Research Articles

CHILDHOOD ADVERSITY AND VULNERABILITY TO MOOD AND ANXIETY DISORDERS

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Based upon epidemiological surveys, adverse childhood events are proposed to be risk factors for adult depressive and anxiety disorders. However, the extent to which these events are seen in clinical patient populations is less clear. We examined the prevalence of a number of proposed risk factors for depression in 650 patients with mood and anxiety disorders at the time of presentation for treatment in an outpatient subspecialty clinic. Emotional abuse, physical abuse, or sexual abuse (childhood adversity) was found in approximately 35% of patients with major depression and panic disorder, was more common in women than men, and was associated with an earlier onset of symptoms. Childhood adversity was also strongly associated with marital discord/divorce, and psychopathology in a parent, suggesting family discord predisposes to childhood abuse. Furthermore, the association of childhood abuse with parental mental illness suggests that genetic and environmental factors are difficult to separate as etiological factors in vulnerability. Depression and Anxiety 5:66–72, 1997. *© 1997 Wiley-Liss, Inc.*

Key words: *childhood adversity; family discord; mood and anxiety disorders; emotional abuse; physical abuse; sexual abuse*

INTRODUCTION

Stressful life events clearly precede the onset of many episodes of depression or anxiety; but stress per se is not sufficient to account for the occurrence of depressive and anxiety disorders. Another key element is vulnerability (Brown and Harris, 1989; Breslau et al., 1991; Kessler et al., 1995). Though life events may trigger a depressive episode in a vulnerable individual, the vast majority of individuals experiencing stressful life events do not develop depression or stress-related anxiety disorders such as posttraumatic stress disorder (PTSD) (Brown and Harris, 1989; Breslau et al., 1991; Kessler et al., 1995). Brown and Harris (1989) reported that four-fifths of women who experienced a severe life event did not become depressed. Breslau et al. (1991) found that 39% of an epidemiological sample from a large HMO were exposed in a 12-month period to traumatic events that could cause PTSD; of those exposed, only 24% developed PTSD. Recent studies by Kessler et al. (1995) found an overall lifetime exposure rate to traumatic events of 60.7% for men and 51.2% for women, but a lifetime prevalence of PTSD of 5% for men and 10.4% for women. These epidemiological studies demonstrate that stressful life experiences are very common and that most individuals do not develop psychiatric disorders following stressful or traumatic life events in the absence of vulnerability factors.

Vulnerability could be genetic, but it could also be shaped by prior life experience. Numerous non-human studies have demonstrated that early separation from the mother can produce life-long abnormalities in both stress hormone regulation as well as abnormal behavioral responses to stress and social situations (Ladd et al., 1996; Hinde et al., 1978; Suomi et al., 1973; Meyer et al., 1975; Lyons and Levine, 1994), supporting a role for childhood developmental stres-

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sors leading to increased vulnerability to stress. Data from Kendler et al. (1993a) also support a role for developmental events (parental loss, lack of perceived parental warmth) and recent life events interacting with genetic factors in the onset of major depressive disorder. However, Kendler's data are based upon an all-female community sample. Childhood events that have been proposed to constitute risk factors for adult depression include loss of a parent through death or divorce, childhood abuse, parental psychopathology, and violence in the home. Recently Brown and Harris (1993) and Brown et al. (1993) have proposed that the specific childhood experiences of parental neglect/emotional abuse, physical abuse, and sexual abuse constituted clear risk factors for both Major Depression and Panic Disorder in women, suggesting that early life events may contribute to subsequent illness vulnerability in their all-female sample. Consequently, epidemiological data support developmental factors as contributing to vulnerability to depressive and anxiety disorders in women, but the relevance of childhood adversity to these disorders in men has received less attention. Furthermore, childhood events may be less relevant to clinical psychiatric populations where family history/genetic loading may be more important than developmental factors in the development of mood and anxiety disorders. The prevalence of childhood abuse is underemphasized in current biological psychiatry, and its relevance for patients in subspecialty clinics not well addressed. If the prevalence of childhood abuse is as common in mood and anxiety patients as suggested by epidemiological data, then additional studies may need to address its relevance in treatment response to medications as well as psychotherapy, and further consideration given to co-morbidity with other disorders, especially personality disorders. Finally, developmental events may play a role in definition of pre-morbid function and in predicting which patients may be able to maintain gains with short-term treatments vs. who may need more long-term pharmacotherapy and psychotherapy.

The following analyses were conducted to determine the relevance of the Brown and Harris childhood adversity/vulnerability model to the patient population seen in a subspecialty clinical setting, where we expect that the patient population would be quite different than either an epidemiological sample or a primary care sample with respect to precipitating factors, age of onset, and family history of psychiatric disorders. We have collected information on multiple childhood experiences, stressful life experiences, and parental history of psychiatric disorders and substance abuse disorders on all patients seen in the past 6 years in an anxiety disorders subspecialty clinic at the University of Michigan. In the current analyses, we examine whether the incidence of childhood adversity from a clinical population is similar to that of Brown and Harris' data (1993) from an epidemiological sample, i.e., whether childhood adversity is also a common finding in both mood and anxiety patients seen in this clinic. We further compared the incidence of these adverse childhood experiences in women and men, to determine if childhood experiences are important for the development of mood and anxiety disorders in men as well as women. Finally, we examined the relationshp between childhood adversity and age of onset of these disorders and symptom severity.

MATERIALS AND METHODS

SUBJECT SELECTION

Data were collected on all patients who presented for evaluation at the University of Michigan Anxiety Disorders Program. All subjects were adults at the time of the evaluation. Subjects were briefly screened by the evaluating clinician over the telephone to be sure that they had some anxiety symptoms and were not psychotic, but no specific exclusion criteria were used for entry to the initial evaluation stage.

DATA COLLECTION

A standardized form with 120 clinician rated variables was used to collect information during a clinical interview on factors relevant to the onset and course of illness, life event stressors, co-morbid psychiatric symptoms, childhood events, and family history information. The clinicians responsible for rating symptoms and collecting data were 3 clinical nurse specialists, one senior master's level social worker, and a small number of fourth-year Psychiatry residents. In all cases, a senior level psychiatrist (R.M.N. or G.C.) also saw the patient and confirmed both the diagnosis and main clinical and developmental features gathered and entered into the database. Variables were rated as continuous rather than categorical variables whenever possible with "1" defined as not at all present, "2" as mild, "3" as moderate, "4" as severe, and "5" as very severe. Clinicians rated the severity of each event based upon how an average child would respond, not based upon the patient's subjective report of its impact. While diagnoses were based upon clinical interview rather than structured interview (SCID), the clinicians responsible for evaluation were also trained SCID interviewers and a small proportion of the patients also participated in ongoing biological research studies for which they received both a clinical and SCID interview. Diagnostic agreement between the clinical interview and SCID interview was greater than 90%.

DATA ANALYSIS

The database contains entries on childhood experiences for approximately 1,000 individuals. We restricted the analysis to subjects with mood and anxiety disorders with complete data on all the factors for a total of 650 cases. The final data set contained entries on 338 patients with Panic Disorder, 177 patients with

Obsessive Compulsive Disorder, 103 patients with Major Depression, 33 patients specific phobias, and 29 patients with generalized anxiety disorder. The patients with a primary anxiety disorder diagnosis did not meet criteria currently for Major Depression, although some individuals may have met criteria for depression in the past. Of the 103 patients with Major Depression, 45 had at least one panic attack in their lifetime, but only 24 had experienced 2 or more panic attacks in the last month. Some variables were transformed from continuous to categorical variables (absent or present) for statistical analyses. These categorical variables were: emotional abuse/neglect, physical abuse, sexual abuse, marital conflict/divorce, childhood separation, psychopathology in the parent, and substance abuse in the parent. The definition of "present" was a score of 3 (moderate) or greater on the continuous variable except for parental substance abuse and psychopathology, which we coded as present if scored 2 (mild) or greater. An additional summary categorical variable of "childhood adversity," which consisted of either emotional abuse, physical abuse, or sexual abuse, was created for these analyses. Relevant continuous variables included age of first symptoms, age when subject first met DMS-IIIR criteria for the disorder, and severity of symptoms over the lifetime. Symptoms rated include depressed mood, panic attacks, general anxiety symptoms, alcohol abuse, and substance abuse. Statistical analyses consisted of Chi-square for categorical variables and ANOVA with post-hoc testing (Bonferroni corrected Dunn) for continuous variables and McNear's test for comparison of frequencies as further indicated. The actual *P* values are reported in Tables 1–4. The results of all analyses conducted, whether significant or not, are presented in this paper.

RESULTS

Initial analyses examined the frequency of childhood risk factors that have been associated with depression. Table 1 lists the prevalence of these risk factors and the combined variable childhood adversity in our patient population by diagnostic categories. The most common adverse childhood events are psychopathology in parent and substance abuse in parent. Only substance abuse in parent demonstrated diagnostic specificity, with significantly higher occurrences in patients with Major Depression and Panic Disorder.

Table 1B includes the data from Brown and Harris (1993) on emotional abuse (patental indifference), physical abuse, sexual abuse, and the summary variable childhood adversity in an epidemiological sample with depression and anxiety for comparison to our data. Similar to the data of Brown and Harris (Table 1B), in our data emotional abuse is the most common and sexual abuse the least common event ($P < .0001$ for comparison between 3 types of abuse by McNemar's test). The prevalence rate of childhood adversity is quite similar in the clinical and epidemiological samples for major depression, but differed for panic disorder (Table 1; McNemar's test, $P < .05$). The high incidence of childhood adversity in specific phobias was surprising. Because situational phobia may be more closely linked to panic/agoraphobia spectrum disorders, we examined these separately from small phobias, but the two types of phobias showed similar rates of childhood adversity.

Since Brown and Harris' sample consisted only of

TABLE 1. Prevalence of adverse childhood (CH) events in mood and anxiety disorder patients (%)

	A. University of Michigan data					
	GAD (29 cases)	Panic disorder (338 cases)	Specific phobia (33 cases)	Major depression (103 cases)	OCD (177 cases)	Total (680 cases)
Emotional abuse	17	32	33	34	28	31
Physical abuse	10	16	18	20	16	16
Sexual abuse	0	9	12	12	7	9
CH adversity	17	36	36	39	33	35
Marital conflict/divorce	28	43	39	43	49	43
Separation	24	25	24	24	21	23
Psychopathology in parent	45	49	30	59	51	50
Substance abuse parent*	17	40	24	39	24	34

	B. Brown and Harris data		
	Anxiety (92 cases)	Depression (125 cases)	Controls (279 cases)
Parental indifference	32	24	11
Physical abuse	31	24	10
Sexual abuse	15	16	3
CH adversity	48	39	17

**Chi-square*, P = *.0001*.

women, we divided our sample by sex to address sex differences in these adverse childhood events (Table 2). As might be expected, sexual abuse ($P = .0006$, Chi-square), physical abuse ($P = .0003$), emotional abuse ($P = .0001$), and childhood adversity ($P = .0001$) are significantly more common in the female patients than male patients. Note that the incidence of childhood adversity in the female panic disorder patients (41.6%) is no longer different ($P > .05$) than the incidence of 48% observed by Brown and Harris for panic disorder in their all-female sample.

Predictably, the various types of adverse events are not independent of each other. For example, when either physical or sexual abuse was present, emotional aubse was likely to be present (90% of cases with physical abuse and 34% of cases with sexual abuse). There was also a significant relationships between childhood adversity and marital conflicts/divorce and childhood separations. Marital conflict/divorce was present in 72% of the sample with childhood adversity but only 28% of the sample without childhood adversity (significant by Chi-square, $P = .0001$). Similarly, childhood separation was present in 35% of the cases with childhood adversity, while it was present in only 16% of the cases without any adverse childhood events (Chi-square, $P = .0001$). Psychopathology in a parent was significantly associated with marital conflict in both directions (60% of cases with parental psychopathology also had marital conflict; 72% of the cases with marital conflict also had parental psychopathology, Chi-square, $P \leq .0001$); and parental psychopathology was present in 77% of cases with childhood adversity ($P < .0001$). Marital conflict was found in 62% of the cases with substance abuse in a parent (Chi-square, $P < .0001$). Finally, only 39% of the Patients experienced neither parental psychopathology nor parental substance abuse in their childhood.

We examined the effect of adverse childhood events on the age of onset of symptoms, predicting that exposure to childhood adversity would be associated with an earlier age of onset. Table 3 presents the data for the entire populaton (all diagnoses) with age of first symptoms and age first met full DSM-IIIR criteria for the diagnosis. As can be seen, emotional abuse, sexual abuse, the summary variable childhood adversity, and parental marital conflict/divorce all predispose to an earlier age of onset of symptoms, approximately 2 years earlier in each case. Note that the 2-year earlier age of onset also occurs for physical abuse although it is not statistically significant. In contrast, only sexual abuse led to an earlier age of meeting full criteria for a mood or anxiety disorder, where both symptom onset and full syndromal onset occur 3.9 years earlier in patients with sexual abuse. If we examine the data by specific diagnoses, for Major De-

TABLE 2. Sex differences in childhood (CH) adversity (%)

	Females only			
	Panic disorder (238 cases)	Specific phobia (26 cases)	Major depression (78 cases)	OCD (112 cases)
Emotional abuse	39	34	41	19
Physical abuse	19	15	27	12.6
Sexual abuse	12.7	7.6	14	14
CH adversity	41.6	30.7	43.6	37.5

	Males only			
	Panic disorder (108 cases)	Specific phobia (10 cases)	Major depression (26 cases)	OCD (82 cases)
Emotional abuse	22	37.5	20.8	18
Physical abuse	11.3	20	11.5	8
Sexual abuse	3	0	8	3
CH adversity	31	30	23	17.5

TABLE 3. Relationship between childhood events, age sex onset, age met DSM IIIR criteria

		Age first symptom	Age met criteria
Emotional abuse	Present	19.6 ± 0.8****	23.6 ± 0.8
	Absent	21.8 ± 0.5	24.2 ± 0.5
Physical abuse	Present	19.4 ± 1	21.8 ± 0.99
	Absent	21.2 ± 0.4	23.6 ± 0.4
Sexual abuse	Present	17.6 ± 1***	19.8 ± 1*****
	Absent	21.3 ± 0.4	23.7 ± 0.4
CH adversity	Present	19.5 ± 0.7**	22.9 ± 0.7
	Absent	21.6 ± 0.5	23.5 ± 0.5
Marital conflict/divorce	Present	19.6 ± 0.6*	22.5 ± 0.6
	Absent	21.9 ± 0.5	23.9 ± 0.5

*P = *0.005*.
**P =*0.02*.
***P = *0.04*.
****P = *0.0066*.
*****P = *0.0036*.

pression sexual abuse again led to a 9.8-year earlier age of first symptom (mean age 17.8 for sexually abused patients, P = .00027 by Chi-square) and 11-year earlier age at which first met criteria (mean age 19.7 for sexually abused patients, P = .015 by Chi-square); childhood adversity also led to a 7.5-year earlier age at first symptom (mean age 21.5 for patients with adversity, P = .0098 by Chi-square). For Panic Disorder, sexual abuse led to a 4-year earlier onset of symptoms (mean age 19.5 for sexually abused patients, (P = .01 by Chi-square) and a 4-year earlier age at which first met criteria (mean age 22.1 for sexually abused patients, P = .02 by Chi-square); marital conflict/divorce also affected age of first symptoms (mean age 21.6 for patients with family conflict, P = .00067 by Chi-square), while childhood adversity had little effect. Finally, for OCD, none of these adverse childhood events affected age of onset of symptoms and age when first met full criteria (mean age first symptom 12–14 years for both groups of patients).

The final set of data analyses examined the influence of a childhood adversity on lifetime ratings of depression, panic attack severity, generalized anxiety, and alcohol/substance abuse across the entire patient population (Table 4). Although for every disorder, the presence of childhood adversity significantly increased the severity of these symptoms, the impact of childhood adversity upon depressive symptoms is the greatest.

DISCUSSION

The main findings of these analyses can be summarized as follows: (1) childhood abuse was extremely common in a population of anxiety and mood disorder patients presenting for treatment in a subspecialty clinic, with no major differences between diagnostic groups; (2) these adverse events appear to be linked with marital conflict/divorce and psychiatric problems in a parent, suggesting that they occur in the context of family discord; (3) adverse childhood events are more common in women; (4) adverse childhood events were associated with a significantly earlier age of onset of symptoms and, in the case of sexual abuse, a significantly earlier age meeting DSM-IIIR criteria; and (5) the presence of childhood abuse was associated with a significant increase in lifetime severity of a number of psychiatric symptoms, across all diagnoses.

TABLE 4. Effect of childhood (CH) adversity on lifetime ratings of symptom severity

	CH adversity positive	CH adversity negative
Depression	3.2 ± 0.07	2.5 ± 0.05*
Panic attacks	3.1 ± 0.09	2.9 ± 0.07
GAD sx	2.3 ± 0.07	2.0 ± 0.05**
Alcohol abuse	1.7 ± 0.07	1.5 ± 0.04***
Substance abuse	1.5 ± 0.06	1.3 ± .03****

*P < *0.0001*, **P = *0.007*, ***P = *0.0003*, ****P = *0.008*.

This study sought to replicate the community-based findings of Brown and Harris on childhood adversity to determine whether these adverse childhood experiences were also common in a clinical sample. We were impressed by the similarity of the two data sets; the association of childhood abuse with marital conflict/violence and separation from parent also replicates their work. Our finding of an additional association of childhood adversity with parental substance abuse further suggests that it is not a single factor, but a combination of developmental factors that is strongly associated with later vulnerability to mood and anxiety disorders. This conclusion is supported by the work of a number of others suggesting that it is the associated disruption in parental care rather than loss per se that is associated with adult psychopathology (Tennant, 1988; Breier et al., 1988; Favarelli et al., 1986; Parker, 1983; Rutter, 1972). While the current study did not have a "control" group of non-psychiatric case controls, both the data of Brown and Harris (1993) shown in Table 1B and the recent large-scale National Comorbidity Survey of Kessler and colleagues (1995) demonstrate that the incidence of physical abuse and emotional abuse/neglect are much lower in the general population than reported in these clinical populations. The National Comorbidity Survey, which interviewed almost 6,000 individuals, estimated the prevalence of childhood physical abuse and neglect to range from 2.1–4.8% (Kessler et al., 1995), while the Brown and Harris sample (1993), which shows a prevalence of abuse of 17%, consisted of women only and a lower socioeconomic class only, two factors which would likely increase the prevalence of childhood abuse observed in their sample. Kessler et al. (1995) also reported data on the incidence of rape and sexual molestation in their sample of 6,000, but did not separate these data into childhood and adult ages, so a comparison of our childhood sexual abuse with their population data is not possible. However, our data and that of Brown and Harris suggest that childhood sexual abuse is less common than emotional and physical abuse.

Kessler's epidemiological data also demonstrated a significantly greater prevalence of sexual abuse, childhood physical abuse, and childhood neglect in women, in agreement with our clinical population data (Kessler et al., 1995). Despite the greater prevalence of childhood abuse in women, this factor is unlikely to account for the greater prevalence of mood and anxiety disorders in women, since childhood abuse was more common in women than men for all psychiatric disorders examined including OCD, despite the equal prevalence of OCD in men and women. Additionally, the data of Breslau et al. (1991) and Kessler et al. (1995) suggest that following exposure to a trauma, women are more likely than men to develop PTSD symptoms, suggesting that gender contributes to vulnerability beyond more frequent exposure.

As might be expected, childhood advesity influ-

enced the age of onset of symptoms for these disorders. When we examined separately the elements of childhood adversity as well as the summary variable by each diagnosis, we found that sexual abuse ws the most striking in terms of influencing both age of onset of symptoms and age meeting full criteria. However, for Major Depression, the summary variable childhood adversity was also very important in affecting the age of first symptoms, with both childhood adversity and sexual abuse leading to symptom onset 8–10 years earlier than for those without these adverse experience. These data agree with those of Kessler and Magee (1993) who found that adverse events in childhood, particularly parental alcohol abuse, parental mental illness, and the death of either parent, significantly increased the odds ratio for depression with onset before age 20, but not for later onset depression. In contrast, violence in the home and parental marital problems increased the odds ratio for both early and later onset depression (Kessler and Magee, 1993). Finally, the importance of intervening factors in mediating the effects of adverse childhood events need to be considered. For example, parental divorce may influence the liability for adult depression by affecting choice of marital partner, age of marriage, and educational opportunities (Harris et al., 1987).

While a number of previous studies have focued upon depression, the degree to which these adverse childhood events are risk factors specific for depression is still open to question. Thus, Brown and Harris reported that childhood adversity was a risk factor for the development of both major depression and panic disorder (1993). Recent analyses by Kessler et al. (1997) of the National Comorbidity Survey found that childhood adversity was significantly and positively associated with a recent prevalence of almost all psychiatric disorders, and that the effect was strongest for early onset cases than later onset cases. Kendler et al. (1992) found that parental loss prior to the age of 17 was significantly related to mood and anxiety disorders including phobias with no diagnostic specificity among these disorders. From the data presented here, childhood adversity was common in all disorders and affected the age of onset for both depression and Panic Disorder. Finally, for OCD, none of these adverse childhood events affected age of onset of symptoms and age when patients first met full criteria. This may be because the mean age of first symptoms for OCD in our patient population is quite young, occurring in the teenage years. Even sexual abuse did not have a significant effect in this population. In many cases, the onset of OCD symptoms are occurring during the period of greatest risk for sexual abuse and thus sexual abuse might not be expected to affect the age of onset to the same degree as these other disorders. This is further supported by the fact that the disorder with a latest age of onset, depression, shows the greatest effect of adverse childhood experiences on the age of symptom onset. However, given that these data are collected retrospectively, there may be difficulties in defining age of onset of symptoms when the time interval can be 15–20 years between onset and presentation for treatment.

The relationship between adverse childhood experiences and genetic factors in the development of vulnerability is a key to understanding what constitutes vulnerability. From these data analyses, it appears that parental psychopathology is associated with childhood adversity, so that genetic factors may predispose to both the disorder and to the verbal and physical abuse behaviors in the parent, as suggested by Kendler and colleagues (Kendler et al., 1992, 1993b, 1996). In his twin sample, Kendler et al. was able to examine parental loss as a specified environmental risk factor and found that while it explained 2–5% of the variance in liability for major depression, the genetic contribution is approximately 25 times greater, reflecting the difficulty in separating environmental from genetic factors (Kendler et al., 1992). However, for alcoholism, parental loss was found to be a significant and direct risk factor for the development of alcoholism, underscoring the need to analyze these risk factors separately for different psychiatric disorders (Kendler et al., 1996). Furthermore, these adverse events may be functioning as "stressful life events," which precipitate depressive and anxiety disorders at the time of their occurrence in genetically predisposed individuals, which would agree with our earlier age of onset data. Thus, more comprehensive and sophisticated studies are necessary to disentangle the genetic and environmental components of these disorders and the role of these adverse childhood events in vulnerability.

Finally, we examined the effects of childhood adversity on symptom severity. While the severity of anxiety symptoms was slightly increased, the effect of childhood adversity was most profound on the severity of depressive symptoms, whether or not diagnostic criteria for depressive disorder were met. Of note is the fact that these childhood traumas only slightly increased alcohol and substance abuse, a finding that may be surprising to clinicians who often see significant childhood abuse in individuals with these diagnoses. However, subjects with prominent current alcohol and substance abuse would not routinely be seen in this clinic, so these data may not be representative of patients with primary substance abuse problems.

In conclusion, the data presented here reveal substantial evidence of adverse childhood events in mood and anxiety clinic patients. Childhood adversity appears to be linked to marital conflict/divorce and psychopathology in a parent, suggesting that disturbed family environments may set up the conditions for emotional abuse. The high prevalence of psychiatric disorders and substance abuse in the general population in recent epidemiological studies (48%) (Kessler et al., 1994), suggests that the majority of families will have at least one parent who is affected, so the degree to which these risk factors are really developmental

factors as opposed to genetic factors will be crucial in future explorations of mood and anxiety disorders. Other unanswered questions include whether the presence of both genetic factors and developmental traumas are additive leading to a different clinical course, earlier onset of illness, and to stressor sensitization as has been proposed for recurrent affective illness by Post (1992). Finally, our study is retrospective and depends upon patient recall and cooperation in assessing childhood abuse. We do not know how the presence of a psychiatric disorder may influence this recall process, including whether cognitive distortions may influence the descriptions of childhood events or whether different diagnoses may affect the recall process differently. Prospective studies into adulthood of children at risk will be needed to answer many of these questions.

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