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# Mental health following traumatic injury: Toward a health system model of early psychological intervention

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**Abstract**

In 2005, over 2 million people in the United States of America were hospitalised following non-fatal injuries. The frequency with which severe injury occurs renders it a leading cause of posttraumatic stress disorder and other trauma-related psychopathology. In order to develop a health system model of early psychological intervention for this population, we review the literature that pertains to mental health early intervention. The relevant domains include prevalence of psychopathology following traumatic injury, the course of symptoms, screening, and early intervention strategies. On the basis of available evidence, we propose a health system model of early psychological intervention following traumatic injury. The model involves screening for vulnerability within the hospital setting, follow-up screening for persistent symptoms at one month posttrauma, and early psychological intervention for those who are experiencing clinical impairment. Recommendations are made to facilitate tailoring early intervention psychological therapies to the special needs of the injury population.


**Contents**



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## 1. Introduction

In recent years, much information has been published about early intervention for posttraumatic stress disorder (PTSD) following traumatic events. Issues pertaining to early intervention have been explored in numerous review articles (e.g., Ehlers & Clark, 2003; McNally, Bryant, & Ehlers, 2003; Watson & Shaley, 2005), and books/chapters (e.g., Bisson, 2003; Bryant, 2004; Litz, 2004; Shalev, 2002b; Watson et al., 2003). Furthermore a number of best practice guidelines for early intervention following trauma have been developed (Australian Centre for Posttraumatic Mental Health, 2007; National Collaborating Centre for Mental Health, 2005; U.S. National Institute of Mental Health, 2002). Despite this, there is a paucity of literature that attempts to integrate information about early intervention and present it in a coherent model for health system application.

The aim of this review is to examine information relevant to early psychological intervention in terms of its applicability to a specific trauma population — traumatic injury survivors. This population of trauma survivors warrants a specific focus because traumatic injury is one of the leading causes of posttrauma psychopathology (Creamer, Burgess, & McFarlane, 2001; Kessler, Sonnega, Hughes, & Nelson, 1995). The process of reviewing what is known about early intervention then provides a basis to present and discuss a health system model of service delivery for the identification and early treatment of posttrauma psychopathology following traumatic injury.

The traumatic stress health service model we will present is a three stepped model that involves screening injury survivors to identify those at risk for poor psychological adjustment, reassessment of those who screen as high risk at a later point in time, and early intervention for those who have persistent symptoms at one month post injury. In order to justify the model we will review the prevalence of psychopathology following injury, describe the course of symptoms over time, review screening information, and describe relevant early intervention studies.

As the "signature disorder" for trauma survivors, posttraumatic stress disorder (PTSD) is the main focus of this paper (Breslau, 1998, p. 1). It would be, however, a mistake to focus exclusively on PTSD given the frequency with which other psychopathology develops following traumatic injury (O'Donnell, Creamer, & Pattison, 2004; O'Donnell, Creamer, Pattison, & Atkin, 2004). Although it is more difficult to determine whether other disorders (e.g., depression) are etiologically linked to a traumatic event because their diagnostic criteria do not directly require experience of a traumatic event, the finding that many psychiatric disorders are prevalent in the aftermath of trauma has implications for early posttraumatic intervention strategies.

It is important at this point to address what we mean by "traumatic injury". Most studies that have investigated psychopathology following injury have assessed people with injuries severe enough warrant an admission to an emergency department or hospital. So the literature uses injury severity as a necessary, but not sufficient, condition to warrant classification as "traumatic injury". It is important to note, however, that injury severity is not a good indicator of whether a person regards their injury experience as an emotionally traumatic experience. For example, characteristics about the injury such as injury severity score (ISS: Baker, O'Neil, Haddon, & Long, 1974), length of hospitalisation, or admission to an intensive care unit are not strong predictors of later PTSD (Mayou, Bryant, & Ehlers, 2001; O'Donnell, Creamer, & Pattison, 2004; Schnyder, Moergeli, Klaghofer, & Buddeberg, 2001; Zatzick, Kang et al., 2002). This has led some authors (e.g., Shalev, 2002a) to describe events as "potentially traumatic events". That is, just because someone experiences an injury does not mean that they experience it as emotionally traumatic. There are many characteristics about being injured that will contribute to whether an individual experiences being injured as a traumatic event. These may include the perception of fear, helplessness or horror at the time of the injury event, the experience of invasive and painful hospital procedures, and dealing with the consequences of the injury (such as disability, disfigurement, and pain). Shalev (2002a) provides a useful distinction between the primary stressors (e.g., the motor vehicle accident), and secondary stressors (e.g., dealing with bodily disfigurement), but recognises that both sets of stressors are important contributors to whether an individual perceives their injury experience as traumatic. In this review when we make reference to traumatic injury we are really referring to injury as a potentially emotionally traumatic experience.

The traumatic injury literature is not yet at the point where it can differentiate whether some types of injury are more likely to elicit emotional traumatic responses than others. We do know from other literatures that interpersonal violence has higher rates of PTSD associated with it than non-interpersonal traumatic events (Breslau et al., 1998) so we can assume that traumatic injury as a result of assault may be more likely to lead to poorer psychological adjustment than other events such as motor vehicle accidents. Large epidemiological studies are still required to investigate in depth whether there are potentially key mechanisms inherent to certain physical injuries that increase risk of poor psychological outcomes.

## 2. Prevalence of psychopathology following injury

Studies using consecutive or random hospital admissions report that the prevalence of PTSD following injury ranges from 2% to 30% (Ehlers, Mayou, & Bryant, 1998; O'Donnell, Creamer, & Pattison, 2004; Schnyder et al., 2001; Zatzick, Jurkovich, Gentilello, Wisner, & Rivara, 2002) at 12 month post injury, with the majority of studies falling between 10% and 30%. The considerable variance in prevalence rates has been attributed to methodological factors, such as the use of self-report vs structured clinical interview, as well as cultural factors and hospital factors, such as catchment area and mechanism of injury (O'Donnell, Creamer, Bryant, Schnyder, & Shalev, 2003). Even if we consider a modest prevalence rate of 10%, this would mean that in 2005 over 200,000 injury survivors developed PTSD in the United States of America.

Prevalence of other posttrauma psychopathology is less well known. O'Donnell et al. (2003), in their review of the area, found that depression was a common occurrence following injury. Rates of depression vary considerably between studies, ranging from 6% to 42% post injury (Blanchard et al., 2004; Holbrook, Anderson, Sieber, Browner, & Hoyt, 1998; Koren, Arnon, & Klein, 1999; Mason, Wardrope, Turpin, & Rowlands, 2002; Mayou et al., 2001; Michaels et al., 2000; O'Donnell, Creamer, & Pattison, 2004). The few studies examining comorbid PTSD and depression following injury generally indicate that both disorders frequently co-occur, with between a half to a third of injury survivors with PTSD having a comorbid diagnosis of depression (Blanchard et al., 2004; O'Donnell, Creamer, & Pattison, 2004; Shalev, Freedman et al., 1998). The nature of the relationship between PTSD and depression, and the high prevalence of posttraumatic depression, suggests that it is vital to consider depressive symptomatology in the assessment and treatment of psychological pathology following traumatic injury.

Prevalence of posttrauma anxiety disorders has not been the focus of large, methodologically rigorous studies. As a consequence it is premature to draw conclusions about prevalence rates of anxiety disorders such as panic, agoraphobia, social phobia and generalised anxiety disorder that may develop in the aftermath of trauma. Preliminary studies suggest that rates of posttrauma anxiety disorders may range between 4% and 24% (Blanchard et al., 2004; Koren et al., 1999; Malt, 1988; Mason et al., 2002; Mayou et al., 2001; O'Donnell, Creamer, & Pattison, 2004; Silove et al., 2003). Rates of reported PTSD and comorbid anxiety vary considerably across studies, ranging between 3% and 60% (Blanchard et al., 2004; Koren et al., 1999; Mayou et al., 2001; O'Donnell, Creamer, & Pattison, 2004). The development of travel phobia as a consequence of MVA has received increasing attention with post accident rates reported to range from 4% to 29% (Blanchard et al., 2004; Hamanaka et al., 2006; Mayou, Bryant, & Duthie, 1993). While more prevalence studies investigating the rates of posttrauma anxiety disorders are required, it can be concluded that health service models that aim to treat the consequences of poor psychological adjustment following traumatic injury must include the assessment and intervention of anxiety disorders.

Most of the studies examining substance use in injury populations have been concerned with intoxication as a potential cause of injury, rather than as a posttraumatic outcome. These studies demonstrate that hospitalised injury survivors have high rates of preexisting substance abuse and dependence (Mason, Turpin, Woods, Wardrope, & Rowlands, 2006; Zatzick, Jurkovich et al., 2004). Less is known, however, about the development of these disorders as a consequence of the trauma itself. Some studies show that hazardous drinking declines post injury (Dunn et al., 2002) while others report an increase in substance use posttrauma (Michaels et al., 2000). While large prospective studies are required to explore the nature of posttrauma substance use, the finding that substance use disorders are prevalent within this population has implications for a traumatic stress health service model. It suggests that substance use issues need to be assessed and treated if required.

## 3. Trajectory of traumatic stress symptoms

An understanding of the course of traumatic stress symptoms is an important part of this review because it provides useful information about when early intervention should be targeted. Immediately following traumatic injury, the majority of individuals experience some posttraumatic stress symptoms. These reactions may include shock, anxiety, depression, agitation, and dissociative-like symptoms (Shalev, 2002a). These symptoms begin to look similar to PTSD and depression symptoms after one to two days posttrauma (Shalev, Freedman et al., 1998). While even these mild symptoms are often distressing, the finding that most people experience them is consistent with the suggestion that they may have adaptive qualities (Shalev, Schreiber, & Galai, 1993). These early symptoms may enhance communication, recruit social support, and assist in the reappraisal of the traumatic event and its consequences.

In the majority of injury survivors these early symptoms dissipate quickly so that by the end of the first week posttrauma the majority of people experience only mild traumatic stress symptoms (O'Donnell, Elliott, Lau, & Creamer, 2007). A significant minority of survivors will, however, continue to experience high levels of acute stress symptoms, and this is associated with poor emotional recovery post injury (O'Donnell et al., 2007). Specifically, high levels of acute stress symptom severity has been shown consistently to predict later PTSD and depression (Mellman, David, Bustamante, Fins, & Esposito, 2001; O'Donnell, Creamer, & Pattison, 2004; Schell, Marshall, & Jaycox, 2004; Schnyder et al., 2001). Furthermore, there is evidence that those who develop PTSD not only have high acute stress symptoms, but that these symptoms escalate over time (O'Donnell et al., 2007; Orcutt, Erickson, & Wolfe, 2004). O'Donnell et al. (2007) found that those who developed a 12-month PTSD showed an increase in symptoms of avoidance and arousal during the first three months of post injury, while those who did not develop PTSD generally maintained low levels of morbidity across the entire 12-month period following trauma.

In general, the prevalence of depression and anxiety morbidity tends to decline over the first few months following the traumatic event. For example, Wang, Tsay, and Bond (2005) found that 78% and 72% of injury survivors experienced depression and anxiety symptoms at 1 week posttrauma, while at 6 weeks this had decreased to 63% and 59% respectively. Similarly, Holbrook, Anderson, Sieber, Browner, and Hoyt (1999) reported a decline in depression from 60% at hospital discharge to 31% 6 month post injury. While it does appear that symptoms of depression and anxiety tend to ameliorate during the initial weeks and months following injury for the majority of injury survivors, this is not the case for those who go on to develop depression or anxiety disorders.

The trajectory of recovery also needs to recognize the potential for delayed-onset PTSD. DSM-IV identifies delayed-onset PTSD when the condition develops at least 6 months after the traumatic event. Although delayed onset is reported to be reasonably common, properly controlled studies indicate that this is, in fact, an uncommon condition in the context of traumatic injury. Large-scale studies of civilian trauma have reported delayed-onset PTSD in a very small minority of cases, with rates ranging between 4% and 6% (Bryant & Harvey, 2002; Buckley, Blanchard, & Hickling, 1996; Ehlers, Mayou et al., 1998). There is growing evidence that individuals who are diagnosed with delayed-onset PTSD usually have subsyndromal levels of PTSD at earlier assessments (Bryant & Harvey, 2002; Buckley et al., 1996; Carty, O'Donnell, & Creamer, 2006; Ehlers, Mayou et al., 1998). That is, these studies suggest that delayed-onset PTSD cases fail to meet full criteria for PTSD initially but subsequently satisfy criteria because their condition deteriorates to the point of meeting diagnostic threshold. The implication of this for our traumatic stress health service model is that entry criteria for intervention needs to include those with subsyndromal levels of disorder in order to 'capture' those who may develop more severe symptoms in the future.

In commenting on the poor psychological outcomes following traumatic injury, it is important to note that the majority of injury survivors show remarkable resilience to the trauma of injury. While a discussion about the resilience literature is beyond the scope of this paper, we do recognise that the notion of resilience may be useful to inform some early intervention models.

## 4. Screening for disorder

As the majority of individuals exposed to a traumatic experience recover without persistent emotional problems, it has been argued that the provision of trauma intervention to all trauma survivors is "impractical, inefficient, and arguably unethical" (Gray & Litz, 2005, p 191). The NICE guidelines for the treatment of PTSD (National Collaborating Centre for Mental Health, 2005) recommended that single session debriefing and educational approaches that are routinely applied to all trauma survivors are unlikely to have a "clinically important effect on subsequent PTSD" (p. 142). Rather, an approach is recommended where intervention is specifically targeted at those who develop posttrauma psychopathology. Screening is an essential issue in the identification of those who require intervention. There are two main forms of screening — screening for current disorder and screening for vulnerability for future disorder. We will review each of these forms of screening separately, although it is important to note that most research in the area of screening has not involved injury populations.

There has been much work in the development of screening indexes that identify individuals who have a diagnosis of PTSD. In his review of screens for PTSD, Brewin (2005) identified 13 screens that had (a) 30 or less items; and (b) were validated against structured clinical interviews. He concluded that screens with fewer items, simple scoring methods and simple response scales performed as well as longer, more complex scales. Some of the available scales in this review performed near to their maximum potential. This review found that the Impact of Event Scale (cut off 35)

(IES: Horowitz, Wilner, & Alvarez, 1979) and the Trauma Screening Questionnaire (cut off 6) (TQS: Brewin et al., 2002) performed consistently well and had been validated on a number of independent samples. Importantly, measures in these studies were administered some time after the traumatic event and, therefore, identified symptom profiles that had developed over periods of weeks or months.

Use of screening tools to detect depression has also received considerable attention. Screening measures include existing, well-validated questionnaires (e.g., Hospital Anxiety and Depression Scale, HADS, Zigmond & Snaith, 1983), shortened versions of questionnaires (e.g., Beck Depression Inventory-Primary Care, BDI-PC, Beck, Steer, Ball, Ciervo, & Kabat, 1997), positive responses on multi-dimensional interviews (e.g., Mini International Neuropsychiatric Interview, MINI, Sheehan et al., 1998), and even asking a single question about mood (Mallon & Hetta, 2002). Following their review, Schade, Jones, and Wittlin (1998) concluded that screening instruments were generally superior to clinician judgement and frequently performed as well as more extensive questionnaires. Likewise, Williams, Hitchcock Noel, Cordes, Ramirez, and Pignone (2002) reviewed primary care studies and reported reasonable adequacy for depression screening instruments containing 1–30 items when diagnoses were verified using structured clinical interviews.

Use of assessment tools to screen for anxiety has not been reviewed as extensively and at present there is little research regarding the utility of such instruments in the general population. In a study of psychiatric outpatients, Zimmerman and Mattia (2001) found that the Psychiatric Diagnostic Screening Questionnaire (PDSQ), which assesses common DSM-IV Axis I diagnoses, provided a useful screening tool for a range of anxiety disorders when validated against the SCID. Preliminary evidence has also indicated that screening tools designed to assess a specific anxiety disorder (e.g. social phobia, PTSD, hypochondriasis) are useful (Balon, 2005). Mixed findings have been reported for the utility of the Beck Anxiety Inventory (Beck, Epstein, Brown, & Steer, 1988) and the shortened BAI-PC (Beck et al., 1997), with some studies finding adequate efficiency to screen for disorders such as generalised anxiety, panic disorder and PTSD (Beck et al., 1997; Leyfer, Ruberg, & Woodruff-Borden, 2006; Mori et al., 2003) and others finding less predictive accuracy for this instrument (Krefetz, Steer, Jermyn, & Condoluci, 2004; Manne et al., 2001).

It should be noted that screening instruments for anxiety and depression often lack specificity and may assess the general presence of anxiety or depression without differentiating between the two disorders (Mori et al., 2003; Schade et al., 1998; Stauder & Kovcs, 2003). Accordingly, instruments that assess anxiety disorders and depression, such as the Anxiety and Depression Detector, appear to effectively screen for individual disorders as well as providing an indication of more general distress that suggests the presence of any of the disorders (Means-Christensen, Sherbourne, Roy-Byrne, Craske, & Stein, 2006). Thus, at present, while there is some evidence to suggest that certain measures may provide useful screening tools, further research is required to review the utility of instruments to detect a range of anxiety or depressive disorders across a variety of settings.

## 5. Screening for vulnerability

Another form of screening, which has particular relevance to early intervention models, is that for vulnerability to later psychopathology. This form of screening aims to identify in the acute aftermath of trauma individuals at risk for poor emotional recovery. This is particularly important in the case of PTSD where screening for the disorder usually occurs at least one month after the traumatic event (to take into account the time criterion). Screening for vulnerability allows at risk individuals to be identified earlier.

A number of injury studies have examined the predictive utility of a diagnosis of acute stress disorder (ASD) in identifying later PTSD. The Stanford Acute Stress Reaction Questionnaire (SASRQ: Cardena, Koopman, Classen, Waelde, & Spiegel, 2000) is a 30-item self-report inventory that encompasses each of the ASD symptoms. The Acute Stress Disorder Interview (ASDI: Bryant, Harvey, Dang, & Sackville, 1998) is a structured clinical interview that is based on DSM-IV criteria. The Acute Stress Disorder Scale (Bryant, Moulds, & Guthrie, 2000) is a self-report inventory that is based on the same items described in the ASDI. Each of these measures has been used in a range of prospective studies that have attempted to identify people who are high risk for subsequent PTSD. These studies generally find that, while a high proportion of those with an ASD diagnosis go on to develop PTSD, a large number of individuals who developed PTSD do not have an ASD diagnosis (Creamer, O'Donnell, & Pattison, 2005; Harvey & Bryant, 1999, 2000). This seems to be associated with the low sensitivity of the dissociative symptoms (high false negative rate) (see for discussion, Marshall, Spitzer, & Liebowitz, 1999). Creamer et al. (2005) found that, of the four ASD clusters, the acute arousal and reexperiencing clusters had the highest sensitivity and were the strongest predictors

of both a categorical PTSD diagnosis and dimensional severity of PTSD. This is consistent with findings from other populations (Brewin, Andrews, Rose, & Kirk, 1999).

This raises the question as to how well acute symptoms identify vulnerability relative to other risk factors. Two meta-analyses have identified that a number of factors increase an individual's risk to PTSD (Brewin, Andrews, & Valentine, 2000; Ozer, Best, Lipsey, & Weiss, 2003). Brewin et al. (2000) identified that while some predictors were consistent predictors across populations (such as psychiatric history, childhood abuse and family psychiatric history), many predictors were population specific.

Within injury populations, many different factors have been associated with vulnerability to later PTSD (Ehlers, Clark et al., 1998; Holbrook, Hoyt, Stein, & Sieber, 2001; Michaels et al., 1999; Ursano et al., 1999; Zatzick, Kang et al., 2002). While the severity of acute posttraumatic symptoms are often the strongest indicator of later PTSD vulnerability relative to other risk factors (e.g., Mellman et al., 2001; O'Donnell, Creamer, & Pattison, 2004), it is important to note that the addition of other pretrauma, peritrauma and posttrauma variables generally improve predictive models. This is also the case in predicting posttraumatic depression (O'Donnell, Creamer, & Pattison, 2004).

To date few studies have examined the usefulness of instruments in the identification of vulnerability to later psychopathology. Table 1 presents those studies that have tested the predictive ability of existing instruments in the identification of posttrauma vulnerability. Studies in this table were included according to methodology similar to that used by Brewin (2005) such as having 30 or less items; and being validated against structured clinical interviews. While it is important to note that the majority of studies listed in this table are limited by their small sample size, they do suggest that some tools such as the BDI and BAI have high levels of sensitivity although their specificity is only modest. Predictive screening instruments also appear to have high negative predictive power. That is, they are especially good at identifying those people who will not develop posttraumatic psychopathology. Future research is required to develop predictive screening tools which utilise current symptom levels as well as other known vulnerability factors (e.g., history of prior trauma or psychopathology) to see if this improves our ability to predict vulnerability to later psychopathology.

There have also been attempts to screen for vulnerability by using biological markers that are commonly collected in public health settings. Much research has focused on resting heart rate shortly after traumatic injury. This is based on the proposition that increased resting heart rate may reflect adrenergic increase, which may represent a marker of fear conditioning that leads to chronic PTSD (Orr et al., 2003; Pitman, 1989; Yehuda, McFarlane, & Shalev, 1998). Numerous studies have indicated that elevated heart rate shortly after traumatic injury is linked to subsequent development of PTSD in injury survivors (Bryant & Harvey, 2002; Bryant, Harvey, Guthrie, & Moulds, 2000; Bryant, Marosszeky, Crooks, & Gurka, 2004; Kassam-Adams, Garcia-Espana, Fein, & Winston, 2005; Kuhn, Blanchard, Fuse, Hickling, & Broderick, 2006; Shalev & Freedman, 2005; Shalev, Sahar et al., 1998; Zatzick et al., 2005). It needs to be noted, however, that this pattern has not been observed in other samples (Blanchard, Hickling, Galovski, & Veazey, 2002; Buckley et al., 2004; O'Donnell, Creamer, Elliott, & Bryant, 2007). Furthermore, the accuracy with which heart rate predicts PTSD is only modest and is insufficient to warrant its use as a screen for subsequent PTSD in public health settings (Bryant, 2006).

In summary, we still require further research to develop predictive screening instruments with high specificity and sensitivity. Existing screens, however, are useful in excluding those people who will not have posttraumatic mental health problems. By excluding those who are resilient to psychopathology, it enables us to focus limited resources on those who are more likely to develop problems. This point will be elaborated further when we present our model of early intervention.

## 6. Early psychological intervention

Eight studies to date have examined early psychological intervention in injury survivors. These studies range from efficacy studies to more recent effectiveness trials. The majority of these studies focus exclusively on preventing and treating PTSD specifically, although some do comment on how the PTSD focused treatment affects depression symptomatology. One study also intervenes with alcohol abuse. A summary of each study is presented in Table 2.

The majority of studies use, as their screening process, either a trauma-related psychiatric diagnosis or trauma-related symptomatology above a nominated threshold to identify individuals requiring early intervention. The series of studies conducted by the Bryant group use a diagnosis of ASD for entry criteria into their studies (Bryant, Harvey, Dang, Sackville, 1998; Bryant, Moulds, Guthrie, & Nixon, 2003; Bryant, Sackville, Dang, Moulds, & Guthrie, 1999),

Table 1
A Comparison of Predictive Screening Instruments

| Author | Instrument/ cutoff | No. of items | Sample (size) | Administered vs predicting | Content of screen | Disorder | Sensitivity | Specificity | PPP | NPP | Overall efficiency |
|---|---|---|---|---|---|---|---|---|---|---|---|
| Bryant, Harvey, Dang, and Sackville (1998) | ASDI | 19 | Injury survivors (65) | 2–21 day posttrauma | Acute stress symptoms | ASD | .91 | .93 | .67 | .98 | – |
| Bryant et al. (2000) | ASDS/56 | 19 | Injury survivors (82) | Between 2–24 day posttrauma predict 6–7 month posttrauma | Acute stress symptoms | PTSD | .91 | .93 | .67 | .98 | .93 |
| Shalev, Peri, Canetti, and Schreiber (1996) | IES/19 | 15 | Injury survivors (51) | 1 week predicting 6 month posttrauma | Acute stress symptoms | PTSD | .92 | .34 | – | – | – |
| Shalev, Freedman, Peri, Brandes, and Sahar, (1997) | IES/25 | 15 | Injury survivors (207) | One week, and one month predicting | Posttraumatic stress symptoms | PTSD | .91 | .46 | .26 | .62 | – |
| | SANX/45 | 20 | | four months posttrauma | Anxiety symptoms | | .88 | .43 | .24 | .65 | – |
| | Mississippi/80 | 35 | | | PTSD symptoms | | .81 | .58 | .28 | .46 | – |
| | CAPS/−=40 | 17? | | | PTSD symptoms | | .93 | .80 | .48 | .23 | – |
| Silove et al. (2003) | IES/35 | 15 | Injury survivors (60–66) | Within 2 week posttrauma predicting 18 month posttrauma | Posttraumatic stress symptoms | PTSD, depression, anxiety | .78 | .84 | .78 | .88 | – |
| | BDI/12 | 21 | | | Depressive symptoms | | .86 | .76 | .64 | .91 | – |
| | BAI/13 | 21 | | | Anxiety Symptoms | | .81 | .71 | .59 | .88 | – |
| | GHQ/3 | ? | | | Nonpsychotic psychiatric disorders | | .91 | .32 | .43 | .86 | – |
| | IES+BDI/35+12 | | | | | | .81 | .95 | .89 | .91 | – |
| Ehlers et al. (2003) | PSS/20 | 17 | MVA survivors (967) | During hospital admission and 3 month posttrauma predicting 12 month posttrauma | PTSD symptoms | PTSD | – | .71 | .93 | .90 | – |
| Vaiva et al. (2003) | Either described or were asked about a fright reaction during the event | 1 | MVA survivors (123) | 2–5 day posttrauma predicting 2 month posttrauma | Fright Reaction | PTSD | .93 | .60 | – | – | – |
| Fuglsang, Moergeli, and Schnyder (2004) | ASDS/56 | 19 | MVA survivors (90) | Within 1 month posttrauma predicting 6–8 month posttrauma | ASD | PTSD | .28 | – | – | – | – |
| Walters, Bisson, and Shepherd (2007) | Trauma screening questionnaire | 10 | Assault survivors (562) | 1–3 week predicting 6 month PTSD | PTSD symptoms | PTSD | .85 | .89 | .48 | .98 | .90 |

Table 2
A comparison of methodologies and results for early psychological intervention studies following traumatic injury

| Study | Participants | Treatment and control condition | Active intervention | Exclusion criteria | Assessment | Results |
|---|---|---|---|---|---|---|
| Bryant, Harvey, Dang, Sackville et al. (1998) | –24 Injury survivors | CBT ($n=12$) | –5×1.5 h sessions | –Current suicidal ideation, psychosis or substance abuse, brain injury | –ASDI assessed ASD pretreatment | At end of therapy |
| | –Mean days post injury 10 days (SD 4) | –Supportive counselling ($n=12$) | –CBT: psychoeducation, relaxation, imaginal exposure to traumatic memories, cognitive restructuring, graded exposure to avoided situations | | –CIDI assessed PTSD at posttreatment and 6 months | –1/12 PTSD in CBT group |
| | | | SC: psychoeducation, general problem solving | | –IES, BDI, STAI posttreatment and 6 months | –10/12 PTSD in supportive group had PTSD |
| | | | | | | At 6 months with PTSD<br>–2/12 CBT group<br>–8/12 supportive<br>The CBT had significantly lower scores than SC posttreatment and 6 months on IES-I, IES-A. CBT lower scores on BDI only at 6 months.<br>Clinical treatment effect posttreatment: IES-I, IES-A; BDI (not STAI)<br>6 months: IES-I, IES-A, BDI (not STAI) |
| Bryant et al. (1999) | –45 ASD or subsyndromal ASD criteria following MVA or assault | –Supportive counselling ($n=16$) | –5×1.5 h sessions | –Current suicidal ideation, psychosis or substance abuse, brain injury | –ASDI assessed ASD pretreatment | At end of therapy |
| | –Mean days post injury 10 (SD 3.34) | –PE ($n=14$) | –PE/anxiety management: psychoeducation, anxiety management, prolonged imaginal exposure, cognitive restructuring | | –CAPS assessed PTSD posttreatment and 6 months | –3/15 PTSD in PE/CR group |
| | –11 Drop outs | –PE and anxiety management ($n=15$) | PE — psychoeducation, prolonged imaginal exposure, cognitive restructuring, supportive counselling | | –IES, BDI, STAI 6 months | –2/14 PTSD in PE |
| | | | SC: psychoeducation, general problem solving | | | –9/16 PTSD in supportive group had PTSD |
| | | | | | | At 6 months with PTSD<br>–3/13 PE/CR group<br>–2/13 PE<br>–10/15 supportive<br>IES-I: SC significantly higher than PE condition posttreatment (not follow-up, not PE/anxiety)<br>IES-A: SC significantly higher at 6 months than both treatment conditions. |

| | | | | | | |
|---|---|---|---|---|---|---|
| | | | | | | (not posttreatment)<br>STAI-SC significantly higher at 6 month follow-up than both groups<br>BDI — no difference between groups<br>Clinical treatment effects (2 SD) in both treatment conditions posttreatment in IES-I, IES-A, STAI; At 6 months only treatment effects for IES-A. No effect for depression |
| Bryant et al. (2005) | 69 ASD patients randomly assigned to groups | Treatment conditions — CBT only, CBT and hypnosis (CBT/H) vs Supportive therapy | 6×1.5 h sessions CBT, CBT-hypnosis, Supportive therapy | Psychosis, organic brain syndrome, substance dependence, current suicidal ideation, childhood sexual abuse | ASDI, BDI, BAI, NART (verbal intelligence) CAPS | Posttreatment: |
| | 18 drop outs | | CBT; psychoeducation, supportive counselling, breathing training, imaginal exposure, cognitive restructuring, relapse prevention<br>CBT/hypnosis: same as CBT (without supportive counselling)' 15 min hypnotic induction and 2 min suggestion<br>Sc: psychoeducation and problem solving | | Pre, post, 6 months | CBT and CBT/H significantly lower than SC<br>–CAPS-I:<br>–CAPS-F<br>–IES-I<br>–IES-A:<br>–BAI:<br>–BDI: no difference between groups<br>Follow-up<br>CBT and CBT/H significantly lower than SC<br>–CAPS-I:<br>–IES-I<br>–IES-A:<br>–BDI and BAI: no difference between groups<br>CBT — no difference on CAPS-F<br>Diagnosis: posttreatment<br>46% SC met PTSD criteria<br>13% CBT<br>9% CBT/H<br>Follow-up<br>59% SC<br>21% CBT<br>22%CBT/H |
| Gidron et al. (2001) | 17 consecutive admissions to ED following MVA and with $P>95$ BPM Intervention 2 days posttrauma | Memory structuring intervention vs supportive listening | 2 Sessions of Memory structuring intervention conducted over phone<br>2 Sessions of Supportive listening | >24 h admission, brain injury | PDS at 3 months | MSI less total PTSD symptoms, less intrusion and arousal but not avoidance<br>4/9 Controls had PTSD vs 1/8 in treatment condition |

(continued on next page)

Table 2 (*continued*)

| Study | Participants | Treatment and control condition | Active intervention | Exclusion criteria | Assessment | Results |
|---|---|---|---|---|---|---|
| Ehlers et al. (2003) | Emergency department admissions who met PTSD criteria or score > 20 on PDS entered study. All entered 3 week self monitoring phase. If < 14 on PDS then excluded | Cognitive therapy ($n$ = 28), self help booklet ($n$ = 25), repeated assessment ($n$ = 26) | Commencing 4 months post injury | LOC > 15 min or amnesia for accident, history of psychosis, alcohol/substance abuse, BPD, suicidal | PDS | CT significantly better outcomes on all measures than SH or RA (posttherapy and 3 months). 9 months — better PTSD severity, disability, BDI, BAI |
| | 85 Randomly allocated | | Up to 12 weeks of CT over 3 month period + 3 monthly booster sessions. Initial session 90 min followed by 60 min | | CAPS | Significantly less cases of PTSD in CT group |
| | | | SH-64 page booklet, CBT principals | | BAI | |
| | | | | | BDI<br>Sheehan Disability Scale<br>Assessments at initial assessment, after 3 week self monitoring, preintervention, 3 months post intervention, 9 months, 12 months | |
| Bisson et al. (2004) | Emergency department admissions — contacted at home at 1 week post injury (AIS mean 1) | 116 Randomly allocated into CBT or treatment as usual | 4 × 1 h sessions of CBT conducted between 5 and 10 week post injury | Preexisting psychiatric disorder, major physical disability or illness | IES, HADS PTSD diagnostic scale pre- and post intervention and 13 months | IES scores significantly lower in intervention group relative to control at 13 months (but not 3 months). IES avoidance scores lower at 3 months. No differences in PTSD diagnostic scale scores, no significant differences in the HADS (anxiety or depression) |
| | –Reporting acute psychological distress (PSS–PTSD criteria, | | | | | 30% in both groups had PTSD at 3 months; at 13 months 16% in CBT and 27% in control group had PTSD |

| | | | | | | |
|---|---|---|---|---|---|---|
| | HADS>15, ISS>35) (Screened 201, 152 eligible) | | | | | |
| Zatzick, Roy-Byrne et al. (2004) | Randomised selection of patients admitted to acute injury at trauma service. Screened using PCL, CESD. Selected if PLC>44, CESD>15. (152 screened, 24 not symptomatic) | Random allocation to collaborative care (CC)condition ($n$=59) or usual care (UC) ($n$=61) | CC — case management, psychopharmacological, psychotherapy treatment. | Injury caused by self harm, active psychosis, head injury | PCL | 12/50 CC patients had PTSD at 3 months so all offered therapy or drugs |
| | | | First 6 months – case management – elicited post injury concerns, developed care plan, coordinate links with community | | CIDI alcohol abuse/dependence | Those in CC condition did not increase rates of PTSD over 12 months where as US condition increased rate (6%) |
| | | | –Those with positive alcohol or post injury alcohol abuse received MI intervention (30 min+ad hoc) Sustained PTSD symptoms offered SSRIs in the first 3 months At 3 months, SCID PTSD administered — those with PTSD diagnosis were given their preference for CBT or SSRIs or combined treatment Case management continued for 12 months as needed. | | Assessments at pre, 3, 6, 12 months | Significant decrease in alcohol abuse at 6 and 12 months |
| Shalev et al. (2005) | Consecutive emergency department admissions; screened for ASD or depression Screened using PSS, ASDI, K6 | CT, PE, SSRIs, placebo, or 3 month PE (control) | Details not yet available | Details not yet available | Details not yet available | Details not yet available |

and others use early PTSD diagnosis (Ehlers et al., 2003). Bisson, Shepherd, Joy, Probert, & Newcombe (2004) used levels of acute psychological distress as determined by crossing a set threshold on one of three self-report instruments: the PTSD Diagnostic Scale (PDS: Foa, Riggs, Dancu, & Rothbaum, 1993), the Hospital Anxiety and Depression Scale (HADS: Zigmond & Snaith, 1983) or the Impact of Events Scale (Horowitz et al., 1979). Zatzick, Roy-Byrne et al. (2004) used threshold scores on the PTSD Checklist (Weathers, Litz, Herman, Huska, & Keane, 1993) or the Centre for Epidemiological Studies Depression Scale (CES-D: Radloff, 1977). In the only study not to use a self-report measure of psychological symptomatology, Gidron, Gal, Freedman, Twiser, Lauden, Snir et al. (2001) used a heart-rate threshold level.

The first early intervention efficacy trials with injury survivors (with and without mild traumatic brain injury) were conducted by the Bryant group (Bryant, Harvey, Dang, Sackville, and Basten, 1998; Bryant, Moulds, Guthrie et al., 2003; Bryant, Moulds, Guthrie, & Nixon, 2005; Bryant et al., 1999). These early intervention studies generally utilised a trauma-focused cognitive behavioural therapy (CBT) paradigm. This approach typically includes psychoeducation, anxiety management (e.g., breathing retraining), cognitive therapy, and imaginal and in vivo exposure. These studies provided strong evidence that, in people with ASD, CBT as an early intervention significantly reduces cases of subsequent PTSD relative to supportive counselling. More specifically, CBT appears to decrease intrusion and avoidance levels significantly, with these differences maintained at 4-year follow-up (Bryant, Moulds, & Nixon, 2003). The results are less consistent with regard to general anxiety and depression. It can be concluded from these studies that 5–6 sessions of CBT is useful in preventing PTSD and lowering intrusive and avoidant symptoms in injury survivors who have high initial symptoms consistent with ASD.

Bisson et al. (2004) conducted a study of emergency department admissions with mostly minor injuries. Individuals who met inclusion criteria were contacted by phone at one week post injury and those who were eligible and interested were sent screening questionnaires. Those meeting criteria for acute psychological distress were invited to participate in the study. Participants were randomly allocated to intervention (4 sessions of CBT) or the control condition (standard care — no formal psychosocial intervention). There was a mean reduction in intrusion and avoidance at 13 months but not in general anxiety or depression. There also was a reduction in PTSD status in the intervention (16%) vs control group (27%).

Gidron et al. (2001) conducted a pilot randomized control trial (RCT) ($n=17$) with emergency room admissions (minor injury). Individuals were screened in hospital using resting heart rate (>95 BPM). Those in the intervention condition were contacted by telephone at 24 hours post injury and the memory structuring intervention (MSI) was administered. This intervention aims to shift processing of traumatic memory from uncontrollable somatosensory and affective processes to a more controlled linguistic and cognitive processes. It aims to provide patients with organization, labelling and causality within memory (Gidron et al., 2001). Patients were given either two sessions of MSI or 2 sessions of supportive listening control sessions. Those in the MSI condition reported significantly less intrusive, arousal and total PTSD symptoms at 3 months than controls as assessed by the PDS (Foa, Cashman, Jaycox, & Perry, 1997). Given the novelty of this technique, replication of these findings is required.

Ehlers et al. (2003) conducted a RCT with emergency department admissions. In this study, patients were sent information about PTSD at one month following their admission and those who were interested in seeking help contacted the study team. Those who screened high in PTSD symptoms completed a structured clinical interview and those meeting PTSD diagnosis were invited to participate in the study. Participants in the study completed 3 weeks of self monitoring and those who remained symptomatic were randomly allocated to cognitive therapy (Ehlers, Clark, Hackmann, McManus, & Fennell, 2005), a self help condition, or repeated assessment condition. Participants in the cognitive therapy condition reported significantly less PTSD, depression, anxiety and disability symptoms than those in the other two conditions at follow-up, and at 3 and 9 months.

These efficacy trials, which are derived from the evidence-based paradigm, aimed to maximise internal validity. The rigorous methodology criteria that characterise these trials include random allocation of patients to intervention and control groups, delivery of standardized manualized treatments on a session by session basis, treatment integrity checks, and highly trained therapists. In contrast, effectiveness trials evaluate the impact of treatments delivered in the real world setting. Effectiveness trials, therefore, attempt to approximate usual care treatment conditions by using providers who will deliver interventions in standard treatment settings and by targeting patients with multiple comorbid conditions. To date, there have been two partial effectiveness trials to investigate an early intervention effectiveness model within a traumatic injury population. Zatzick, Roy-Byrne, Russo, Rivara, Droesch, and Wagner (2004) and Zatzick, Jurkovich et al. (2004) tested whether collaborative care (case management, psychopharmacology and trauma-

focused CBT) would be more effective in preventing/treating PTSD relative to usual care. Admissions to a trauma service were screened for high levels of acute stress symptoms (both anxiety and depression symptoms) and those who scored over a threshold were randomly assigned to collaborative care or usual care conditions. Individuals in the collaborative care condition received case management over the first three months post injury before being assessed for PTSD. Those with a PTSD diagnosis were then offered their choice of trauma-focused CBT and/or psychopharmological treatment (medication). At 12 months, collaborative care was shown to significantly prevent the worsening of PTSD symptoms over time relative to usual care. Over the course of the study, the majority of those in the intervention condition received psychopharmocology, with very few participants opting for the trauma-focused CBT intervention.

Shalev, Freedman, Israeli-Shalev, Frenkiel-Fishman, and Adessky (2005) conducted a large trial involving a wide range of injury survivors admitted to an emergency department. Participants were followed up two weeks after trauma and interviewed via telephone. Those who had high symptoms of ASD or depression were invited to attend an assessment session. At the face to face assessment, patients were assessed for ASD and those diagnosed with ASD or partial ASD and who agreed to participate were allocated to early prolonged exposure, early cognitive therapy, early SSRI or delayed PE (four months posttrauma) starting within four weeks of the traumatic event. If participants declined one form of treatment, they were randomly allocated to another of the options. While only preliminary data are currently available for a small percentage of participants, there is early evidence of decreased PTSD severity in those that received treatment compared with the waitlist group. While the authors caution that it is too early to determine the benefits of the treatment programs, they claim that the findings demonstrate the feasibility of implementing a comprehensive outreach program to identify injury survivors who display high levels of acute posttrauma pathology. Subsequent clinical assessment reduces this group to a small, highly symptomatic proportion of the original group for treatment intervention and evaluation.

It is important to note that no early intervention studies to date have examined the effectiveness of group based early interventions. While there are many advantages about group interventions such as cost effectiveness and social support, studies are required to validate this format of early intervention.

## 7. Developing a health service model of early psychological intervention

This review provides evidence that posttraumatic mental health conditions are a significant problem following traumatic injury and that early psychological interventions may have an important role to play. It provides modest evidence that early psychological intervention using a brief trauma-focused CBT approach is effective in the prevention and treatment of PTSD. There is less evidence to suggest that trauma-focused interventions specifically targeting PTSD will have an effect on depression or anxiety symptom levels. Treatments have been trialled in real world settings using effectiveness models with promising early results. There is, however, a need to translate these empirically supported interventions into models that can be applied as part of routine quality healthcare delivery in trauma settings.

We would argue that these models are important not only because they may prevent human suffering but that they would be cost effective. The societal and economic costs of trauma-related psychopathology are huge. Kessler (2000), for example, found that the impairment associated with untreated PTSD was comparable to or greater than that of other seriously impairing mental disorders. He suggests that these impairments have cost implications to the individual (e.g. work absenteeism, stress on social relationships), as well as on the health care system (e.g., increased service use, inappropriate service use). There is early evidence to suggest that early intervention strategies are a cost effective way of dealing with trauma-related psychopathology (National Collaborating Centre for Mental Health, 2005), although more empirical research is necessary to establish how cost effective they may be.

The model we propose, according to the taxonomy outlined by Institute of Medicine (IOM), is a prevention model using an *indicated* intervention. An indicated intervention is one that is targeted to trauma survivors who demonstrate aspects of a disorder but who may be subsyndromal or subclinical in terms of diagnosis (Mrazek & Haggerty, 1994). Our model explicitly identifies and targets individuals who are in the early stages of their disorder with the aim of preventing a chronic course and escalation of symptom severity.

Our model recognises that limited resources are available for mental health interventions in acute care medical settings (Zatzick, Simon, & Wagner, 2006). Stepped care models offer a method of maximising effectiveness of intervention while minimising costs. Stepped care refers to the practice of offering the least expensive and least intrusive intervention first, and then increasing the intensity (and therefore cost) of intervention as is necessary to

achieve a desired therapeutic outcome (Davidson, 2000). We propose that the literature review above supports a three stage model of early intervention that consists of screening traumatic injury survivors to identify those who are vulnerable for poor adjustment, monitoring those at risk, then offering early intervention to those who remain symptomatic after a period of time (see Fig. 1).

The first step in the model involves screening for vulnerability. The question of when to assess trauma survivors for risk of developing trauma-related psychopathology is most likely to be dependent on when they can be easily accessed. For injury survivors, a brief window of opportunity exists within the hospital setting. This may be in the emergency department or, for those who are admitted, on a hospital ward. Discharge planning procedures could incorporate a simple screen administered by care coordinators who would then notify psychological/psychiatric services when a patient records over an established threshold. While we recognise that research into the development of predictive screening instruments is limited, symptom based tools such as the TQS and the BDI could be utilised to identify vulnerability. The total score on these measures can be used to classify respondents as being either at 'low risk' or 'at risk' of developing subsequent psychological sequelae. The process of screening could also play a duel role of educating patients about the mental health following traumatic injury. Although there is evidence to suggest that psychoeducation is not associated with the prevention of PTSD (Ehlers et al., 2003), a discussion about mental health during the screening process may increase awareness of psychological health. This, in turn, may improve help-seeking behaviour and the readiness to engage in mental health interventions if necessary at a later date.

The second step in this model is to follow-up patients classified by the screening process as being 'at risk'. We suggest that this could be achieved by telephone at one month post injury, in an effort to minimise the cost and other resources. At one month after injury, the capacity to predict subsequent functioning is considerably more accurate than at earlier times because many transient stress reactions have abated. During this telephone call, patients are screened for high psychopathology symptoms utilising tools such as the ASDS, IES or TSQ for PTSD, and the HADS for

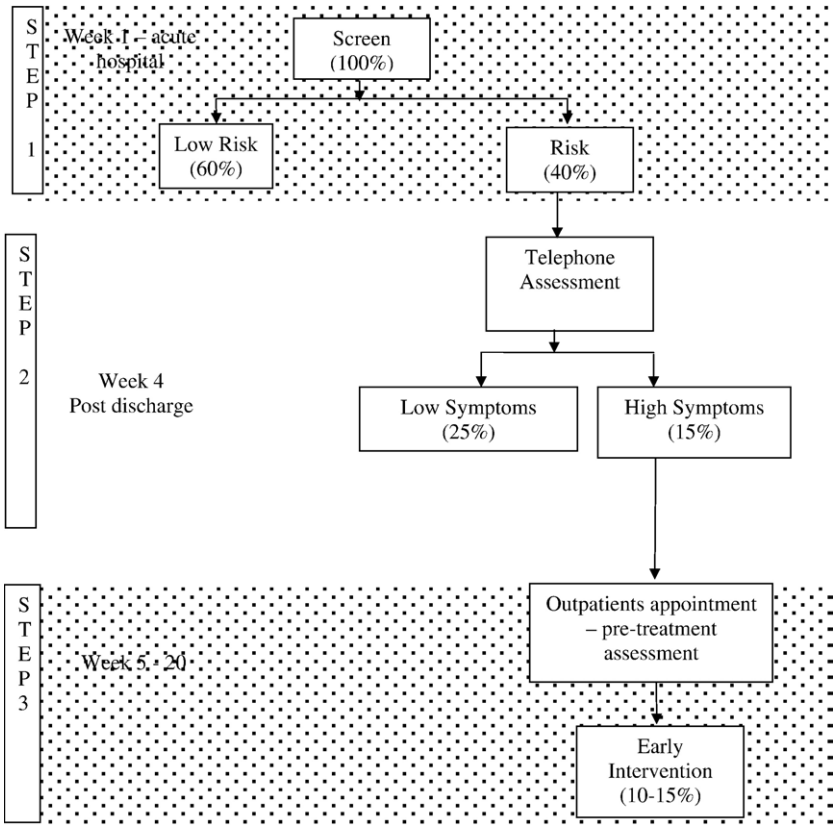


Fig. 1. Model of early psychological intervention service delivery. Note: The numbers in brackets represent the estimated percentage of screened patients moving through each level of the model. They do not take into account injury patients who refuse to be followed up or who refuse intervention. Qualifications of these estimates are required when this model is empirically tested.

depression and anxiety. Those who screen above the recommended cut off points for each instrument are asked if they would like to attend a more in-depth assessment with a qualified mental health practitioner to determine whether therapy would be useful. It is important to note that this screening procedure aims to identify individuals who are experiencing symptoms at a level that causes significant distress or impairment to the individual's social or occupational functioning. That is, we strongly recommend that individuals reporting impairment are offered a more in-depth clinical assessment even if they do not cross the diagnostic cut off point on any of the tools. This increases the probability that individuals with subsyndromal levels of symptoms (especially PTSD) are offered intervention.

The final step in the model is to conduct a face-to-face assessment and to offer treatment to those where it is appropriate. While a discussion of assessment content/processes is beyond the scope of this paper it is important to recognise that there may be an overlap between symptoms associated with physical injury and an individual's emotional responses. We refer readers to an earlier review (O'Donnell et al., 2003) for a more detailed discussion about this point.

Generally, early intervention studies suggest that a CBT approach is useful with this population. A review of the elements of the CBT approach is beyond the scope of this paper. There are a couple of points, however, that are worth noting. First, it needs to be recognised that injury patients who present for psychological treatment are likely to have complex trauma histories (Ramstad, Russo, & Zatzick, 2004), multiple comorbidities (O'Donnell, Creamer, Pattison et al., 2004), and a degree of impairment that may require multi-faceted approaches to treatment of the kind that are not normally addressed within randomized controlled trial (RCT) designs (Zatzick et al., 2006). Furthermore, given these patients have recently been injured, issues of poor physical health increase the complexity of the clinical presentation. This includes pain management problems and adjustment issues such as altered role difficulties, relationship conflict, and coping with other stressors such as financial problems. Therefore, manualized treatments that have demonstrated effectiveness under RCT conditions need to be adapted to meet the heterogeneous presentation of these 'real world' patients. Calhoun, Moras, Pilkonis and Rehm (1998) recognise this point in their discussion about second generation treatment manuals which emphasize strategies more than the strict application of specific techniques. In this sense, therapy manuals are used as strategic tools rather than formulaic prescriptions (Davidson, 2000). Similarly, flexible approaches to treatment manuals may offer a way of individualizing empirically supported therapy to meet the needs of individual patients (e.g., Goebel-Fabbri, Fikkan, & Franko, 2003).

The complex presentation of injury patients may also be associated with a lack of motivation to initiate or complete therapeutic interventions. Zatzick et al. (2006) suggest that, in developing service delivery models, empirically supported psychotherapeutic treatments should be combined with care management strategies. Care management strategies that increase patient engagement and adherence include intensive in-person and telephone-outreach efforts, problem solving barriers to therapy, identification of patient concerns, and motivational interviewing to address poor engagement with therapy.

Finally, the measurement of treatment outcome should not be limited to symptom reduction or loss of a diagnosis. Outcome variables that focus on functional assessment such as improvements in disability, quality of life, pain management, and social and occupational functioning are most important to patients (Newman, 2000). These areas of functioning also have direct relevance to the costs associated with psychopathology (Newman, 2000).

This proposed model has been developed from a strong empirical basis and it provides a clear structure that can be tested within an effectiveness paradigm. It has the advantage of identifying individuals at high risk of poor adjustment following traumatic injury and selectively targeting them for psychological therapy. This approach has the advantage of facilitating access to mental health treatment. This is particularly important for this population because, although the nature of chronic posttrauma psychopathology is disabling (Green et al., 1990), at present the majority of injury survivors are not identified or treated for their posttraumatic psychological problems (Zatzick, 2003).

It important to note that this model has been developed around a specific trauma population — injury survivors. There may be aspects of the model that are relevant for other trauma populations such as disaster survivors or other hospitalised groups such as patients diagnosed with cancer. It is important to note, however, that the generalisability of this model to other trauma populations is unknown, and more appropriate models may arise from a review of the literature specific to these groups.

## 8. Conclusion

In conclusion, PTSD and depression are common consequences of experiencing a traumatic injury. Other anxiety disorders and substance use disorders also occur relatively frequently in this population. Poor mental health has

enduring consequences in terms of quality of life, lower return to work and higher levels of disability. While the majority of individuals will recover from a traumatic injury, a significant minority will experience high acute stress symptoms which will escalate over the following 3 month post injury and will develop into longer-term psychological disorders. A number of screening instruments exist that can identify individuals at risk for posttraumatic psychopathology following injury. Furthermore, several studies show that early psychological interventions may prevent the development or escalation of subsequent PTSD symptoms (and, to some extent, depressive symptoms) following traumatic injury. We have proposed a stepped care model of service delivery that aims to screen, monitor then offer intervention to those who are at risk for mental health problems following traumatic injury. Developed from a strong empirical basis, the model provides a clear structure that can be tested within an effectiveness paradigm. Only when we address the psychological injury that arises as a consequence of physical injury can trauma care services claim to be addressing the total health of injury survivors.

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