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SPECIAL ISSUE

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The contribution of epidemiology to the study of traumatic stress

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Abstract Post-traumatic stress disorder (PTSD) has been a controversial construct because of the complex set of factors that have been hypothesized to influence its onset and prevalence, such as compensation and withdrawal from combat duty. Epidemiology has done much to objectively clarify these controversies in the study of stratified population samples. The symptoms characterizing PTSD have been repeatedly described in large population samples where compensation is not a confounding issue and this has done much to support the validity of the construct. Epidemiology has also highlighted that the prevalence of exposure to traumatic events is far greater than was previously estimated. Emphasizing the importance of these events is accounting for the major burden of disease. Kessler (2000) has suggested the socio-economic effects of PTSD represent a burden of disease not dissimilar to that associated with depression. Traumatic events provide a unique opportunity to implement a preventative and public health approach to the management of psychiatric morbidity. Of particular importance is the apparent longevity of the influence that these events have on psychological adjustment.

Key words PTSD – epidemiology – disability – suicide – alcohol abuse

Introduction

More than any other discipline, epidemiology has done much to clarify the controversy about the legitimacy of PTSD. As an observational science, it has provided evidence concerning the prevalence and longitudinal course of PTSD, thereby clarifying the many controversies and social factors that have confused clinical observations in this field.

PTSD was first defined in DSM-III (APA 1980) to describe a specific pattern of symptomatic distress characterized by intrusive distressing recollections of the traumatic event associated with hyperarousal, estrangement and numbing. This has provided a major focus for a significant body of research into this condition. DSM-III embodied the notion that there was a single entity following traumatic events. This perspective articulated that there was a common final pathway in response to extremely traumatic events, in contrast to the use of terms such as the "rape syndrome", "war neurosis" and "concentration camp syndrome", to name a few of the descriptive titles that have been given. The idea that there was a common syndrome was based upon observations about the similarity and consistency of the phenomenology independent of the nature of the initiating stressor.

Confounds of observation

The idea that there was a single syndrome following these events was not a new idea. Oppenheimer (1890) had first coined the term *traumatic neurosis*. In the Annals of Medical Science, Seguin (1890) had said 'It would do much to finally set the status of this topic if those terms, "railway spine", "railway brain", "compensation neurosis", as well as the words "concussion", and "hysteria" were dropped'.

Much of the uncertainty and controversy that has surrounded this field has arisen because of the multiple factors that have influenced the conceptualization of traumatic syndromes (McFarlane 2000). It is always presumed that clinicians are able in a careful and scientific way to observe the phenomenology of patients. However, this field, perhaps more than any, demonstrates that the observational framework of a clinician is sub-

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stantially biased by a variety of social concerns and political factors.

Firstly, models of psychopathology are critical. The emergingly dominant psychoanalysis in the first half of the twentieth century focused on the importance of early developmental experiences, and did not address the capacity of experience in adult life to have a major impact on people's psychological function. This was despite the fact that Freud (1922) emphasized that traumatic neurosis was a different phenomenon from the broad body of psychopathology. In particular in relation to the veterans of World War I, the predominance of psychoanalytic-thinking focused on blaming early developmental factors as the primary aetiological force in the majority of patients despite the horrific experiences they had faced in combat (Glass 1974).

A second major factor intruding into the patient/clinician relationship was compensation law. Post-traumatic syndromes entered the domain of compensation through the notion of 'nervous shock'. Both in compensation claims after civilian accidents and in the setting of the chronic disability of war, one view was that this pathology was substantially attributable to the existence of financial compensation (DeViva and Bloem 2003). Hence, the fact that a social system supported the damaged individual was seen to be the driver of the disorder, rather than the primary traumatic experience (Seguin 1890).

The third factor influencing the clinician is the experimental paradigms that exist and their capacity to elucidate causal relationships (Kuhn et al. 1988). The substantial amount of knowledge about the nature of stress in animal populations has been significant in providing a paradigm for a better understanding of the nature of the traumatic stress response (Yehuda and McFarlane 1997).

Finally, there are larger social forces articulating and advocating the specific interests of various groups. War veterans and women who have been raped were seen to have been poorly served by mental health professionals (Herman 1992). This advocacy came to bear on clinician's perspectives of traumatic stress syndromes. These factors have combined to obfuscate and confuse the observations made about the victims of traumatic events.

Types of epidemiological studies

There are broadly five different types of epidemiological study that have been conducted into the effects of traumatic events.

Specific traumatic events

The early literature focused on specific disasters. Stierlin (1911), a Swiss psychiatrist, who described the pattern of symptomatology in the victims of the Messina earthquake in Italy in 1907, conducted the first study of a disaster. In the intervening years, there were intermittent reports of other events, such as the Coconut Grove fire (Adler 1943), which built upon Caplan's model of crisis intervention.

Following the definition of PTSD in DSM-III, studies were conducted into a series of disasters, such as the Buffalo Creek disaster (Green et al. 1990), the Ash Wednesday Bushfires (McFarlane 1988) and a factory fire in Norway (Weisaeth 1989), which provided within defined populations some of the early evidence about the prevalence of PTSD and its aetiology. In more recent times, much larger and comprehensive studies have been conducted after events such as September 11th (Galea et al. 2002) and Hurricane Andrew (Khoury et al. 1997). These studies have provided particular insights into the impact of the gradient of exposure and a variety of social factors as being critical determinants of prevalence and course.

Specific occupational groups

The second body of research that has played a continuing role in the understanding of PTSD has been of particular occupational groups. War veterans are the group who have been examined the most. The military has a major interest in understanding the aetiology and prevalence of these conditions. Also, war veterans are a group with significant political power, and have been able to argue for careful description of the nature and prevalence of psychopathology. For example, the NVVRS (Kulka et al. 1990), a study mandated by the US Congress, examined the prevalence of psychopathology amongst 3016 Vietnam veterans. This indicated that 15% of all males who were involved in active war service had a current PTSD. In contrast, the Centre for Disease Control Study (1988) examined 2490 Vietnam veterans and 1170 soldiers who did not serve in Vietnam and found that about half of the veterans reported experiencing one or more symptoms related to trauma combat events, while 15% met the diagnostic criteria for PTSD at some time during or after service. However, only 2.2% were still suffering from the disorder during the month prior to interview. In addition, Vietnam veterans were more likely to meet the diagnostic criteria for alcohol dependency, generalized anxiety disorder and depression.

Subsequent to the 1990 Gulf War when Iraq invaded Kuwait, a series of major studies have been conducted on the veterans from a number of countries, to explore the nature of unexplained medical symptoms as well as the prevalence of psychiatric morbidity (Ikin et al. in press). Emergency service workers have also been the focus of epidemiological studies because of the particular occupational hazards they face and the resultant prevalence of psychiatric morbidity that emerges (McFarlane 1989).

Victims of specific types of events

Victims of particular types of small-scale traumatic events have also become the increasing focus of interest.

In contrast to disasters, these individuals have been involved in events and situations that involve individuals or small groups. The largest body of research has been conducted on motor accident victims and victims of crime (Bryant and Harvey 2003; Ehlers et al. 1998; Kilpatrick and Resnick 1993). These events represent individual disasters in which the same communal and collective interest is not evoked. However, they are responsible for considerable morbidity. They also provide the opportunity to conduct longitudinal studies of the onset and course of these conditions. Refugees are another group that have provoked particular interest, because they elucidate the role of cultural and social dislocation in the onset of a disorder (Steel et al. 2002).

However, these studies are often conducted in the setting of financial compensation to the victims, and have left some of the uncertainties about the impact of compensation systems on patients' distress.

Stratified population samples

Stratified national population samples that have been used to examine the prevalence of psychiatric morbidity have done a great deal to clarify and address some of these unknown controversies (De Girolamo and McFarlane 1996). Studies such as the Epidemiological Catchment Area Study, the National Comorbidity Study (Kessler et al. 1995) and the Australian National Mental Health and Wellbeing Survey (Creamer et al. 2001) have addressed these issues. The majority of the more recent studies have depended upon the Composite International Diagnostic Interview (CIDI) (Peters and Andrews 1995) as the instrument for defining the prevalence of psychopathology.

Prospective population samples

An attempt to try and clarify a number of the methodological issues identified in critiquing these larger studies has led to the investigation of prospective samples. Most noteworthy is the study of young adults in Detroit by Breslau et al. (2003). This study has done much to clarify issues such as the role of traumatic events in contributing to substance abuse and somatization, as well as interaction with other risk factors.

Contributions of epidemiology

The prevalence of traumatic events

The investigation of stratified population samples has brought a scientific approach to illuminate many of the controversies about the impact of traumatic events. DSM-IIIR (1987) defined traumatic events as "being outside the range of normal human experience". This arose from the assumption that disasters at a communal level, and extreme violence and horror were isolated experiences within our communities. Whilst there were many studies of life events in general using scales such as those of Holmes and Rahe (1967), there had been no systematic attempt to define the prevalence of these phenomena in the broader community.

The strategy used in the National Comorbidity Study (NCS) of listing the specific types of events that may precipitate PTSD led to the dramatic observation that, in the USA, ranges from 41.2 % of women and 61 % of men had been exposed to traumatic events (Kessler et al. 1995). Such findings have now been replicated in a number of epidemiological studies in other cultures, such as Australia. Creamer et al. (2001) found very similar rates of traumatic experiences with 49.5 % of women and 64.6 % of males having experienced at least one event. With the exception of female rape, the relative prevalence of each category of traumatic experience was very close across both studies. Other studies have found trauma rates as high as 84 % (Vrana and Lauterbach 1994). By no means will all of the people who have experienced trauma go on to develop PTSD. The exact prevalence of the disorder in the community, however, remains a matter of some debate. Findings across studies are often hard to compare owing to different methodologies, the use of different diagnostic criteria, and the fact that some studies report lifetime rates while others report current rates of the disorder. Epidemiology has, therefore, demonstrated that the incidence of traumatic events is much greater than anticipated.

The probability of developing PTSD has been shown to depend upon the nature of the traumatic experience. The NCS (Kessler et al. 1995) reported a consistently high prevalence of PTSD among rape victims. Approximately 65 % of men who had been raped, and 46 % of women, qualified for a diagnosis of PTSD at some stage in their lives. Other high-risk categories of experience were combat or physical abuse for men (39 % and 22 %, respectively) and physical abuse or being threatened with a weapon for women (49 % and 33 %, respectively). Similarly, the Detroit Area Survey of Trauma (Breslau 1998) found that the highest risk of PTSD was associated with violent assault (which included rape).

Outside the field of traumatic stress, psychiatry has been slow to assimilate this observation because it indicates that traumatic events may play an important role in understanding the aetiology and course of psychopathology above and beyond PTSD. Studies such as that of Breslau et al. (1997) have found even higher rates of trauma exposure because of the propensity of people to under-report these experiences.

The prevalence of morbidity in the absence of compensation

The observations about the prevalence of PTSD in civilian populations who are not involved in compensation have done much to clarify the prevalence and chronicity of this disorder where it cannot be attributed to compensation or malingering. Some earlier studies, such as the Epidemiologic Catchment Area (ECA) Program,

found relatively low lifetime prevalence rates (based on DSM-III criteria) of between 1 % and 1.3 % (Helzer et al. 1987; Davidson et al. 1991). Later research using DSM-IIIR criteria has generally reported higher rates of lifetime prevalence. Breslau and her colleagues, for example, in a study of young adults from Detroit, found that 11 % of women (31 % of those exposed to trauma) and 6 % of men (14 % of those exposed to trauma) had a lifetime history of PTSD (Breslau et al. 1991). Similarly, in a national telephone survey of women, 12 % of respondents (18 % of those exposed to trauma) were found to have a lifetime diagnosis of PTSD (Resnick et al. 1993). The NCS (Kessler et al. 1995) found a PTSD lifetime prevalence of 7.8 % in the total sample, with the rate for women being twice that of men (10.4 % vs. 5 %). One of the few studies to adopt the more recent DSM-IV criteria (APA 1994) found a current prevalence of 2.7 % for women and 1.2 % for men among a Canadian community sample (Stein et al. 1997). In a German community sample of young people (aged 14–24), Perkonigg et al. (2000), also using DSM-IV criteria, found relatively low current rates of 1 % for males and 2.2 % for females.

Overall 12-month PTSD prevalence rates from the NCS are available (Kessler et al. 1999); however, they reveal three times the rate of PTSD in the American compared with the Australian sample (3.9 % vs. 1.3 %) (Creamer et al. 2001). This difference is not explained by the frequency of trauma exposure since the Australian sample (both men and women) had slightly higher exposure rates than their American counterparts. There is evidence elsewhere to suggest that higher 12-month rates of DSM-IV mental disorders among the American compared to the Australian population are not restricted to PTSD, although the difference may be more marked in PTSD than in other disorders (Kessler et al. 1995; Andrews et al. 2001).

The issue of delayed onset

One of the major impediments to understanding the effect of traumatic events has been the conceptual challenge of classifying individuals who manage to deal with the stress of the acute event, but at a time some months or years later develop psychiatric symptoms. One presumption was that it was very difficult to attribute symptoms that emerged at a later time to an event in the past. Longitudinal studies have done much to clarify the question of delayed/late onset of PTSD, which is defined in the DSM-IV (APA 1994) as a disorder meeting the diagnostic criteria for PTSD which is present after a post-trauma adjustment period of at least 6 months during which diagnostic criteria were absent or sub-threshold (Buckley et al. 1996).

Although delayed-onset PTSD has now been recognized by the American Psychiatric Association as a legitimate sub-category of PTSD in the DSM-IV and DSM-III, a number of alternative hypotheses were proposed to explain the delayed emergence of these symptoms. These included the hypotheses that the victims were malingering in order to gain financial compensation (Atkinson et al. 1982) or that the symptoms were actually reflecting other types of post-combat psychopathology such as alcohol abuse (Atkinson et al. 1982; Sparr and Pankratz 1983). Other authors have suggested that the presentation of PTSD symptomatology is not actually delayed, but has simply failed to be recognized, detected and consequently diagnosed (Pary et al. 1986) (referred to as late detection or late recognition PTSD).

The prevalence of delayed-onset PTSD has been reported in a variety of trauma populations. Two studies of motor vehicle accidents, for example, reported prevalence rates of delayed PTSD, ranging from 13 % nine months after the accident (Epstein 1993), to 32 % one year after the accident (Mayou et al. 1993). Buckley et al. (1996), in a sample of 158 motor vehicle accident (MVA) victims, reported that 7 % of the sample fitted criteria for delayed-onset PTSD at 1 year. All of these subjects met criteria for sub-syndromal PTSD at initial assessment (3 days after the accident), with the average interval from accident to developing full PTSD being 8.5 months (Buckley et al. 1996). Ehlers et al. (1998) found that only 6.3 % of MVA victims who did not meet the criteria for PTSD at 3 months reported PTSD at 1 year (34 of 549).

Studies with other trauma populations have shown similar prevalence rates. Green et al. (1990), in a longitudinal study of the victims of the Buffalo Creek Dam disaster, reported 11 % of the sample met the criteria for delayed-onset PTSD 12 years following the disaster. McFarlane (1988) reported that 20 % (63 of 315) of the fire fighters exposed to the Ash Wednesday Bushfires in South Australia in 1983 experienced delayed-onset PTSD. Of these 63 individuals, 27 % were symptomatic at 11 months but not at 29 months, and 56 % only became symptomatic by 29 months; only 13 % were symptomatic on both occasions. This indicates that the majority of the delayed-onset cases emerged in the second year after the disaster and that only 48 % of the cases that had emerged at 11 months remained chronic.

Delayed/late-onset PTSD has also been reported in combat veterans. An early study by Solomon (1989) investigated the prevalence of delayed-onset PTSD in a sample of 150 veterans who were treated from 6 months to 5 years following the Lebanon war. They reported 10 % of the sample to have delayed-onset PTSD, with latency periods ranging from several weeks to several years. Watson et al. (1988), in a sample of 63 veterans with PTSD, reported that 49 % of participants reported a delay of at least 6 months before PTSD symptoms first appeared. Other, earlier studies reporting a delayed onset include studies using World War II veterans (Archibald and Tuddenham 1965), Vietnam veterans (Boulanger, 1985), survivors of the Holocaust (Ben Shoshan 1985) and survivors of concentration camps (Chodoff 1963). A number of case studies reporting late-onset PTSD have also been published which are generally characterized by extremely long latency periods of up to 30 years (Van Dyke et al. 1985; Herrman et al. 1994; Pomerantz 1991; Lim 1991). The problem with many of

these earlier studies is that conclusions were made in the absence of longitudinal documentation of the individual's symptoms. It was quite possible that the individual may not recall the post-traumatic symptoms from an earlier period accurately.

Harvey and Bryant (2000) have examined the retrospective recall of symptoms and found that individuals with high levels of symptoms tend to over-report early distress. In contrast, individuals with less severe symptoms reported less early symptoms than they were recorded as having experienced at the time. Such findings would suggest that the memory of symptoms is less accurate than is often presumed in legal and clinical settings. The evidence suggests that retrospective recall of symptoms can lead to both exaggeration and diminution of severity. A prospective study of Gulf War veterans examined rates of PTSD in a sample of 2949 army personnel (Wolfe et al. 1999). A doubling of rates of PTSD was found in the 2-year period post combat, providing support for the diagnostic category of delayed-onset PTSD and the increasing severity of symptoms with time.

To date, delayed/late PTSD is generally accepted as a valid diagnosis in cases where there is a latency period of at least 6 months before the onset of PTSD symptoms (APA 1994). This group of patients presents a challenge to the current models of aetiology of PTSD that focus on the acute stress reaction as the primary determinant of the disorder. In this group of patients, the delayed emergence of symptoms cannot be accounted for by these aetiological models.

Burden of disease

The disabling impact of PTSD has been specifically examined in the NCS (Kessler and Frank 1997). Again, there are very few truly longitudinal studies that have answered this question. Using a retrospective life history method, Kessler et al. (1995) suggested that 60 % of cases would spontaneously remit. The mean time before remission was 72 months. However, the interval was significantly different for different types of trauma. For example, the mean time before remission following assaultative violence was 120 months, in contrast to situations where the subject witnessed somebody being seriously injured or killed, which was 13 months. The time to remission for combat was 85 months (Kessler, personal communication). The NCS indicated that PTSD is often a chronic and disabling condition even in the absence of compensation. These observations led to the conclusion that there is an enduring pattern of chronic morbidity wherein this disorder is long-lasting and seemingly treatment-resistant. Hence, this pattern of chronicity is not restricted to groups such as veterans, who can be rewarded with pensions, but is apparent in a sub-group of individuals with this condition.

In the Australian national epidemiology study (Creamer et al. 2001), the work impairment in terms of days cut back per month was 2.8 (days). The odds ratio for the current unemployment was 3.2, and for the increased use of outpatient health care, 28.2. Such findings led Kessler to conclude that PTSD and major depression represent the two most substantial causes of burden of disease by psychological disorders. Estimates of the associated disability and burden of disease have identified much more substantial effects than were anticipated in the WHO estimates of the burden of disease in 2020. In a study of the costing of PTSD, Marshall et al. (1998) have indicated that Vietnam veterans consume $79.00 per fortnight more medical services if they have a PTSD diagnosis than if they do not. In a study of Third Party motor vehicle accident claims, Chan et al. (2003) identified that the cost of the claim was increased by 50 % if the individual experienced PTSD. In a study of veterans' health utilization, Deykin et al. (2001) indicated that PTSD, alone and in combination with depression, has a direct negative relationship with physical health that, in turn, is associated with more frequent use of primary health care services.

There have been a number of studies that have suggested that the socio-economic status of veterans is compromised (Kulka et al. 1990). In the most carefully controlled examination of this issue, McCarren et al. (1995) used the non-affected co-twin in an examination of the socio-economic effects of PTSD. They found that employment was the one factor that was different in the PTSD twin. No difference was found in the combat discordant twins that could account for this finding, indicating the disadvantage in employment capacity was an effect of PTSD. Hence, the socio-economic effect of PTSD is an area requiring further assessment, but the evidence to date indicates a level of burden of disease similar to that associated with depression (Kessler 2000).

Non-PTSD post-traumatic morbidity

Epidemiological studies of a prospective nature have also identified that PTSD is not the only consequence of traumatic events. While there are significant rates of comorbidity with PTSD (Kessler et al. 1995; McFarlane and Papay 1992), one of the more pressing questions is the role of traumatic events in the aetiology of disorders other than PTSD. A substantial body of research demonstrates that comorbidity in PTSD is the norm rather than the exception (e. g. Kessler et al. 1995; Breslau 1998; Creamer et al. 2001). Few attempts have been made, however, to examine systematically the psychiatric impact of traumatic events in the absence of PTSD. Breslau (1998) found that odds ratios for other disorders in a traumatized sample without PTSD ranged from 1.3 to 1.5 (none statistically significant). She concluded that exposure to trauma *per se*, in the absence of PTSD, does not increase the risk of other disorders.

This conclusion, however, is at odds with several studies reporting increased prevalence of other disorders in the absence of PTSD following traumatic exposure. Shalev et al. (1998), for example, found that 29 % of

those with major depression did not have comorbid PTSD and concluded that major depression and PTSD are independent sequelae of traumatic events. Similarly, Schnyder et al. (2001), in a sample of severely injured accident victims, reported that nearly 20% suffered from depression and/or anxiety independent of PTSD at 12 months post trauma. These morbidity statistics have so far not been taken into account in calculating the burden of disease arising from these events.

Embedded in these results are several possible relationships. First, the disorders may have been triggered directly by the traumatic exposure. Secondly, the disorders may have been present at the time of the traumatic experience. Thirdly, a history of prior psychiatric disorder may increase the probability of subsequently developing another disorder following trauma. Finally, an Axis I or II disorder may increase the probability of experiencing a traumatic event (Breslau et al. 1995).

One study has examined this interaction with existing morbidity and a traumatic event. Two cohorts of the ECA study were subsequently subjected to a disaster that provided a unique opportunity for the prospective longitudinal effects of a traumatic event to be studied. The Times Beach area was discovered to have been built on a dioxin dump and also floods occurred in the region. Following the disaster, the exposed population had apparently greater symptoms of depression, somatization, phobia, generalized anxiety, PTSD and alcohol abuse. However, when the symptoms that existed prior to the disaster were taken into account, the differences were less dramatic, with the differences for only depression and PTSD being significant (Solomon and Canino 1990). In contrast to the symptoms of PTSD where the occurrence of symptoms was a de novo onset, the depressive symptoms were a recurrence of previous symptoms. Many of these symptoms had resolved within a year of the disaster. The Puerto Rico cohort experienced a hurricane that involved loss of life and property and demonstrated similar findings (Solomon and Canino 1990). The sparse literature about the association between traumatic events and other psychiatric disorders could be further developed by contrasting the rates of morbidity in the population who have been exposed to trauma (excluding those who developed PTSD) with those who have no history of trauma.

Thus, the longitudinal effects of trauma are complex and involve the initiation of new symptoms, particularly those of PTSD, but also the emergence of symptoms of depression and anxiety which represent both the onset of new symptoms and the reactivation of prior affective and anxiety disorders. Traumatic events may also modify the longitudinal course of these disorders. The exploration of these issues is important to the full characterization of the burden of PTSD.

Behavioural comorbidities

Addiction and abuse. The complications of PTSD have also been better characterized in epidemiological data sets. Whilst it is recognized that there are multiple determinants of drug and alcohol abuse, it appears that PTSD is the one disorder where there is significant evidence of the self-medication hypothesis. The NCS of Kessler et al. (1995) examined associations with PTSD, and showed that, amongst males, 88% had a comorbid disorder and, in women, 79% had an accompanying disorder. Alcohol abuse was present in 52% of men and 30% of women. These data also suggested that, at least amongst women, substance abuse tended to be the consequence of the disorder rather than its antecedent. This was somewhat different from the ECA study cohort in St. Louis, where Cottler et al. (1992) suggested that, in a significant percentage of people, drug and alcohol abuse preceded the development of PTSD and could, therefore, contribute to the occurrence of traumatic events.

The self-medication model specifically relates to the notion that individuals seek specific substances to alleviate painful effects. This response to the symptoms of abuse depends on the underlying assumption that substances of abuse are not randomly chosen, rather, they are selected by the afflicted individual based on the specific psychopharmacological effects on different internal states of dysphoria (Mueser et al. 1998). Chilcoat and Breslau (1998) concluded that survival analyses support the self-medication hypothesis, but provide little evidence of alternative causal pathways. They found that pre-existing PTSD increased the risk of subsequent drug abuse and dependence. This result is consistent with the findings from other epidemiological cohort studies (Breslau et al. 1997; Kessler et al. 1995; McFarlane 1998).

Jacobsen et al. (2001) examined two possible primary pathways to explain the overlap between PTSD and substance abuse. In the model where PTSD precedes development of substance abuse disorders, the use of substances represents a form of self-medication. Patients report that CNS depressants, such as alcohol, cannabis, opioids and benzodiazepines, acutely improve PTSD symptoms. Consistent with this, patients with PTSD report that the onset and severity of substance abuse paralleled the onset and escalation of PTSD symptoms.

The relationship between trauma exposure, PTSD and substance abuse is, therefore, one that cannot be ignored in any discussion of the aetiology, prevention and policy about the negative effects of drugs and alcohol.

Suicide. The relationship between depression and suicide and attempted suicide is the primary rationale of policies used in an attempt to decrease the costs to society. The role of PTSD has been given relatively little consideration, despite emerging evidence of its significance. Kotler et al. (2001) concluded that individuals with PTSD are at higher risk of suicide on the basis of an extensive review of the literature (Bullman and Kang 1994; Davidson et al. 1991; Ferrada-Noli et al. 1998; Hendin and Hass 1986; Kramer et al. 1994; Prigerson and Slimack 1999). Davidson et al.'s (1991) use of the North Carolina subset of the ECA study best exemplifies the risk. They concluded that of those with a lifetime history

of PTSD, 19.8% had attempted suicide compared to 3.9% with other DSM-III diagnoses and 0.8% of the general population. Thus, after controlling for comorbid depression, those with PTSD were 8.2 times more likely to attempt suicide.

In the Australian National Mental Health and Wellbeing Survey, the relationship between suicidal ideation and major depressive disorder in the last 12 months had an odds ratio of 29.9. The disorder with the next highest odds ratio is PTSD, with an odds ratio of 22.8. These are highly significant associations. The relationship with suicide attempt in the past 12 months and major depressive disorder was illustrated by an odds ratio of 25.2, in contrast to PTSD where the odds ratio is 26.8. Current major depressive disorder had an odds ratio of 7.2 for the lifetime risk of attempted suicide, whereas PTSD had an odds ratio of 14.6. These data suggest PTSD should be considered in assessing the risk factors for suicide and attempted suicide.

Conclusion

The emerging epidemiological literature suggests that traumatic life events increase the risk of a range of psychopathological outcomes including PTSD, depression and suicide for an extended period. Epidemiological outcome studies have made a major contribution to resolving some of the controversies about PTSD because of their inherent capacity to consider the effects of trauma in large populations where financial compensation is not an issue. An atheoretical observational approach that does not presume a hierarchy of diagnosis has also allowed the complex patterns of morbidity to be defined, for instance, with substance abuse.

These findings have several implications. First, the life events literature has traditionally focused on a window of effect of 6 months' duration. The findings from the study of traumatic events suggest a much more prolonged period of influence than has been associated with more day-to-day experiences in community samples. Secondly, the impact of traumatic events has a role in the aetiology of disorders other than PTSD that needs more detailed consideration in determining the burden of disease from such events. Furthermore, the question arises as to whether non-PTSD disorders emerging in this setting have different risk factors and neurobiology from their counterparts with no context of exposure to a traumatic event. While there is a small and emerging literature about the prevalence of PTSD in psychiatric patient populations, the contribution of traumatic events to the patterns of morbidity and treatment outcomes remains to be clarified in a number of disorders where this question has not been systematically analysed, such as schizophrenia, bipolar disorder and personality disorders.

Traumatic events provide a unique opportunity to implement a preventive and public health approach to the management of psychiatric morbidity. The exposure to the event represents a risk factor that can be addressed at population and individual levels, and offers the opportunity for early intervention programmes. The major burden of disease associated with PTSD is largely unrecognized at a policy level and in the design of services that deal with the victims of solitary events. Given the longevity of the effect of traumatic events, there are likely to be significant benefits in assessing the opportunities for implementation.

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