events significantly predicts major depression. Secondly, the residual category of other adversities, which consists largely of one-time natural or man-made disasters, is related to the disorders that we would expect to be associated with loss (mood disorders) and danger (anxiety disorders) and maladaptive coping with these reactions (addictive disorders), but not to either CD or AAB.

The effects of pure adversities

Previous research has shown that the analysis of respondents with pure adversities sometimes fails to replicate the results of additive multivariate analyses in which statistical controls are used to adjust for overlap among adversities (Mullen *et al.* 1996; Portegijs *et al.* 1996). In an effort to see whether this is also true in the NCS data, we re-estimated the model in Table 4 in the subsample of respondents who reported either none or one and only one of the adversities considered here. Results are reported in Table 6 for all adversity-disorder pairs containing at least five respondents (i.e. at least five respondents with the disorder who retrospectively reported the adversity).

The number and sign pattern of significant ORs (30 greater than 1·0 and five less than l·0) in Table 6 are very similar to those for comparable coefficients in Table 4 (29 greater than 1·0 and three less than 1·0), verifying that individual adversities are significant predictors of disorder onset even when they do not occur in clusters. The same general pattern can be seen within each of the four broadly defined classes of adversity. There are more large ORs in Table 6 than Table 4, but the ratio of these ORs that are greater than 1·5 *versus* less than 0·67 (about 2:1) is lower than in Table 4 (about 4:1), suggesting that the larger size of the ORs in Table 6 is probably caused by increased noise in the data associated with the comparatively small number of people with pure adversities.

Subgroup specifications by sex and timing of outcome disorders

We next investigated whether the broad pattern of results reported above differs significantly by sex and timing of the outcome disorders.

Regarding sex, comparative analyses showed that only 13·7% of the coefficients in Table 4 differed significantly by sex of respondent at the 0·05 level of significance and that the number larger among men (5·5%) and larger among women (8·2%) were quite similar. Based on this pattern of results, we concluded that there is no systematic sex difference in the associations between the childhood adversities and disorders assessed in the NCS.

Regarding timing of the outcome disorders, we investigated whether childhood adversities are significantly stronger predictors of early-onset than later-onset disorders, where the operational definition of 'early-onset' was a disorder that began at an age earlier than the median age of onset of the disorder in the total sample. Conduct disorder and adult antisocial behavior were excluded from this part of the analysis, as the definitions of these disorders in DSM-III-R stipulate age ranges of onset. More than two-thirds (68·8%) of the ORs for the interaction terms associated with the remaining disorders in these equations showed adversities to be stronger predictors of early-onset than later-onset disorders and 17·9% of all these interaction terms were statistically significant at the 0·05 level. No more interactions were significant in the other direction (i.e. adversities stronger predictors of later-onset than early-onset disorders) than would be expected by chance (1·3%). Consistent with these results, a larger proportion of early-onset than later-onset ORs were found to be substantively important (27·9% *v.* 18·9%) and statistically significant at the 0·05 level (11·2% *v.* 8·7%).

It is of interest to investigate the shape of the time decays in the associations between childhood adversities and subsequent disorder onset. If these decays are due to a simple distance effect of the sort postulated by Surtees (1989; Surtees & Ingham, 1980) for the association between stressful life events and onset of major depression, we would expect the ORs to become successively smaller as time since occurrence of the adversity increases and to asymptote either at or below 1·0 after some period of time. If, however, there is a residual scarring effect of exposure to childhood adversities, we would expect the ORs to asymptote at a value that is significantly greater than 1·0 and to vary over time as a function of time-varying exposure to

stressful experiences that potentiate the effect of the scar.

There are 312 (26 × 12) adversity-disorder combinations in which to examine time decay, but the number of cases in any one of these cells is too small for statistically powerful analysis. As a result, we examined the shape of time decay in an aggregate analysis by focusing on the subsample of respondents who reported one and only one of the four time-varying adversities (i.e. those with discrete ages of occurrence) that had significantly larger associations with early-onset than later-onset disorders. These four are parental separation or divorce, repeated molestation, repeated rape, and physical attack. Two nested expansions of the model in Table 6 were estimated to compare the respondents who experienced one of these adversities as a group with those who experienced no adversities on first onset of any NCS/DSM-III-R disorder. The first model evaluated the effect of a linear time decay term (i.e. number of years since the occurrence of the adversity) of time since exposure to adversity, which was significant (OR = 0·94; 95% CI: 0·91–0·97). A second model added a square of the time decay term to capture any non-linearity in the decay. This non-linear term was found to be significant (OR = 1·003; 95% CI; 1·001–1·005).

The predicted shape of the time decay from the non-linear model and the observed values of the ORs for onset of any disorder as a function of time since the occurrence of the adversity are shown in Fig. 1. We can see there that the relative-odds of disorder onset are highest during the first few years after the occurrence of the adversity and decline within approximately a decade to a level equivalent to that found among people who were not exposed. There is also evidence that the asymptote for long-term risk is lower among those people who did not have an onset of disorder during the first decade after exposure than among the controls.

Bivariate associations between specific adversities and disorder persistence

Likelihood-ratio chi-square tests were compared for additive and non-additive models of the associations between adversities and the persistence of each NCS/DSM-III-R disorder in the subsample of respondents who had a history of the outcome disorder prior to the past 12
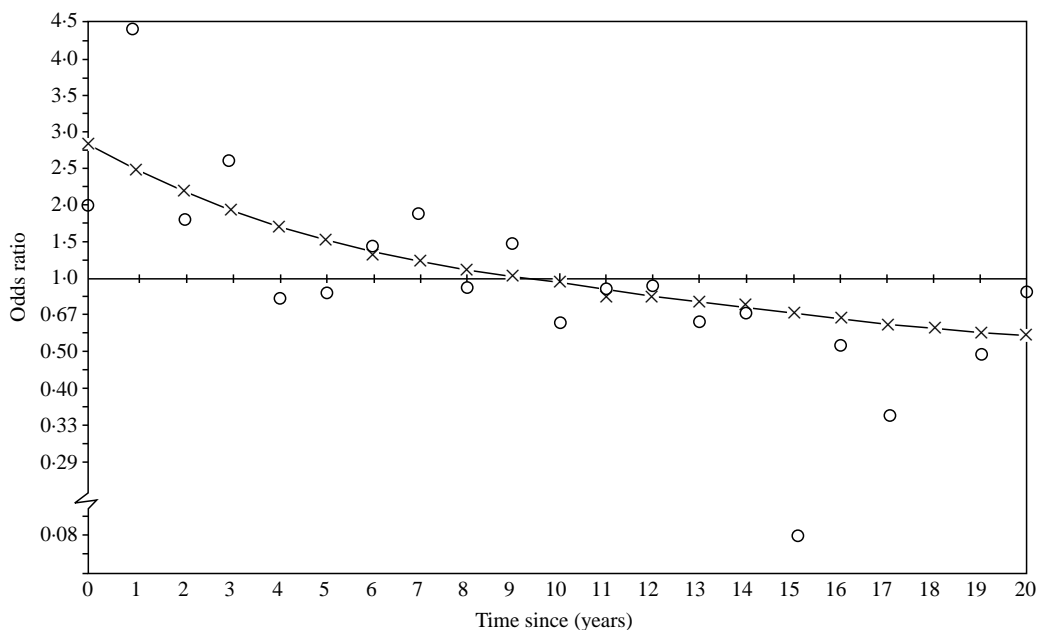
FIG. 1. Time decay in the association (odds ratio) between childhood adversities and the subsequent onset of any NCS/DSM-III-R disorders as a function of time since occurrence of the adversity, (○, observed; ×, predicted). (Only a subset of adversities was included in the analysis, consisting of all those found to have a significant time decay in bivariate models. These include parental separation/divorce, repeated molestation, repeated rape and physical attack. The cases used in the analysis were limited to respondents who reported the occurrence of either none or one and only one of these adversities. The measure of adversity was a time-varying predictor variable coded 0 for years prior to the year of the adversity's occurrence and 1 thereafter. Controls variables included all sociodemographics summarized in the footnote to Table 5.)

months. The additive model was not a significant improvement over the control model for any of the individual disorders, although it was significant in predicting a summary measure of any disorder. Furthermore, none of the equations for non-additive models was significant. Based on these results, we concluded that childhood adversities are generally not strongly related to the persistence of individual NCS/DSM-III-R disorders.

DISCUSSION
Limitations
Limitations of this report include the fact that results are based on a cross-sectional survey in which adversity was assessed retrospectively rather than prospectively, and the fact that we did not assess childhood or adolescent psychopathologies that might have intervened between childhood adversities and the adults disorders that were treated as outcomes. In addition, adversities were assessed with respondent-based

methods rather than more sensitive interviewer-based contextual rating methods. As noted in the methods section, some of the adversities were assessed with standard scales. These have been shown to have good reliability and validity (Endicott *et al.* 1978; Straus, 1979). However, the adversities that were assessed with checklists could well suffer from the same or more severe reliability and validity problems that have been shown to exist in checklists of recent life events (Kessler & Wethington, 1991). Another limitation is that our list of childhood adversities, although much more comprehensive than in previous studies of adults, was still incomplete. For example, we did not access illness of family members when respondents were children, foster care, loss of friendships, or death of a sibling. These limitations could well have affected our results. For example, our failure to find evidence that childhood adversities influence the persistence of disorders is inconsistent with the results of several previous studies that used contextual ratings of adversity (Brown & Moran, 1994; Brown, *et al.* 1994; Andrews *et al.* 1995)

and could have been due to the greater coarseness of our measures than those in these previous studies. The results need to be interpreted with these limitations in mind.

Prevalences

Significant childhood adversities were reported by three-fourths of respondents and multiple adversities were reported by more than half of respondents. Although most previous studies have not assessed such a large set of adversities, this finding of widespread exposure is consistent with the one earlier national survey in the US that assessed a similar range of childhood adversities (Kessler & Magee, 1993). It is conceivable that some reported adversities are due to false memories. However, as there were no apparent secondary gains associated with these reports it is more likely that adversities were under-reported due to recall failure and conscious unwillingness to disclose potentially embarrassing and painful memories.

Bivariate associations between adversities and disorders

The bivariate associations found here between childhood adversities and first onset of NCS/DSM-III-R disorders are similar to those found in previous studies in that they are consistently positive and for the most part both substantively important and statistically significant. Also consistent with previous research are the findings that parental marital break-up is a stronger predictor than other loss events (Tennant, 1988; Oakley-Browne *et al.* 1995*b*) and that sexual abuse is a stronger predictor than other interpersonal traumas (Bryer *et al.* 1987; Mullen *et al.* 1993; Mancini *et al.* 1995). However, the finding that maternal psychiatric disorders are somewhat stronger predictors than paternal disorders is inconsistent with the finding of no consistent effect of sex of parent in the small number of previous studies that have examined this issue (Merikangas *et al.* 1985; Reich *et al.* 1988).

As in previous retrospective studies, it is not clear that the bivariate associations documented here are causal. It might be that they are due to unmeasured common causes, that they are induced by retrospective reporting bias, or that some combination of causal and methodological influences is at work. To the extent that unmeasured common causes exist, furthermore, it is not necessarily the case that they are environmental in nature, as underlying genetic common causes could equally well be involved (Kendler *et al.* 1996).

The consequences of overlap among adversities

We noted in the introduction that most previous retrospective studies have examined the associations between only one or a small number of childhood adversities and only one or a small number of outcomes. It is not known whether these associations are direct, are partly explained by prior adversities or disorders, or are partly mediated by intervening adversities or disorders. Our results are consistent with the few recent studies that have examined this issue (Kessler & Magee, 1993; Mullen *et al.* 1993; Portegijs *et al.* 1996) in showing that there is considerable overlap among the adversities and that the majority of the ORs of individual adversities attenuate when controls are introduced for overlap. These results suggest that future studies should use a broad assessment of childhood adversities rather than a more focused assessments of only a small number of adversities.

It can also be argued, based on these results, that caution is needed not to interpret the results of previous studies too narrowly. A question can be raised, for example, about previous suggestions that there is something special about the childhood experience of parental loss that predisposes to the later onset of adult depression by creating a specifically depressogenic intrapsychological vulnerability (Harris *et al.* 1990). While this might be true, it would be a mistake to think that this is the dominant pathway linking childhood loss to adult depression based on our findings that these events are powerful predictors not only of mood disorders but also of other disorders and that many of the associations between early loss events and subsequent mood disorders are attenuated when we control for other adversities.

Despite this caution, though, we found that a number of ORs remained significant in multivariate models as well as in models that focused on respondents with pure adversities, a finding that is inconsistent with the conclusion in recent studies that the 'matrix of childhood disadvantage' (Mullen *et al.* 1996) from which clusters

of adversity emerge is more important than the component adversities that make up the cluster and that individual adversities generally do not importantly affect subsequent psychopathology when they occur alone rather than as part of this larger matrix.

It is important to appreciate that the adversities considered here varied in the extent to which they overlapped with larger clusters of adversity. This led to variation in the extent to which the ORs associated with particular adversities attenuated in multivariate models. For example, while the bivariate model suggested that parental marital break-up is a stronger predictor of subsequent disorders than the other childhood loss events, most of this difference was explained by the stronger clustering of parental break-up than other loss events with other adversities. Similar attenuation was also found in the effects of parental psychopathology and interpersonal traumas. This means that some part of the variation in the relative effects of different adversities found both in our study and in previous investigations is due to differential clustering rather than to differences in the unique effects of particular adversities.

Additivity of the effects of multiple adversities

We found that the effect of exposure to more than one adversity is additive in a multiple logistic regression model in which additivity is judged in terms of log-odds of probabilities. This means that there is a multiplicative effect in the probabilities (Rothman, 1986); that is, that the joint effects of multiple adversities are generally associated with a higher probability of onset than the sum of probability differences associated with each component adversity.

Due to the extremely large number of logically possible combinations of adversities, it will be important to use theory to guide future investigations of the joint effects of particular combinations of adversities and to cross-validate results in multiple samples. Although we have only begun to scratch the surface of the NCS in this regard, early work shows clearly that there are substantively important and powerful specifications to be found in this type of analysis. For example, preliminary results suggest that the significant effects of parental separation/divorce in predicting subsequent mood disorders and

addictive disorders are powerfully affected by whether or not there was parental violence and psychopathology in the household prior to the break-up and whether exposure to these adversities was reduced as a result of the separation (Kessler *et al.* 1997*a*). There are some situations – such as one in which the father was a violent alcoholic – where our data suggest that parental divorce and subsequent removal of the respondent from exposure to the father might actually be associated with a significant improvement in the respondent's subsequent disorder risk profile, a possibility that has important social policy implications.

The effects of co-morbidity

Previous analyses of the NCS have documented pervasive lifetime co-morbidity among the DSM disorders (Kessler, 1995), several strong clusters within the larger pattern of overall co-morbidity (e.g. a cluster involving panic, generalized anxiety disorder, and depression and a separate cluster involving alcoholism, adult antisocial behaviour and drug abuse) (Kessler, 1997) and evidence that temporally primary disorders within these clusters are powerful predictors of the subsequent onset of secondary disorders (Kessler, 1997; Kessler *et al.* 1996*a*, *b*, 1997*b*).

These results mean that any analysis aimed at determining whether there is specificity in the effects of particular childhood adversities on the subsequent onset of different psychiatric disorders needs to control for co-morbidity. In the absence of such controls, an association will be induced between prior adversities and the subsequent onset of secondary disorders when the adversities were actually influencing the primary disorders but not the secondary disorders. Consistent with this concern, we found that many of the bivariate ORs between adversities and disorder onset attenuated when we controlled for prior disorders.

The control analyses documented that the effects of particular adversities are not confined to any single class of outcome disorders. This is consistent with the findings of previous studies that have focused on individual outcome disorders and have documented effects of childhood adversity on depression (Oakley-Browne *et al.* 1995*b*), anxiety (Stein *et al.* 1996), borderline personality disorder (Paris *et al.* 1994), eating disorders (Andrews *et al.* 1995), and psychosis

(Greenfield *et al*. 1994). We also found that the relative effects of different adversities across different classes of disorders are more notable for their similarities than their differences after we control for lifetime co-morbidities. This result is consistent with the findings of the few previous studies that have examined the relative effects of multiple childhood adversities over a range of adult psychiatric outcomes (Bushnell *et al*. 1992; Mancini *et al*. 1995; Mullen *et al*. 1996).

Specifications of adversity effects have been found in some previous studies. For example, Portegijs *et al*. (1996) found in a survey of general practice patients with high consultation frequencies that childhood lack of care was related to adult somatization, while childhood abuse was related to adult depression. Specifications have also been found in studies of other outcomes. For example, Mullen *et al*. (1996) found that childhood sexual abuse was associated with adult sexual problems, while childhood physical abuse was related to adult relationship problems. The strongest specification in our data is that childhood loss events are more strongly associated with the subsequent onset of mood disorders than anxiety disorders. This result is consistent with both theoretical expectations (Freud, 1959; Bowlby, 1973) and past research (Finlay-Jones & Brown, 1981; Prudo *et al*. 1981; Tennant *et al*. 1982; Miller & Ingham, 1985; Rutter, 1989). However, loss events in the NCS were found to predict only mania and dysthymia, not major depression, and this is inconsistent with expectations based on the previous literature.

Distinguishing predictors of onset and persistence

As noted in the introduction, most previous retrospective studies have been concerned with the associations between childhood adversities and current adult disorders. Such a focus makes it impossible to know whether the adversities are associated with increased risk of disorder onset, with persistence, or with both onset and persistence. Our results show clearly that the adversities considered here are associated with risk of onset but not with persistence (recency of disorder). This result is consistent with recent work in other samples that has documented stronger associations of childhood adversities with first onset than with illness course (Kessler

& Magee, 1993, 1994; Kendler *et al*. 1993). It is inconsistent, though, with several studies that have documented associations between childhood adversities and chronicity of depression (Brown & Moran, 1994; Brown *et al*. 1994; Zlotnick *et al*. 1995).

Time decay in the risk associated with childhood adversities

Our finding that there are stronger associations of childhood adversities with early-onset than later-onset disorders is consistent with the results of our previous studies in smaller surveys (Kessler & Magee, 1993; 1994; Kessler *et al*. 1997*a*). However, the large sample size of the NCS made it possible to examine the shape of this time decay for the first time. The persistence of the elevated OR for a number of years after the occurrence of the childhood adversity is much longer than the lag effect of a few months typically found in adult studies of life events (Surtees, 1989), indirectly suggesting that there is some residual scarring effect that persists beyond the period that would normally be expected to provoke an onset of disorder. The fact that the ORs drop to 1·0 within a decade after the adversity occurred implies that any such scar is not permanent. This is an encouraging result that with further study might have implications for intervention.

The fact that the ORs produce an asymptote to a value lower than 1·0 is presumably due to a selection effect; that is, those respondents who do not develop a disorder despite exposure to a childhood adversity and after having passed through the risk period associated with such an adversity are more resilient than the typical person who was fortunate enough not to experience any childhood adversity. Whether this resilience is due to strengths that resulted from the adversity cannot be known from these data, although there is evidence for the development of strength through adversity in the literature (Elder 1974; Werner 1982; Garmezy 1990).

The importance of these time decay findings is compromised by the fact that our investigation of time decay was limited to an analysis of the aggregate effect of discrete-onset adversities on a summary measure of onset of any disorder. We do not know if the same shape of time decay exists for all adversities. Neither do we know

anything from this result about the shape of time decay of chronic childhood adversities. Furthermore, as we focused on people who experienced only one adversity, we know nothing about time-decay effects in the presence of other adversities, including those that might be part of a cascade of adverse experiences associated with a single core occurrence such as paternal violence leading to parental divorce leading to financial adversity.

Although it is beyond the scope of this initial report, future investigations need to evaluate these specifications. It would also be useful to distinguish effects on separate classes of outcome disorders and to take into consideration the influence of age of occurrence of discrete adversities on their subsequent pathogenic effects in the light of suggestive evidence in previous studies that the effects of childhood adversities depend importantly on the ages of their occurrence (Tennant *et al*. 1982).

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