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An exploration of family communication style and its impact upon Post Traumatic Stress Disorder

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ABSTRACT

In 1999, Tarrier, Sommerfield and Pilgrim (1999) demonstrated that individuals with PTSD showed a poorer treatment outcome if they lived with relatives who were high in Expressed Emotion (EE). However, no exploration of how this effect was mediated was made at the time.

The current study is an initial exploration into the possible links between Expressed Emotion and PTSD. Possible variables linking the two concepts were identified through an examination of the theoretical models of both EE and PTSD. These variables were then measured in a one off interview session with individuals prior to the onset of treatment. The EE level of their key identified relative was also calculated during the session through a measure which examines the individuals' perception of their relatives' behaviour.

The findings demonstrated an association between the perceived level of EE of the relative and the cognitions about the world and the cognitions regarding self-blame held by the individual with PTSD. As this was only an exploratory study, findings were not specific enough to guide more theorising as to the links between EE and PTSD. However, the findings did suggest useful avenues of further research.

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An exploration of family communication style and its impact upon Post Traumatic Stress Disorder : A review of the literature

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An exploration of family communication style and its impact upon Post Traumatic Stress Disorder: A review of the literature

Introduction

In 1999, Nick Tarrier and colleagues carried out a study investigating the link between Expressed Emotion (EE) and recovery from Post Traumatic Stress Disorder (PTSD) (Tarrier, Sommerfield and Pilgrim, 1999). Their findings demonstrated that individuals who lived with high EE relatives showed a poorer recovery rate than individuals who lived with low EE relatives. This seminal paper is the first to investigate the link between EE, a well-known and much researched measure of family interaction, and PTSD. Although this study is as yet unique, it allows us to tentatively link the current extensive research into EE and PTSD. Two important consequences follow: firstly, linking existing literature pertaining to the impact of EE on psychological disorders and that focused on factors precipitating and maintaining PTSD may provide important clinical insights which may help to guide treatment protocols. Secondly, linking these two bodies of research may give us an indication of where best to direct future research efforts.

This review aims to begin the process of linking these two areas of research. The research into both EE and PTSD is extensive. This review will therefore limit itself to an exploration of the theoretical links between the two areas, incorporating a brief history of the two theoretical concepts, a summary of the most commonly utilised theoretical models

underpinning the concepts and some ideas and suggestions regarding how the two areas may be linked. It will also generate suggestions of possible directions for future research.

The history of Expressed Emotion

The concept of EE originated from a study completed by Brown, Carstairs and Topping, (1958), which investigated the link between the outcome for schizophrenic patients and their social environment. This study showed that individuals with schizophrenia who returned to a family environment after hospitalisation had a higher rate of relapse than individuals who moved to supported or individual housing. This suggested that there was an influence stemming from relationships within the home. Brown, Monck, Carstairs and Wing, (1962) extended this study to investigate the source of this influence. They interviewed the families extensively and measured both verbal and non-verbal interactions on numerous scales. A factor analysis of the data identified five key aspects of interaction which were strongly predictive of outcome. These five aspects were labelled criticism, hostility, emotional over-involvement (EOI), positive comments and warmth. Over the next two years, a structured interview (the Camberwell Family Interview) was developed to measure these five identified aspects of interaction (Brown and Rutter, 1966). Using this measurement system, families were classified into groups according to their scores on the various scales within the measure. Families high in the characteristics demonstrated to show a high correlation with relapse were labelled high in EE.

This concept has since been validated in numerous studies (Jenkins et al., 1986; Leff and Vaughn, 1985; Neuchterlein et al., 1986). These studies have shown that the chance of relapse increases by a factor of 4 when a patient returns to a family meeting the criteria for high EE (Hahlweg et al., 1989). They have also demonstrated that the measure has high internal validity (Kuipers and Bebbington, 1988) and inter-rater reliability (Kuipers, 1979). Cross-cultural validity has also been demonstrated as level of EE has been found to predict relapse in North India (Leff et al., 1987), Mexico (Karno et al., 1987), as well as in America and the United Kingdom (Leff and Vaughn, 1985; Neuchterlein et al, 1986).

Later studies have refined the concept of EE, determining which factors are most predictive of relapse, finding that the five subscales are not equally predictive. The critical comments subscale and, to a lesser degree, the EOI subscale have been found to account for the majority of the predictive power of the concept of EE (Kuipers, 1979; Vaughn and Leff, 1976). Later research has subsequently concentrated predominantly on the levels of criticism and EOI displayed by a family rather than the levels of positive comments, warmth or hostility.

New developments in the use of Expressed Emotion

Research into the concept of EE has been predominantly in the field of schizophrenia. However, the construct has been increasingly applied to other psychological and even physical health problems. Vaughn and Leff (Vaughn and Leff, 1976) were first to investigate EE as a factor in recovery or relapse in disorders other than schizophrenia. Their study

demonstrated that critical comments are equally prevalent in families containing individuals with depression as in families containing an individual with schizophrenia. However, they found that families of depressed individuals rarely displayed EOI. This may well reflect the fact that the majority of depressed patients lived with spouses whereas schizophrenic patients tended to live with parents. Further, Leff and Vaughn found that living with a high EE family increased the probability of relapse in individuals with depression as well as individuals with schizophrenia. This finding has been replicated in several studies (Hooley, Orley and Teasdale, 1986; Hooley and Teasdale, 1989; Okasha et al.,1994).

A similar impact of EE on recovery and relapse has been found in bipolar disorder (Miklowitz, Goldstein, Neuchterlein, Snyder, and Mintz, 1988; Priebe, Wildgrube, and Muller-Orlinghausen, 1989), eating disorders (Blair, Freeman and Cull, 1995; Szmukler, Eisler, Russell and Dare, 1985), alcohol abuse (O'Farrell, Hooley, Fals-Stewart and Cutter, 1998), diabetes (Koenigsberg, Klausner, Pelino, Resnick and Campbell, 1993), epilepsy (Jadresic, 1988, as cited in Weardon, Tarrier, Barrowclough, Zastowny, & Armstrong Rahill, 2000) and obesity management (Fischmann-Havstad and Marston, 1984). EE appears therefore to be a concept not specific to schizophrenia, possibly suggesting that it is representing some aspect of family relationships which has an impact on the psychological well being of the individual. This has led to considerable speculation regarding the possible ways in which the effects of EE may be mediated.

Models of Expressed Emotion

Currently, three main models of EE's effects dominate the literature. Whilst not mutually exclusive, these models explore the effects of EE from different perspectives

Biological models

The first of these models assumes that EE is a form of psychosocial stress. This model originated early in the development of the EE concept and therefore focuses predominantly on the impact of EE in schizophrenia. It suggests that the effects of EE are mediated through increased physiological arousal. Consistent with the diathesis-stress model of schizophrenia, which suggests that a schizophrenic episode is triggered by a combination of biological predisposition and environmental stress, it proposes that being with a relative who is high in EE results in a level of stress which contributes to, if not entirely causes, a relapse and thus the onset of a schizophrenic episode.

This model is supported by Tarrier, Vaughn, Lader, and Leff (1979) who looked at skin conductance levels (an observable measure of physiological arousal) in individuals in the presence of relatives who were high or low in EE. They found that people displayed high levels of arousal when high EE relatives entered the room but not when low EE relatives entered the room. The authors also showed that levels of arousal remained high throughout interviews with high EE relatives, compared to interviews with low EE relatives, where arousal levels showed an initial high peak (although lower than that displayed with the high EE group) followed by a rapid decrease. They suggested that patients whose relatives were

low in EE rapidly habituated to the physiological arousal induced by contact with their relatives, whereas patients whose relatives were high in EE failed to habituate to their initial physiological arousal. This study was replicated by Sturgeon, Kuipers, Berkowitz, Turpin and Leff (1981). Tarrier et al. (1979) concluded that patients with relatives high in EE are more continuously aroused than those whose relatives are low in EE. They suggested that this prolonged physiological stress might account for the greater relapse susceptibility in patients whose families were high in EE. This conclusion was partially supported by the findings of Tarrier and Barrowclough (1987) which also found a higher tonic level of arousal in individuals whose relatives were high in EE.

Valone, Goldstein and Norton (1984) looked at physiological arousal levels in groups other than those with schizophrenia. They found very similar patterns of arousal to those demonstrated by Tarrier et al. (1979). This study suggests that the patterns of arousal seen are not specific to schizophrenia but instead reflect the impact of interacting with high and low EE relatives.

The psychosocial stress theory assumes that something inherent in the presence of the relative induces the high levels of arousal in the individual with schizophrenia. However, it is equally possible that the high physiological arousal is a product of the individual's difficulties which impairs the interaction with their relative. This alternative explanation was neatly challenged by a study carried out by Tarrier and Barrowclough (1987). They found that individuals with schizophrenia who had one relative low in EE and one relative high in

EE showed high physiological arousal in the presence of the high EE relative but not in the presence of the low EE relative. This suggested that physiological arousal was specific to the interaction with a high EE relative rather than specific to the individual.

Despite initial supportive evidence for a biological explanation for the mediation of EE, the psychosocial stress model is challenged by some findings in the literature. Leff, Kuipers, Berkowitz, Eberlein-Fries, and Sturgeon (1982) carried out an educational programme that focused on reducing the EE levels of families. They found that, despite success in decreasing the levels of EE displayed by families, concurrent decreases in levels of arousal in the individual with schizophrenia were not seen. This result is surprising as decreasing levels of EE displayed within a family has been demonstrated to decrease the risk of relapse of the patient living within the family (Falloon, Boyd, McGill, Razani, Moss and Gilderman, 1982). This would suggest that physiological arousal alone cannot determine the effects of EE on relapse in schizophrenia. It is possible that the effects of reducing arousal are delayed and that reductions in levels of EE may eventually lead to reductions in relapse rate. Unfortunately such long-term research has not been carried out.

In summary, the biological explanation of the effects of EE suggests that being with a family member who is high in EE can cause stress that may trigger relapse. No significant differences in the impact of being with a highly critical relative or emotionally overinvolved relative have been found (Tarrier and Barrowclough, 1987), suggesting that it is not the relative's behaviour per se but the resultant stress that it causes that is relevant.

Attributional model

A second model of EE focuses on a cognitive explanation of its ability to influence various disorders. This model of EE focuses on why relatives act as they do rather than specifically how their behaviour leads to a relapse in the patient. The model draws upon attribution theory for its explanation (see Heider, 1958; Kelley, 1967 for a fuller description of attribution theory). These early theorists defined individuals as being

"constantly engaged in a search for causes of events"

(Hayes and Hesketh, 1989)

The basic premise of attribution theory is that we engage in this search to gain understanding of people's behaviour in order to improve our ability to interact with each other and function in the world.

The possible role of attribution theory in explaining how the effect of EE may be mediated was first highlighted by the work of Leff and Vaughn (1985) and has since been proposed by several other authors (Brewin, MacCarthy, Duda and Vaughn, 1991; Hooley, 1986; Joseph, Brewin, Yule and Williams, 1991). These authors all suggest that relatives' displayed emotions, and thus their level of EE, would be related to their attributions about the causes of the patients' illness, behaviour and symptoms. Leff and Vaughn (1985) speculated that attributing symptoms to the patient rather than to the illness was a characteristic of relatives

high in EE. Hooley (1986) clarified this idea by suggesting that relatives hold patients accountable for symptoms that they believe are controllable but blame the *illness* for symptoms over which the patient is not considered to have volitional control. Thus relatives who believe that the patient can control their symptoms are more likely to blame, and thus be critical of, the patient. This may be influenced by the specific symptoms displayed by the patient; symptoms such as withdrawal and social impediments may be seen as being more under the control of the patient than hallucinations and delusions that seem more clearly part of an 'illness'. Consistent with this hypothesis, spouses of patients with more negative symptoms reported significantly lower levels of marital satisfaction than spouses of patients with positive symptoms (Hooley 1986).

Weiner (1986) tried to elaborate on this hypothesis by relating the particular emotion expressed by relatives to the particular symptoms exhibited by the patient. He hypothesised that symptoms believed to be controllable by the patient elicit anger in a relative. This would concur with the idea that perceiving a symptom as controllable elicits hostility and criticism from a relative; the presence of which would lead to the relative being characterised as high in EE (Brewin, 1988).

Although not many studies have explicitly linked relatives' attributions with EE, many have looked at how relatives' attributions impact upon interactions with family members. Butler, Brewin and Forsythe (1986), for example, demonstrated that mothers who perceived their child's enuresis to be uncontrollable by the child were more tolerant than mothers who

believed the child had the ability to control the behaviour. Fincham, Beach and Nelson (1987) found a clear association between marital distress and attributions about the controllability/intentionality of partners' behaviour. These two studies demonstrate a clear link between believing an individual's unwanted behaviour to be under their control and showing anger or hostility towards that individual, supporting the hypothesis of Weiner (1986).

The studies of Brewin et al. (1991), Hooley (1986) and Joseph et al.(199) all suggest a link between high levels of criticism and hostility (the presence of which most commonly results in a rating of high EE being assigned) and attributions of controllability. However, they do not explore other aspects of EE, most notably, EOI. As yet, there is no evidence that believing an individual to be in control of their actions causes a relative to become overprotective or enmeshed. Leff and Vaughn (1985) suggested that EOI may still be explained by attributional theory but felt that *different* relative attributions lead to overinvolved behaviour. They conceptualised EOI as a by-product of relative's feelings of protectiveness and guilt concerning the patient's condition. Weiner's (1986) theory of attributions suggests that we only feel guilt if we believe that negative events are internal to, and controlled by, ourselves. This would suggest that relatives would in some way feel responsible for the patients' symptoms. An alternative explanation is that relatives perceive the patient as experiencing an illness that is external to and uncontrollable by them, causing them to feel pity for the patient and become overprotective (Weiner, 1986). However, this explanation does not clarify what differentiates a low EE relative and an emotionally

overinvolved relative. Interestingly, Brewin et al. (1991) found that emotionally overinvolved relatives held the same attributions as low EE relatives. He suggested that EOI may not be attributionally mediated but instead might reflect difficulties arising from early attachment problems. This argument weakens the attributional explanation for the concept of EE and tends to strengthen arguments of those who see the concept of EE as reflecting several different phenomenon.

The attributional model paints a simplistic view of why relatives express negative emotions towards an individual with schizophrenia or indeed any other psychiatric disorder. Many theorists believe that the interaction between attributions and behaviour may be influenced by numerous factors related to both the relative and the patient. For example, Butler et al. (1986) hypothesised that the older the patient, the more likely the relative is to attribute the illness to factors controllable by the patient and thus the more likely they are to be critical to the individual. This suggests that EE does not simply reflect a characteristic of the relative but rather reflects a characteristic of the interaction between relative and patient that can change with circumstances and time. Brewin et al. (1991) suggest that situational factors, such as the amount of support and information available to the relative, may also affect their attributions.

In summary, the attributional approach to EE seems to offer a possible, if not entirely elaborated, explanation of why relatives express criticism and hostility. Further research is needed to investigate claims that different aspects of EE may be mediated by different emotions (Brewin et al., 1991). Research is also necessary regarding the difficulties the

theory has in explaining emotional over involvement, which may raise questions about the way we conceptualise EE itself.

Social Control

The final model developed to explain the concept of EE and its effects was proposed by Greenley (1986). Greenley argued that the particular characteristics measured by scales of EE, and therefore the concept of EE as a whole, could equally be explained by another, pre-existing concept; that of social control. Greenley argued that all the phenomena characteristic of EE can be viewed as attempts by the relatives to place controls on the behaviours of the patient. Criticism can be seen as an attempt to shape an individual's behaviour through the use of negative reinforcement. EOI can equally be seen as a way of trying to alter an individual's behaviour through treating them in a child-like, helpless way, which is likely to reduce their self-efficacy and thus alter their behaviour. Greenley argued that both EOI and criticism have face value as methods of social control. The behaviours characteristic of high EE would be defined as high intensity interpersonal social control (HIISC) according to social control theory. Other behaviours are also characteristic of HIISC, but to date, research has not focused on whether these behaviours are also displayed in high expressed emotion families.

High intensity interpersonal social control is explained as a means by which families attempt to cope with a difficult situation. The social control model would therefore explain the behaviours demonstrated in a high EE family as an attempt to cope with a stressful aspect of

the environment (Coelho, Hamburg and Adams, 1974). One could predict therefore, that families demonstrating poorer coping skills and greater levels of anxiety and fear to demonstrate higher levels of HIISC and EE. Some evidence for this has been demonstrated (Greenley, 1986), however, research in this area is limited.

As such, the conceptualisation of EE as social control has little to tell us about relatives' behaviour above and beyond that already explained by attributional approaches. However, this conceptualisation does have several advantages. Firstly, it would allow researchers to access the abundant literature available on social control theory, possibly offering insights into the behaviours demonstrated by relatives. Secondly, social control theory suggests that relatives high in EE are not temperamentally determined to act in a particular way; they simply lack the coping skill to deal with the situation. This would suggest that approaches that emphasise the development of coping skills would benefit families and possibly reduce the levels of EE displayed. Some evidence for the benefits of improving coping strategies has been shown (Anderson, Hogarty and Reiss, 1981). More research into this area is necessary before any firm conclusions can be drawn regarding the usefulness of this approach.

Criticisms of the concept of Expressed Emotion

Probably the strongest criticism of the EE concept is its atheoretical origin. As described above, attempts have been made to understand possible theoretical underpinnings of the model. However, creating a concept and then attempting to create a theory to fit can have

serious consequences. The first of these consequences is that one cannot be sure that the concept of EE even exists in the form described. The creation of the concept was limited by the measures initially presented to families. It is entirely possible that several other factors are important in the relationship between family interaction and recovery from psychological disorders. However, the concept is limited to those factors measured and thus the concept is biased by the thinking and the approach of the initial researchers. This criticism is clearly highlighted by the argument regarding social control presented above (Greenley, 1986).

The difficulties in creating a clear concept of EE without theoretical guidance are further demonstrated by changes in the use of the EE concept over the years. Originally, EE was defined as a combination of five factors. However, later research has demonstrated that the scales of warmth and positive remarks have little predictive value above and beyond that of criticism and EOI (Kuipers, 1979; Vaughn and Leff, 1976). This has led to a modification in the way in which EE is measured; many contemporary studies only utilise the subscales of criticism and EOI. However, one could argue that the removal of the other subscales invalidates the concept as a whole. In addition, hostility has rarely been identified in the absence of criticism. The subscales of criticism and hostility are often conceptually merged in contemporary studies, resulting in a further move away from the original conceptualisation of EE. This modification of the concept has lead to poor research standards with different studies utilising the same concept label for a very different combination of factors. This could potentially invalidate much of our ability to compare across studies.

A further criticism of the creation of a unified concept of EE has arisen from attempts to generate a theoretical understanding of the model. All of the models discussed above propose a similar, yet subtly different understanding of the way in which the effects of EOI and criticism/ hostility are mediated. If a differing theoretical understanding underpins the two subscales, can one really argue that they are different aspects of the same concept? Indeed, attribution theory has been unable to offer a clear explanation for EOI (Brewin et al., 1999). They may simply represent two entirely separate aspects of family interaction.

Some researchers have argued that measures of EE represents only a snapshot in time and does not give a true picture of interactions within the family. This criticism has been addressed through studies that have investigated how relatives' behaviour during an interview such as the Camberwell Family Interview (CFI) correlates with their everyday interactions. Several studies have demonstrated a positive correlation between the number of criticisms made during the CFI and the number of criticisms made to the patient whilst observed during a visit in the inpatient unit (Brown and Rutter, 1966; Rutter and Brown, 1966; Strachan, Leff, Goldstein, Doane, and Burrt, 1986). Studies have also demonstrated a negative correlation between the number of critical comments made during the CFI and the proportion of time spent listening to the individual with schizophrenia (Kuipers, Sturgeon, Berkowitz, and Leff, 1983) and, finally, between the number of critical comments made during the CFI and the predictability of the home environment (MacCarthy, Helmsley, Schrank-Fernandez, Kuipers and Katz, 1986). This suggests that the snapshot given by the CFI is representative of relative behaviour, thus negating this criticism.

The lack of theoretical underpinning of the concept has led to an arbitrary system of scoring the concept. EE is measured categorically, implying that there is a qualitative difference between the two subgroups, rather than a quantitative difference. Yet, there is no clear evidence to support this implication. Evidence demonstrating the location of the cut off point at which one shifts from low to high EE is also lacking. Original studies of EE utilised different criteria for the boundary between high and low EE to current studies. For example, Brown et al. (1962) defined a family expressing more than 6 critical comments as being high in EE whereas Hooley et al. (1986) utilised 2 critical comments as a cut off point. This leads to difficulties in comparisons across studies and a weakening of the concept as a whole. Some researchers have actually argued that the measure should be scored on a continuum and not categorised at all (Vaughn, Snyder, Jones, Freeman, and Falloon, 1984). Without a clear theoretical justification for the cut off point, criticism of this issue seems well founded.

A further criticism levelled at the EE research as a whole is that of causality. The EE literature presumes that the behaviour of the relative impacts upon the patient, leading to higher levels of relapse and poorer recovery. Numerous researchers have argued that the relationship may in fact be reversed or, at the least, reciprocal. Research looking at the burden of care placed upon relatives may well support this contention as it clearly demonstrates that caring for a relative with schizophrenia can impact upon the family in numerous ways (Gibbons, Horn, Powell, and Gibbons, 1984), one of which may be altering their behaviour towards their relative. This criticism is somewhat counteracted by studies

which demonstrate that treatment aimed at reducing the level of EE displayed by the relative can reduce the relapse rate within the patient group (Falloon et al., 1982). However, the relationship may be reciprocal. Changes may be seen because relatives alter their response to previously evocative behaviour after attending a training course. Longitudinal research is necessary to explore this issue.

Finally, the concept of EE itself has been criticised for its impact upon relatives and societal perceptions regarding the family role in psychological illness. Families often feel alienated (Hatfield, 1983; Spaniol, Jung, Zipple, and Fitzgerald, 1984) and the idea that professionals believe that they may be responsible for some aspect of their relative's illness further enhances this alienation. The concept of EE fits easily with a blame culture and does not take into account the subtleties of interactions within the family (Hatfield, Spaniol and Zipple, 1987). The idea of high and low EE fits well with the idea of good and bad families, which can lead to blame.

Numerous criticisms, many of which are valid, have been levelled at the EE concept, yet the concept continues to exist. Despite its failings, EE research has clearly demonstrated that some aspects of interaction influence an individual's recovery and relapse. If we discard the EE concept because of its failings, we also discard its useful traits. Currently, EE is still the most clearly defined measure of family interaction utilised within the psychological field. It gives us a means, albeit an imperfect means, of measuring interactions and thus a way of studying the relationship between support and psychological illness. Research developments

into the concept of EE or into other forms of studying family interaction are essential. Until we develop more precise, well-researched measures of family interaction, EE will still have its place in the literature. It is important, however, to recognise the limitations of any research utilising the concept and to interpret any findings accordingly.

Despite its limitations, this review will continue to utilise the concept of EE. The brief study of the EE literature has given us some insight into how its effects may be mediated. However, to fully understand how EE may impact upon post traumatic stress disorder specifically, one also needs to understand the current views on how PTSD is precipitated and mediated. For this understanding, we now turn to a brief overview of current models of PTSD.

Post Traumatic Stress Disorder: A brief introduction

Emotional difficulties in managing the aftermath of traumatic events have been described in the literature for over 100 years (Vaughn and Tarrier, 1992). However, it was not until after both World Wars and, later, the Vietnam War that a real interest in the effects of exposure to trauma was developed. From work exploring the experiences of Vietnam veterans and later work with survivors of other traumatic experiences, the concept of Post Traumatic Stress Disorder was developed. This disorder was characterised by three groups of symptoms: re-experiencing (such as nightmares, flashbacks, and intrusive thoughts), avoidance (such as avoidance of stimuli associated with the trauma and social withdrawal) and increased arousal

(such as sleep disturbance, impaired concentration and irritability) (American Psychiatric Association, 1980).

Estimates of levels of PTSD within the population have varied from prevalence rates of 1% (Davidson, Hughes, Blazer and George, 199; Helzer, Robins, and McEvoy, 1987) up to prevalence rates of 12.3% for women (Resnick, Kilpatrick, Dansky, Saunders, and Best, 1993) and 6% for men (Breslau, Davis, Andreski and Peterson, 1991). Levels of PTSD in specific populations have been shown to be even higher; in one study, 70% of Southeast Asian refugees were diagnosed with PTSD (Kinzie, Boehnien, and Leung, 1990). However, prevalence research demonstrates that not everyone exposed to a trauma develops PTSD. This finding has led to a great deal of speculation about factors which predispose an individual to the development of PTSD and which maintain symptomatology once established.

Models of Post Traumatic Stress Disorder

Biological models

Two approaches to exploring the development and maintenance of PTSD have dominated the literature; a biological approach and a cognitive approach. Other approaches, such as a psychodynamic approach, have been posited but they are less well researched and will not be discussed here. The first of these approaches comes from the work of psychophysiologists

who have explored the biological and physiological changes that occur in the body after trauma.

Early researchers noticed that physiological arousal occurred in response to very small stimuli following a trauma (Kardiner,1941). Later research has investigated this state of heightened arousal and demonstrated that PTSD is characterised by a qualitatively different state of arousal to a simple stress response (Van der Kolk, McFarlane and Weisaeth, 1996). Further, this unique state of arousal is not simply a product of the physiological state experienced during the trauma becoming conditioned to trauma related stimuli. Many individuals do not develop PTSD immediately after the trauma but go on to develop symptoms after being exposed to a reminder to the trauma, such as an anniversary, ruling out the possibility of a simple conditioning process. Researchers suggest that the presence of this unique state of arousal results in extreme prolonged stress on the body which triggers long lasting physiological changes and the use of compensation mechanisms (Van der Kolk et al., 1996).

This state of arousal is one of the characteristic symptoms of PTSD. However, it may also be partially responsible for the presence of a second type of PTSD symptoms; those labelled as intrusive. Rainey and colleagues (Raney et al., 1987) discovered that stimulating the autonomic nervous system can elicit visual images and an affective state commonly associated with flashbacks. In addition, Van der Kolk (1994) demonstrated that 20% of individuals with PTSD experience a flashback when presented with stimuli normally used to

elicit an acoustic startle response. This suggests that an aroused autonomic system can precipitate reliving symptoms in a subsection of individuals with PTSD. However, not all individuals with PTSD demonstrate this response so this cannot explain the full range of symptoms experienced.

Emotions are important indicators of changes in the environment and in the self and thus the presence of an emotion results in heightened attention to the environment (Krystal, 1978). Normally we respond to an emotion by attending to the situation and then adapting in one of two ways; either we change our expectations to fit with what is actually happening or we change the situation to fit with our expectations (Horowitz, 1986). For individuals with PTSD, emotions do not serve as an accurate warning signal for changes in the environment; chronic hyperarousal results in the presence of emotions in the absence of changes in the environment. Emotions lose their psychological purpose and become something to fear in their own right. This often leads to increased attempts on the part of the individual to avoid emotions through withdrawing behaviourally, avoiding triggers, and numbing emotions (Litz and Keane, 1989), and cognitively, through distraction and dissociation. Heightened arousal may therefore explain the individual's need to engage in the withdrawal and avoidance behaviours commonly described in PTSD.

The question of why trauma results in hyperarousal has been addressed. One suggestion put forward by Kolb (1987) is that excessive stimulation of the central nervous system at the time of the trauma may have a permanent effect on neurones in the brain inhibiting their

ability to habituate and thus to learn and to discriminate between stimuli (for a full review of how these changes occur, see Van der Kolk et al., 1996). This proposal is supported by numerous studies, which demonstrate that individuals with PTSD have an abnormal startle reflex. Unlike those without PTSD, people with PTSD fail to habituate to stimuli that produce an acoustic startle response (Ornitz and Pynoos, 1989; Shalev, Orr, Peri, Schreiber, and Pitman, 1992). Failure to habituate to stimuli results in an inability to evaluate sensory stimuli and respond with an appropriate level of physiological arousal (Shalev and Rogel-Fuchs, 1993). This would result in an inability to differentiate between stimuli and thus increased arousal at fear-irrelevant stimuli (McFarlane, Weber and Clark, 1993) which may explain the constant levels of hyperarousal seen in PTSD. As well as producing constant hyperarousal, an inability to discriminate between stimuli may result in an inability to attend to important everyday events, resulting in the kind of withdrawal commonly seen in PTSD. However, this hypothesis is challenged by studies that show that even when an individual with PTSD shows recovery and is no longer symptomatic, they still show an inability to habituate to startle stimuli (Fisler and Van Der Kolk, 1995 as cited in Van der Kolk et al., 1996).

The biological model of PTSD can go someway towards explaining why an individual develops symptoms following a traumatic experience but several questions remain unanswered. Firstly, PTSD purportedly results from neuronal changes occurring at the time of the trauma, yet some individuals fail to develop PTSD in response to a trauma. Secondly, some individuals continue to show an inability to habituate following reduction of symptoms.

Symptoms are supposedly a result of difficulties in habituation but their resolution without a concurrent resolution in the individuals' ability to habituate to stimuli remains unexplained. Finally, changes occurring at the time of the trauma are considered permanent, however many individuals initially develop PTSD like symptoms that spontaneously remit and do not go on to become full-blown PTSD.

Other models of PTSD have attempted to address these unanswered questions, taking a cognitive-behavioural approach to understanding the problem. Biological and cognitive behavioural models are by no means mutually exclusive and they may be viewed as considering the problem from different angles thus together giving a more comprehensive explanation of the development and maintenance of PTSD

The levels of representation model

Brewin, Dalgelish and Joseph (1996) proposed a model of PTSD that utilised biological evidence to support its hypotheses but was based on cognitive concepts. They proposed that trauma-related information is represented on two levels; situationally accessible memories (SAMs) and verbally accessible memories (VAMs). The term SAM refers to representations which are only minimally processed, resulting in memories which are perceptual, affect laden and usually not consciously accessible. The affects stored within the SAM usually represent the negative affects experienced during the trauma. However, additional affects (such as shame, guilt) can become associated with the SAM post traumatically (Grey, Holmes and Brewin, 2000). The minimal level of processing results in a lack of context stored along with

the memory, giving the SAM a very 'here and now' quality. These context free, affect laden memories elicited by activation of a SAM are usually described as flashbacks (Grey, Holmes and Brewin, 2000). SAMs are easily conditioned to a wide range of cues which means they are regularly and unpredictably activated, leaving the individual with a sense of being out of control.

The second level of representation described by Brewin et al. (1996), VAMs, are a form of autobiographical memory. VAMs are fully and consciously processed and stored alongside other, non-traumatic memories in the long-term memory, therefore incorporating contextual information. They are also regularly verbally accessed and re-examined, resulting in modification and reappraisal. However, the amount of information stored as a VAM during the traumatic experience may be minimal as exposure to trauma is liable to interfere with the high level of attentional focus required to form a VAM (Van der Kolk and Fisler, 1995).

Brewin (2001) proposed that treatment should entail integration of the SAM with contextual information and other relevant information. This can be done through activating the SAM during exposure and encouraging processing of information, thus creating a VAM. This process can be hampered if the individual's prior beliefs contradict the contents of the SAM. In this case, treatment must focus on helping the individual to develop new beliefs based on both information prior to the trauma and information gained during the trauma.

This model offers an explanation for the occurrence of flashbacks and gives a good explanation of the processes needed to resolve the traumatic experience and subsequent response. However, this model still has some limitations. It does not fully explain the impact of appraisal on the maintenance of the traumatic memory and it does not fully discuss the role of avoidance behaviour. One could surmise from the model that avoidance behaviours might prevent accessing of the SAM, thus preventing processing and creation of a VAM. However, the SAM *is* accessed (hence the flashbacks), it is simply not processed when accessed.

Other, more cognitively orientated models of PTSD have placed a stronger emphasis on the role of avoidance behaviours and may resolve those questions left unanswered by the Brewin et al. (1996) model. Probably the most comprehensive model currently available is the model proposed by Ehlers and Clark (2000). This model draws upon many earlier biological and cognitive models, including the earlier work of Brewin (Brewin et al., 1996). Probably the other main influence on this model was the work of Foa and Kozak (1986); the fear representation model. To fully understand the model of Ehlers and Clark (2000), we must first look at the fear representation model of Foa and Kozak (1986).

Fear representation model

Foa and Kozak (1986)'s model of fear and its modification is not specific to PTSD. Their model aims to explain how all fears are developed and maintained, particularly irrational or pathological fears. The model is based on the work of Lang (1977, 1979) who conceptualised

fear in terms of propositional representations. He suggested that fear is stored in propositions (networks of information). These networks include information regarding the feared stimulus situation, potential responses, and information regarding potential consequences of the event. The author conceptualised a proposition as a "programme for escape" (Foa and Kozak, 1986, p 21) which gave survival information to individuals to aid them in dealing with potentially threatening situations. Foa and Kozak (1986) stated that, for these propositional networks to be useful in aiding escape, the network must incorporate interpretative information which enables the individual to not only recognise stimulus situations, but also to perceive them as dangerous. They therefore emphasised the importance of interpretation in the knowledge stored within the fear network.

Foa and Kozak (1986) developed the work of Lang (1977, 1979), focussing on the differences between normal fear networks and pathological fear networks that underpin anxiety disorders. Foa and Kozak (1986) proposed that pathological fear networks include excessive response elements and some kind of resistance to modification. This resistance to modification may partially be a product of the rigid structural coherence of the propositional representation (Lang, 1977), but may also indicate the presence of impairments in the mechanisms for the processing of fear-relevant information (Foa and Kozak, 1986).

Foa and Kozak (1986) propose that two conditions are required for the modification of fear representations and specifically, the reduction of fear. Firstly, the fear network must be activated through the presentation of fear relevant material that correlates with that stored

within the network. The second requirement for change is the concurrent presentation of information incongruent with some aspects of the fear network. Therefore a situation must be close enough to the feared situation to evoke its memory but different enough to present modifying information. If information is not sufficiently similar to the network, it will not be activated and if it is not sufficiently different, reinforcement of the original fear network will occur.

The model therefore suggests that a fear memory is activated when a fearful individual is presented with fear information that matches some of the information structure in memory (Lang, 1977). It also proposes that strong fears may be characterised by strongly coherent structures that can be evoked with minimally matching information. As exceedingly strong fears are common in PTSD, it is likely that minimally matching information, such as associated cues or reminders of the event, may be able to trigger recollection of the memory and thus fear and flashbacks.

Foa and Kozak (1986) stated that fear incongruent information is necessary for modification of the fear structure. Studies have demonstrated that in order to promote integration of fear incongruent information, the fear has to be evoked and then habituated to. Habituation within stimulus presentation can be seen as fear incongruent information as it is an example of the presence of the stimulus in the absence of danger. For permanent modification of the fear network to occur, habituation to contradictory evidence needs to occur on numerous occasions (Grayson, Foa and Steketee, 1982). This approach is the basis of exposure

treatment where the individual is exposed to the stimulus until they have habituated on repeated occasions.

As cues to activation of fear networks are prevalent and as PTSD memories are easily evoked, it may appear surprising that this habituation does not occur naturally. However, several factors may prevent habituation. Firstly, physiological changes resulting from the trauma my prevent habituation occurring at a normal rate thus resulting in prolonged arousal in response to activation of the fear network (see biological theory for a full discussion). Secondly, avoidance may prevent exposure to the stimulus for a sufficient length of time to allow habituation to occur, or may prevent the individual attending to the fear, resulting in a failure to attend to the stimulus even if not behaviourally avoided. Therefore this model gives a clear indicator of why pathological fear may be developed in PTSD and why it can be maintained in the absence of ongoing threats.

Cognitive model of Post Traumatic Stress Disorder

The final model of PTSD to be discussed here is the cognitive model of PTSD proposed by Ehlers and Clark (2000). Although a relatively new model, it incorporates many of the ideas presented by Brewin et al. (1996) and Foa and Kozak (1986). The basic premise of the model is that an individual experiences PTSD like symptoms if they process the event and its sequelae in such a way that they perceive an ongoing and current threat. It is suggested that an individual with PTSD does not perceive the trauma as a time limited event. Instead they

appraise the trauma in such a way that it appears to present an ongoing threat. This sense of ongoing threat may arise from beliefs about the self (e.g. I am a poor coper) or beliefs about the world (e.g. no one can be trusted) which result from the trauma. Ehlers and Clark (2000) suggest that appraisals of the traumatic event can present a sense of ongoing threat in one of two ways: through overgeneralising the risks to self following the event, or through viewing the way one acted during the trauma as an indicator of a permanent, negative characteristic of the self which was previously undetected (e.g. an individual becoming aroused during a rape may go on to believe that this indicates they have perverse sexual desires). Appraisals of the sequelae of the traumatic event or consequences of the event (such as scarring) may also lead to a sense of ongoing threat.

Ehlers and Clark (2000) suggest that much of this sense of ongoing threat and many of the sequelae of the trauma come from the unique nature of the way in which trauma memories are stored. They describe trauma memories as being sensory, fragmentary, high in emotional content, triggered by numerous (even neutral) stimuli and not easily subject to modification with time. They state that trauma memories retain these unique characteristics because they are not subject to emotional processing with time (Rachman, 1980). This quite broad description fits with the models of descriptions of trauma memory given by both Brewin and colleagues (1996) and Foa and Kozak (1986). However, unlike the above models, Ehlers and Clark (2000) do not elaborate on how the memories are stored, instead focusing on ways in which they are maintained.

To link the two aspects of the model (the appraisal and that the nature of trauma memory), Ehlers and Clark (2000) suggest that there is a reciprocal relationship between the trauma memory and the appraisal style. They suggest that when the individual remembers the trauma, their recall is biased by their appraisals such that only information consistent with the appraisal is recollected. This results in contradictory information being ignored, thus preserving the original trauma memory. In addition, they suggest that sequelae produced by the nature of the trauma memory can be interpreted as threatening. For example, the fragmented nature of the memory may be appraised as indicating a serious problem with memory or the mind, thus suggesting a current threat to the individual. They also suggest that the nature of the trauma memory may reinforce negative self-beliefs resulting from the trauma. For example, the confused ordering of the memory may lead to a mistaken perception that the individual's actions triggered the traumatic event thus resulting in self-blame. Finally, it is possible that the way in which the memory is *encoded* may influence appraisals regarding the trauma at a later date. Conway (1997a,b) proposed that suggestions and beliefs are encoded with a default true value. Thus, if a thought occurs it will be encoded as true unless evidence to the contrary is present. As encoding of context and thus evidence is poor during the trauma, negative beliefs may be encoded as true making negative appraisals post-trauma more likely.

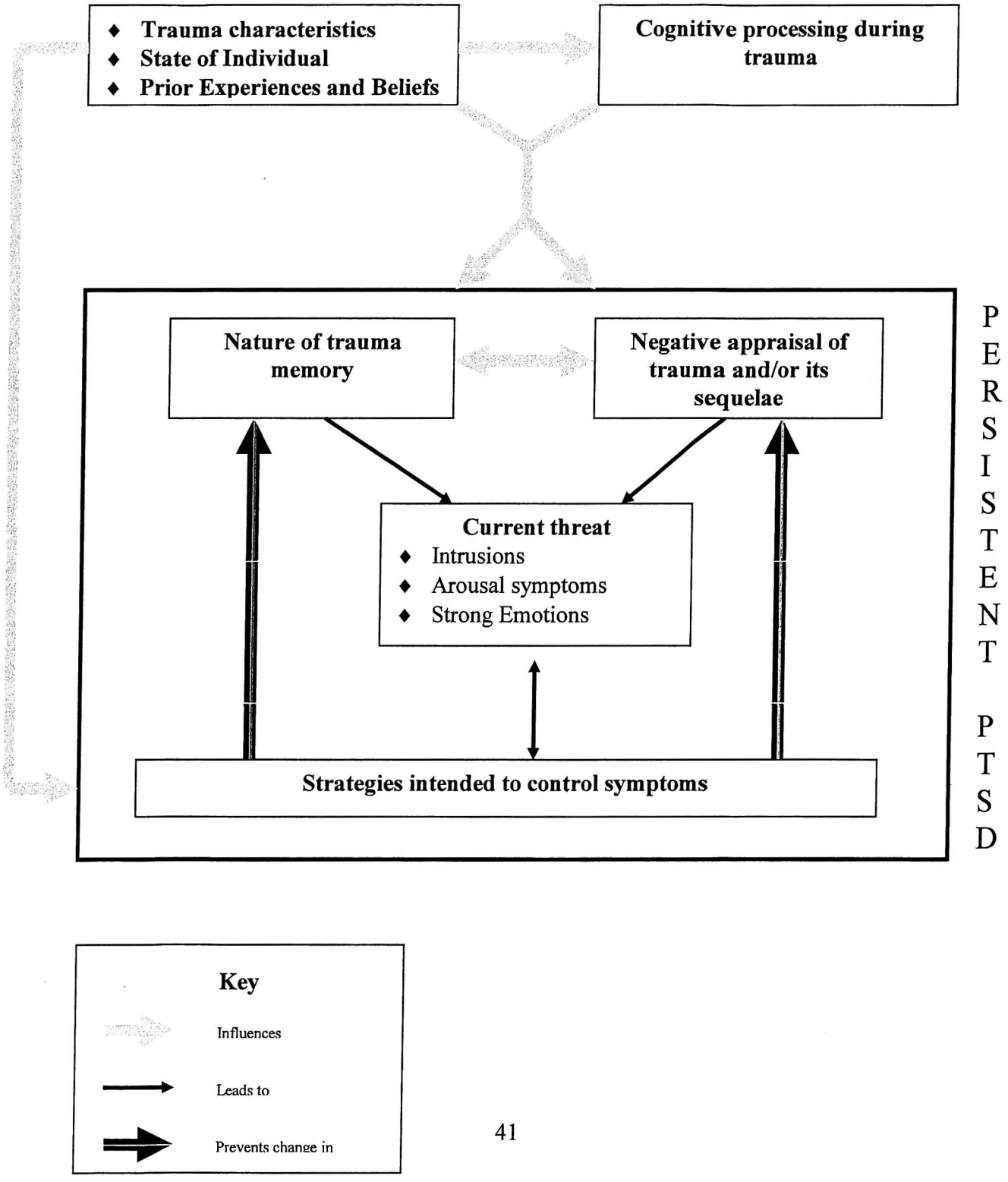
The final aspect of the model focuses on the impact of the behavioural and cognitive avoidance strategies utilised by the individual to deal with the trauma and its sequelae. Individuals often attempt to control symptoms through cognitive strategies such as thought

suppression or distraction. These strategies reduce the chance of integrating the trauma memory with other memories thus maintaining its' fragmented and sensory qualities and prolonging the symptoms. Individuals may also make behavioural attempts to avoid the trauma memory such as avoiding reminders of the event or deliberately attempting to maintain silence on the issue. These behaviours exacerbate the symptoms as they result in a failure to present contradictory information and thus a failure to modify the memory.

The Ehlers and Clark model (2000) helps to explain why some individuals develop PTSD following a trauma whereas others do not. Beliefs and appraisals occurring both during and after the trauma influence how the individual copes with both the traumatic memory and its sequelae. The difference between those who recover and those who go on to develop PTSD would appear to be the way in which the individual appraises both the trauma and its sequelae and the impact of these appraisals on the traumatic memory itself. Individuals who do not hold negative appraisals of self and behaviour are likely to show less behavioural and cognitive avoidance thus allowing greater processing of the trauma memory and thus decreasing PTSD symptoms. Individuals who avoid the traumatic memory and its sequelae fail to process the memory resulting in ongoing symptoms. Characteristics previously identified as predicting development of PTSD following a trauma (such as prior experience of trauma, helplessness during the event etc.) may be mediated through their impact on the individuals' appraisals during and after the traumatic event.

The model is best summarised diagrammatically (see fig.1). This diagram clearly emphasises the reciprocal relationships between trauma memory, behaviour and appraisal.

Figure 1. Diagrammatic representation of Ehlers and Clark (2000) model of PTSD



Expressed Emotion and Post Traumatic Stress Disorder.

The findings of Tarrier et al. (1999) suggest that living in a family of high EE can result in poorer recovery from PTSD. This is a new area of study and thus little research into how this effect could be mediated has yet been carried out. However, the models of both EE and PTSD may give us some indicators of how this effect may be mediated and thus may direct avenues of research.

Biological models propose that EE constitutes a form of psychosocial stress. Individuals demonstrate a high level of arousal in the presence of high EE relatives that lasts for the duration of their contact (Tarrier et al., 1979). In addition, there is some evidence that individuals who live with high EE relatives may demonstrate higher levels of tonic arousal. Biological models of PTSD suggest that the symptoms are maintained by high levels of arousal and a failure to habituate to stimuli that induce startle reflexes (Van Der Kolk et al., 1996). The higher levels of arousal associated with EE may therefore impact upon PTSD in one of two ways. Firstly, the higher levels of tonic arousal demonstrated by individuals living with high EE relatives may increase an individual's predisposition to the development of PTSD. It is possible that these higher levels of tonic arousal combine with the impact of the trauma to increase the likelihood of the development of biological changes which result in PTSD. The second possibility is that the heightened levels of arousal experienced when around relatives does not lead to the development of PTSD, but exacerbates the symptomatology, thus resulting in more frequent and severe symptoms.

A third possible explanation for the link between EE and PTSD at a biological level is demonstrated by the work of Pennebaker (1989). Pennebaker has produced several studies that demonstrate that the more an individual discusses their traumatic experience with others, the greater their recovery (Pennebaker and O'Heeron, 1984; Pennebaker and Susman, 1988). Laboratory studies suggested that the more individuals disclosed, the lower their skin conductance level and thus the lower their level of physiological arousal (Pennebaker, Hughes and O'Heeron, 1987). The authors demonstrated that this effect was not simply related to how much the individual talked about the trauma but to the level of emotional and factual disclosure made by the individual. For example, an individual who talked about the trauma in a factual way but failed to disclose emotions demonstrated higher levels of skin conductance than individuals who discussed the trauma whilst disclosing their emotions about the event (Pennebaker and Barger, 1988, cited in Pennebaker, 1989). Thus individuals demonstrating heightened skin conductance levels may be retaining significant emotions or facts about the trauma (Pennebaker, 1989). It is believed that this increased level of arousal when failing to disclose arises because the individual has to make a concerted effort to suppress certain aspects of the memory.

It is possible that the higher levels of skin conductance demonstrated in the presence of high EE relatives could simply reflect the individual failing to fully disclose their thoughts and feelings to the relative. This effect may not be specific to PTSD; it is possible that also in individuals with schizophrenia some characteristic of their relationship with a high EE

relative prevents them from talking freely and sharing their thoughts and emotions. The increased levels of arousal seen in the presence of high EE relatives may also reflect other factors such as a heightened sense of potential threat. This may be related to beliefs about the relatives' ability to cope with displays of emotion, beliefs about the relatives' reactions (i.e. fear of potential criticism / hostility) or may simply reflect a poorer level of communication which prevents open discussion of difficult emotions. Further research is needed to fully explore the relationship between disclosure and EE.

It is also possible that the effects of EE are mediated through cognitive factors. The attributional theory of EE looks at the impact of a relative's attributions about the illness on their behaviour. This theory suggests that relatives' behaviour is influenced by their beliefs about the patients' ability to control their symptoms. It suggests that critical behaviour (and thus high EE) results from a perception that the patient could modify their symptoms if they so desired. Criticism can therefore be perceived as an attempt to encourage the patient to change their behaviour. The model also suggests that symptoms traditionally labelled as negative (those associated with withdrawal and catatonia) are more commonly believed by relatives to be under the patients' control (Hooley, 1986). These symptoms closely parallel the avoidance symptoms seen in PTSD and thus it may be hypothesised that relatives are more likely to display negative attributions and therefore high levels of criticism/hostility if a patient displays high levels of avoidance behaviour following a trauma.

High levels of overinvolvement are proposed to have their impact through a different attribution. One theory of the impact of EOI is that relatives feel high levels of guilt (Weiner,

1986). This sense of guilt could possibly result from relatives' belief that they could have prevented the traumatic event or from relatives' sense of responsibility for the event (e.g. in situations where the relative was driving the car that crashed, of where they failed to walk a rape victim home etc.). It is therefore possible that relatives who feel high levels of guilt respond by behaving in an overprotective manner.

Although the attribution model of EE gives some indications of why relatives may feel critical/hostile or overprotective to their loved one, it does not explain how the resulting behaviours may contribute to the maintenance or event the production of symptoms in the individual with PTSD. To understand how this effect may be mediated we need to turn to the cognitive model of PTSD described by Ehlers and Clark (2000)[1]. Hostility and criticism by a key relative may impact upon the individuals' appraisal of the trauma and it sequelae. Criticism of a survivor's behaviour either during or after the trauma may cause the individual to appraise their behaviour negatively, thus exacerbating their sense of current threat. This in turn exacerbates symptoms, which is likely to result in even more criticism or hostility from the relative.

It is also likely that criticism and hostility from a relative leads to increased attempts by the individual to control their symptoms through avoidance strategies. Negative feedback about emotional outbursts or repeated retelling of the traumatic incident may lead the individual to attempt to suppress thoughts and feelings about the trauma. Due to the nature of the

[1] The Ehlers and Clark (2000) model is utilised for explanatory purposes here because it incorporates aspects of all earlier models discussed

traumatic memory, thought suppression is ineffectual (see above for a full explanation) and therefore this is likely to actually increase thoughts about the trauma. Emotional outbursts, flashbacks, and failed attempts to control symptoms are also likely to lead the individual to increase their negative appraisal of their symptoms (through increasing their beliefs about being out of control or about having changed permanently etc.), thus further exacerbating their PTSD.

Relatives' overprotective behaviour may impact upon the individuals' PTSD symptoms in a different manner. Firstly, overprotective behaviour is liable to reinforce new beliefs about the world being a dangerous place and encourage the individual to overestimate the possibility of future danger, thus exacerbating the individuals' sense of current threat. Secondly, it may be difficult for survivors to openly express thoughts and feelings about the traumatic event in front of relatives. If relatives appear to feel responsibility or guilt regarding the trauma, or if they appear to be unable to tolerate distress, the individual with PTSD is unlikely to feel able to discuss emotions and feelings freely. This in turn may prevent processing of the traumatic memories, prolonging symptoms. Thirdly, relatives' inability to contain the survivors' strong emotion may model a helpless coping stance which may increase the survivors' sense of helplessness and thus increase their sense of current threat.

Finally, one must also consider the possibility that high EE relatives' impact upon an individual's predisposition to develop PTSD following a traumatic event. Living with a highly critical or hostile relative may influence the individual's beliefs and experiences such

that they are more likely to negatively appraise a traumatic experience. For example, if an individual is exposed to criticism throughout their life, they may develop core beliefs about being useless or hopeless. The occurrence of a traumatic incidence may reinforce this belief, strengthening the belief and leading the individual to appraise the trauma negatively. A relatives' overinvolved behaviour may also influence the chance of an individual developing PTSD following a trauma as being overprotected may leave the individual with unrealistic beliefs about the dangers in the world.

The impact of living with a high EE relative is thus likely to be seen both before and after the trauma. However, some researchers argue that displaying behaviours which are quantified as high in EE is simply the relatives' attempt to cope with a difficult situation (Coelho et al, 1974). This explanation would be consistent with the social control explanation of EE discussed previously (Greenley, 1986). They would therefore argue that the critical / hostile or overinvolved behaviour is a product of the relative coping with the individual's symptoms rather than a cause of the symptoms. Only longitudinal studies can establish whether EE predates the trauma and, to date, no such studies have been carried out.

The cognitive models of both EE and PTSD give us some indications of not only why relatives act in the way that they do, but also why this might impact upon an individual's recovery from PTSD. At this point in time, there is no research available to support any of the above hypotheses. However, extrapolating links from the models may be a useful strategy for guiding further areas of research.

Future areas for research

The hypotheses postulated above give us some clear indicators of where research into this area needs to develop further. Before exploring the links presented above, more generic research into the link between EE and PTSD is needed to confirm and expand on the work of Tarrier et al. (1999). Studies exploring differences between an individual with PTSD whose family is high in EE and an individual whose family is low in EE may give some pointers as to which of the above (if any) possible explanations may be worth further exploration. Following general studies, more research into the cognitions and beliefs of both relatives and individuals with PTSD would give us clear indicators of any potential links between the two concepts. Research into psychophysiological reactions may also give us some indicators of possible mediators of family effects on PTSD. However, this research is liable to be hampered by the findings of Pennebaker (1989); it would be very difficult to separate out family effects from non-disclosure effects[2], making psychophysiological research complex and difficult to interpret.

Research in this area has a great deal of potential therapeutic value as a greater understanding of how social support, particularly family support, impacts upon development and maintenance of PTSD may help to guide developments in treatment protocols. Treatment protocols that take into account family factors may be better placed to address wider

2 see discussion above on biological models of EE and PTSD for more details

maintenance factors and are thus more likely to help the individual process their traumatic memories and reduce their PTSD symptoms.

Conclusions

The aim of this literature review was to begin the process of making links between the existing literature available on EE and that of PTSD. This review has demonstrated that there may be numerous explanations for the finding of Tarrier and colleagues (1999) that living with a high EE relative may have negative impact upon recovery rate of individuals with PTSD. A brief study of our theoretical understanding of both EE and PTSD has proposed several possible explanations for this link. At this stage, any explanations for links between these two areas of research are purely hypothetical. However, the benefit of proposing such hypotheses is that they can be utilised to guide future research. Further research into this area would have great clinical import as, whilst current treatments for PTSD are efficacious for many, some individuals fail to benefit from psychological help. Any research that promotes improvements or modifications to treatment protocols or which may raise awareness of certain factors that may hamper treatment protocols thus has great significance.

In summary, this review has demonstrated that links between EE and PTSD are theoretically possible. The challenge is for researchers to demonstrate these links in practice and to enhance our understanding of the nature of these links.

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An exploration of family communication style and its impact upon Post Traumatic Stress Disorder

Journal Guide : ***Psychological Medicine***

An exploration of family communication style and its impact upon Post Traumatic Stress Disorder

INTRODUCTION

The link between Expressed Emotion (EE) and recovery from Post-Traumatic Stress Disorder (PTSD) was investigated by Tarrier and colleagues (Tarrier, Sommerfield and Pilgrim, 1999). Their findings demonstrated that individuals who lived with a high EE relative showed a poorer recovery rate than individuals who lived with a low EE relative. This seminal paper is the first to investigate the link between EE, a well-known and much researched measure of family interaction, and PTSD. Although this study is as yet unique, its demonstration that there may be a relationship between family interactions and recovery from PTSD suggest the importance of beginning to make links between these two well researched and clinically significant areas.

Numerous studies have shown that individuals who perceive that they have adequate familial support show a better outcome upon developing PTSD (Keane, Scott, Chavoya, Lamparski, and Fairbank, 1985). However, the research of Tarrier and colleagues is the first study to investigate which specific aspects of family interaction are beneficial or harmful to the person with PTSD. Their study, however, did not address how the effect of family factors upon PTSD sufferers might be mediated. In order to broaden our understanding of both the development and maintenance of PTSD and family interaction, the ways in which the family impact upon the individual need to be explored. This study

is an initial exploration of cognitive and behavioural factors associated with PTSD that may be affected by perceptions of EE in the family environment.

The theoretical models of both EE and PTSD may offer possible explanations of the effects of family interaction upon PTSD. The following discussion briefly presents current models of EE and PTSD, exploring potential links that may provide some insight into family impact upon the development or maintenance of PTSD and thus directions for this research.

Expressed emotion

Expressed emotion is an atheoretical concept developed from research carried out in the 1950's by Brown and colleagues (Brown, Carstairs and Topping, 1958). They found that schizophrenics who returned to live with relatives after hospitalisation showed a higher rate of relapse than those who moved into single person hostels (Brown et al., 1958). This higher rate of relapse was seen in individuals who returned to live with families displaying high levels of criticism, hostility and/or emotional over-involvement (EOI) (Brown, Monck, Carstairs and Wing, 1962). The authors also noted that these families showed low levels of positive comments and warmth. Brown and colleagues labelled these families as being high in expressed emotion and developed a structured interview to measure family interactions along these five scales (Brown and Rutter, 1966).

Later research has confirmed the findings of Brown and colleagues (Jenkins et al., 1986; Leff and Vaughn, 1985; Neuchterlein et al., 1986). Increased levels of relapse or poorer outcome in individuals living with high EE relatives has also been found in numerous other psychological disorders such as depression (Hooley, Orley and Teasdale, 1986; Hooley and Teasdale, 1989; Okasha et al., 1994), eating disorders (Blair, Freeman and Cull, 1995; Szmukler, Eisler, Russell and Dare, 1985), and some physical disorders such as diabetes (Koenigsberg, Klausner, Pelino, Resnick and Campbell, 1993). These studies have all helped to demonstrate that the concept of EE has good validity (Kuipers and Bebbington, 1988) and inter-rater reliability (Kuipers, 1979).

Despite numerous supportive findings, several criticisms have been directed at the concept of EE, not least its atheoretical nature. Firstly, one cannot be sure that the interactions seen are part of one unified concept. It is possible that when measuring EE, several different interaction effects are simply being 'clumped' together. This has led to many researchers utilising only certain aspects of the subscale, reducing comparability across studies and invalidating the original concept of EE. Secondly, the concept of EE is limited by the variables measured when the concept was developed. It is possible that several other aspects of interaction also affect relapse rate but were not measured in the original research and therefore not included in the concept of EE. Thirdly, the atheoretical basis has led to difficulties in measuring EE and many have criticised the authors' decision to score categorically rather than continually. Without a theoretical explanation, the choice to categorise and the point at which to do so becomes arbitrary. These difficulties in scoring have increased the difficulties in comparability across

studies as different researchers have utilised different cut-off points. However, despite these and many other criticisms, EE is one of the few models of familial interaction available. The research in this area is extensive and has led to important clinical developments. Consequently, cautious support is maintained until an improved model of family interaction has been developed.

A number of models of EE have been developed in an attempt to answer criticisms of the atheoretical nature of EE and to further our understanding of how these interaction effects are mediated. The two main approaches are biological (see Tarrier, Vaughn, Lader, and Leff, 1979 and Tarrier and Barrowclough, 1987 for overviews of the approach) and cognitive. These models are not mutually exclusive and both have much to offer. This study will focus exclusively on the cognitive approach to both EE and PTSD.

Models of Expressed Emotion

The main cognitive theory of EE draws upon attribution theory (see Heider, 1958 and Kelley, 1967 for a full description of attribution theory). The possible role of attributions in explaining the influence of EE in various psychiatric disorders was first highlighted by the work of Leff and Vaughn (1985) and has been proposed by several other authors subsequently (Brewin, McCarthy, Duda and Vaughn, 1991; Greenley, 1986; Hooley, 1986; Joseph, Brewin, Yule and Williams, 1991). These authors all suggest that relatives' emotions, and thus possibly relatives' EE, are related to their attributions about the causes of the patients' illness, behaviours and symptoms. Hooley (1986) suggested that relatives

hold patients accountable for symptoms that they believe the patient can control but blame the *illness* for symptoms over which the patient does not appear to have volitional control. Weiner (1986) hypothesised that symptoms believed to be controllable by the patient elicit anger in a relative. This would concur with the idea that perceiving a symptom as controllable elicits hostility and criticism from a relative; the presence of which would lead to the relative being characterised as high in EE (Brewin, 1988). This hypothesis is supported by studies that demonstrate that attributions of uncontrollability elicit intolerance and distress in relatives (Butter, Brewin and Forsthye, 1986; Fincham, Beach and Nelson, 1987).

Leff and Vaughn (1985) suggested that another aspect of EE, emotional overinvolvement (EOI), may also be explained by attributional theory but felt that different attributions lead to overinvolved behaviour. They conceptualised EOI as a by-product of relatives' feelings of protectiveness and guilt concerning the patient's condition. Weiner's (1986) theory of attributions suggests that guilt results when we feel negative events are internal to, and controlled by, ourselves, suggesting that relatives might in some way feel responsible for the patient's symptoms. An alternative explanation is that relatives perceive the patient as experiencing an illness that is external to and uncontrollable by them, causing them to feel pity for the patient and become overprotective (Weiner, 1986). However, Brewin et al. (1991) found that emotionally overinvolved relatives held the same attributions as low EE relatives. This suggests that EOI may not be attributionally mediated, weakening the attributional explanation for EE as a unified concept and strengthening arguments that EE may reflect several different concepts.

Models of PTSD

Descriptions of emotional difficulties in managing the aftermath of trauma date back over 100 years (Vaughn and Tarrier, 1992). However, following the Vietnam War, a real interest in the effects of exposure to trauma developed. Exploring the experiences of Vietnam veterans and survivors of other traumatic experiences led to the development of the concept of Post-Traumatic Stress Disorder (PTSD). PTSD is characterised by three groups of symptoms: re-experiencing symptoms (such as nightmares, flashbacks, and intrusive thoughts), avoidance symptoms (such as avoidance of stimuli associated with the trauma and social withdrawal) and symptoms of increased arousal (such as sleep disturbance, impaired concentration and irritability) (American Psychiatric Association, 1980).

A number of cognitive models of PTSD exist within the literature (Brewin, Dalgelish and Joseph, 1996; Brewin, 2001; Foa and Kozak, 1986). However, this review will focus on one of the most recent and comprehensive models of PTSD; that proposed by Ehlers and Clark (2000). This model incorporates many of the ideas presented in earlier models described by Brewin et al. (1996) and Foa and Kozak (1986) and consists of two main components. The first focuses on the individuals' appraisal of the trauma and its sequelae. The model states that an individual experiences PTSD like symptoms if they appraise the event and its sequalae in such a way that they perceive an ongoing and current threat. This sense of threat may arise from appraisals of the self (e.g. I am a poor

coper) or of the world (e.g. no one can be trusted), resulting from the trauma or the symptoms arising post-traumatically.

The second aspect of the model focuses on the nature of trauma memories. Ehlers and Clark (2000) suggest that the unique way in which trauma memories are stored also contributes to a sense of ongoing threat. They describe trauma memories as being mostly sensory, difficult to remember, fragmentary, high in emotional content, triggered by numerous (even neutral) stimuli and not easily subject to modification with time. They state that trauma memories retain these unique characteristics because they are not subject to emotional processing (Rachman, 1980). This broad description fits with the descriptions of trauma memory given by earlier models (see Brewin et al., 1986, and Foa and Kozak, 1986, for a more detailed description).

To link the two components of the model (that focusing on appraisal and that focusing on the nature of trauma memory), Ehlers and Clark (2000) suggest that there is a reciprocal relationship between the trauma memory and the appraisal style. They suggest that when the individual remembers the trauma, recall is biased by the appraisals so that only information consistent with the appraisal is recollected. Thus information contradicting the ongoing sense of threat is ignored, preserving the original trauma memory. Further, Ehlers and Clark (2000) suggest that sequelae produced by the nature of the trauma memory can be interpreted as threatening. For example, the fragmented nature of the memory may be appraised as indicating a serious problem with memory, thus suggesting a current threat to the individual. They also suggest that the nature of the trauma memory

may reinforce negative self-beliefs resulting from the trauma. For example, the confused ordering of the memory may lead to a mistaken perception that the individual's actions triggered the traumatic event thus resulting in self-blame.

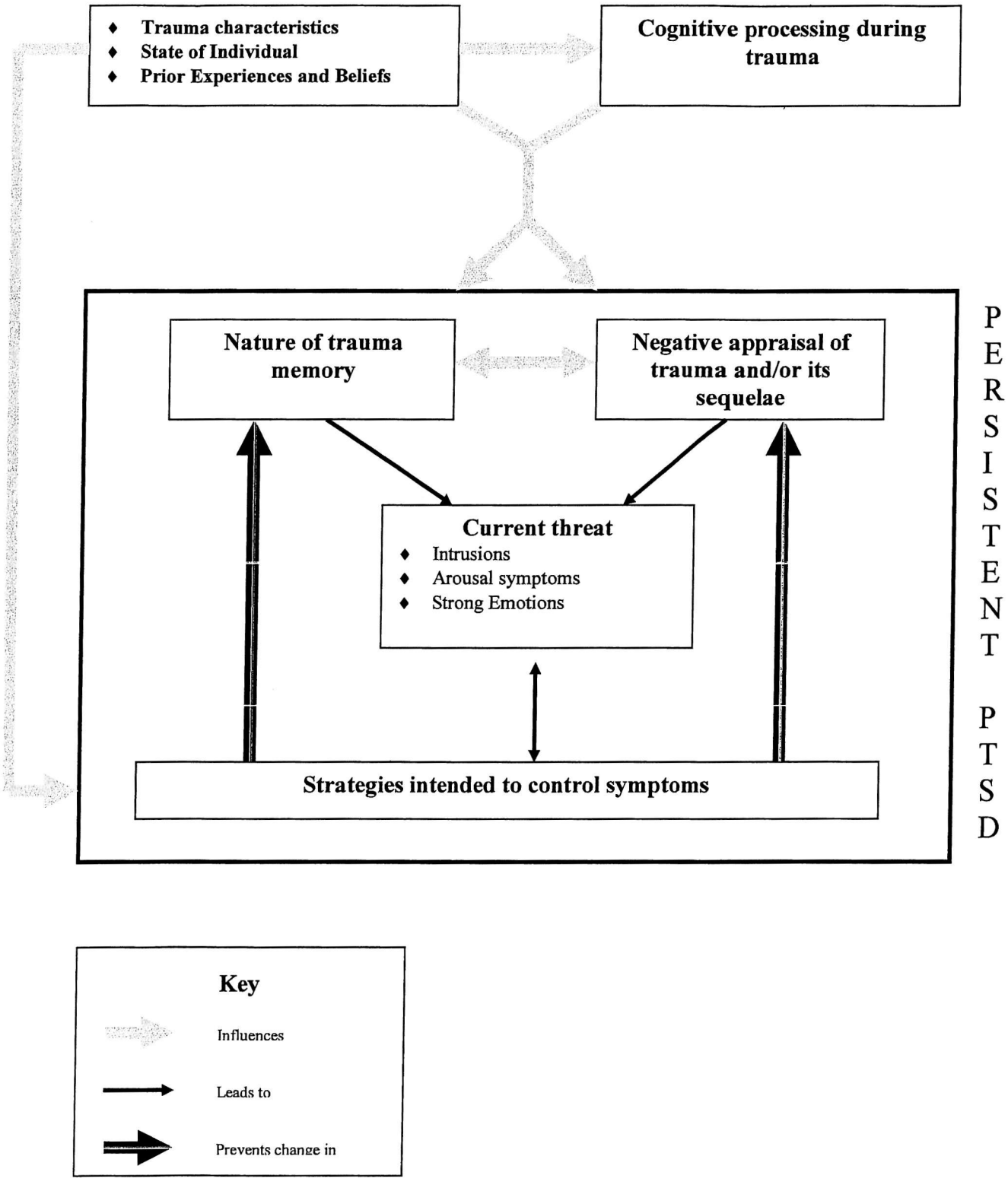
The final aspect of the model focuses on the impact of the behavioural and cognitive avoidance strategies used by the individual to deal with the trauma and its sequelae. Individuals often attempt to control symptoms through cognitive strategies such as thought suppression or distraction. These strategies reduce the chance of integrating the memory with other memories thus maintaining its fragmented and sensory quality. Individuals may also make behavioural attempts to avoid the trauma memory (e.g. avoiding reminders of the trauma). These behaviours exacerbate symptoms as they result in a failure to present contradictory information that would lead to a modification of the memory.

The Ehlers and Clark (2000) model helps to explain why some individuals develop PTSD following a trauma whereas others do not. It suggests that the difference between those individuals who recover and those who go on to develop PTSD is the way in which the trauma and its sequelae are appraised. Individuals who do not hold negative appraisals of self and behaviour are likely to show less behavioural and cognitive avoidance thus allowing greater processing of the trauma memory and decreasing PTSD symptoms. Characteristics previously identified as predicting development of PTSD following a trauma (such as prior experience of trauma, helplessness during the event etc.) may be

mediated through their impact on the individuals' appraisals during and after the traumatic event.

The model is best summarised diagrammatically (see fig.1). This diagram clearly emphasises the reciprocal relationship between trauma memory, behaviour and appraisal.

Figure 1. Diagrammatic representation of Ehlers and Clark (2000) model of PTSD



Links between the models of PTSD and EE

The findings of Tarrier et al. (1999) suggest that living in a family characterised by high EE results in poorer recovery from PTSD, although it is not known exactly how this effect occurs. The models of both EE and PTSD may give us some indicators of ways in which EE might impact upon PTSD.

The attributional model of EE suggests that relatives' emotions (and thus their behaviours and resulting level of EE) are determined by their beliefs about the cause of, and responsibility for, the patients' symptoms. According to the model, believing the patient to be in control leads relatives to feel angry and thus critical. Believing patients' symptoms to result from illness per se or believing themselves to be responsible leads to overprotective behaviour. However, this explanation does not tell us *why* a relative behaving in a certain manner might impact upon the patients' symptoms. The cognitive model of PTSD (Ehlers and Clark, 2000) may offer some potential explanations.

Hostile/critical behaviour may impact upon PTSD through its effects on the individual's appraisals of the trauma and its sequelae. Criticism of a survivor's behaviour either during or after the trauma may cause the individual to appraise their behaviour negatively, thus exacerbating their sense of current threat. This in turn will exacerbate symptoms, which may result in further criticism or hostility from the relative.

It is also possible that criticism and hostility from a relative leads to increased attempts by the individual to control their symptoms through avoidance strategies. Negative feedback regarding emotional outbursts or repeated retelling of the traumatic incident may lead the individual to attempt to suppress thoughts and feelings about the trauma. Due to the nature of the traumatic memory, thought suppression is ineffectual and therefore this is likely to increase thoughts about the trauma (see Van der Kolk, McFarlane and Weisaeth, 1996 for details). Emotional outbursts, flashbacks, and failed attempts to control symptoms are also likely to lead the individual to increase their negative appraisal of their symptoms (through increasing their beliefs about being out of control or about having changed permanently etc.), thus further exacerbating their PTSD symptomatology.

Conversely, overprotective behaviour on the part of a relative may reinforce beliefs about the world being a dangerous place and encourage the individual to overestimate the possibility of future danger, exacerbating the individual's sense of current threat. Further, it may be difficult for sufferers to openly express thoughts and feelings about the traumatic event in front of relatives who appear to feel responsibility or guilt regarding the trauma or appear to be unable to tolerate distress. Also, a relative's inability to contain the sufferer's strong emotions may model a helpless coping stance that may increase the sense of helplessness and thus increase the sense of current threat. These factors may prevent processing of traumatic memories, prolonging symptoms.

Finally, one must also consider the possibility that high EE relatives impact upon an individual's predisposition to develop PTSD following a traumatic event. Living with a highly critical or hostile relative may influence individuals' beliefs and experiences increasing the likelihood of negative appraisals of traumatic experiences. Exposure to criticism throughout life may, for example, lead to the development of core beliefs about being useless or hopeless. The occurrence of a traumatic incidence may reinforce this belief, leading to negative appraisals of the trauma.

The above models offer a number of potential explanations for possible links between EE and PTSD. However, general exploration of any potential links is necessary before developing clear hypotheses regarding the association between EE and PTSD.

The study

The work of Tarrier et al. (1999) demonstrates that living with a relative high in expressed emotion reduces the probability of recovering from PTSD following treatment. An exploration of the models suggests that EE may impact upon an individual's predisposition to develop PTSD and/or factors maintaining the disorder. The current study represents an initial exploration of the possible ways in which EE may impact upon the development and maintenance of PTSD.

Exploration of the links between theoretical models of PTSD and EE results in the generation of several specific hypotheses as to the way in which EE may impact upon

PTSD. The aim of this study is therefore to investigate the validity of these specific hypotheses.

Respecting the confidentiality and privacy of service users together with the organisational constraints of adult mental health services may lead to difficulties in accessing family support systems in order to assess EE status. Therefore this study will utilise a relatively new method of assessing EE level; the Levels of Expressed Emotion Scale (LEE). The LEE is a self-report questionnaire completed by the individual service user which measures *perceived* levels of Expressed Emotion (for more details on the measure, see Method). This has been demonstrated to correlate with actual levels of Expressed Emotion (Kazarian, Cole, Malla, and Baker, 1990) and overcomes many of the difficulties noted above. Thus references made to relatives' level of Expressed Emotion throughout this study will actually refer to relatives' level of Expressed Emotion, *as perceived by the individual with PTSD.*

Research Hypotheses

The following hypotheses were generated from ideas as to possible links between EE and PTSD drawn from theoretical models (see introduction for a more detailed explanation of their genesis).

1. Participants living with relatives rated as high EE on the criticism / hostility scale will display significantly higher levels of / more severe cognitions regarding the trauma and its sequalae.

2. Participants living with relatives rated as high EE on the criticism / hostility scale will display significantly higher levels of avoidance behaviours.

3. Participants living with relatives rated as high EE on the emotional overinvolvement scale will display significantly higher levels of / more severe cognitions regarding the world.

4. Participants living with relatives rated as high EE on the emotional overinvolvement scale will display significantly higher levels of avoidance behaviours

One other final hypotheses was drawn from the literature review; namely that living with a high EE relative may increase one's predisposition to develop PTSD. This hypothesis will not be investigated within this study as it would require a longitudinal design which is outside of the scope of this study.

METHOD

Design

Participants were recruited from waiting lists of secondary care services throughout East and West Hampshire. Individuals who were referred to the services with a tentative diagnosis of Post-Traumatic Stress Disorder or Depression / Anxiety following a traumatic event were identified as potential participants and were contacted and invited to take part in this study. Once informed consent had been obtained, participants were invited to a one and a half-hour assessment session (see assessment: participant assessment).

Participants who met the following inclusion criteria were entered into the main part of the study: A DSM-IV diagnosis of PTSD elicited by the PDS (Foa, 1995); duration of PTSD of at least 6 months but not more than 10 years; childhood sexual abuse was not the index trauma; the individual was not suffering from substance misuse as a primary problem; any medication prescribed to the individual must have been received for greater than three months prior to the assessment phase; the individual must not have received past psychological treatment for their PTSD and must not have engaged in any cognitive behavioural therapy in the past 6 months.

As participants were currently waiting for psychological input, the second half of the assessment session was utilised for their benefit. Participants were given self-help

literature and the opportunity to ask questions about their diagnosis and their forthcoming treatment.

At the assessment session, participants were asked if they were willing for their relatives to be contacted. If consent was given, a letter was sent to the identified key relative to ask if they would also be willing to participate in the study. Once informed consent was obtained, relatives were asked to participate in a 10 minute phone conversation, during which the five minute speech sample was completed (assessment 2: relative assessment).

Sample

51 individuals were identified as potential participants and sent letters of invitation to participate in the study. Of these, 29.4% (15/51)) responded. Those responding all took part in the study and, at interview, 53% (8/51) participants agreed to allow their relatives to be contacted.

75% of relatives contacted agreed to take part in the study (6/8) and all participated in a telephone conversation as requested.

Participants

Of the 15 participants, 53% (8/15) were female and 47% (7/15) were male. The mean age of the participants was 39.13 (s.d. = 11.49, range 20 to 58). Of the 15 participating, 33% (5/15) named a spouse as their key relative, 20% (3/15) named a common law partner,

20% (3/15) named a close friend with whom they lived and 27% (4/15) named a parent. The participants experienced a wide range of traumatic experiences including; accident involving a vehicle (n = 6, 40%), assault (n = 3, 20%), rape (n = 3, 20%), conflict (n = 1, 6.7%), torture (n = 1, 6.7%) and work accident (n = 1, 6.7%).

Of the 6 relatives agreeing to take part in the study, 50% (3/6) were the participants' common law partner, 33% (2/6) were the participants' spouse and 17% (1/6) was the participants' mother. 33% (2/6) of the relatives were female and 66% (4/6) male. Other personal details were not collected from relatives.

Assessment

Assessment 1: Participant assessment

Participants were asked to complete the following self-report questionnaires:

1. *The Post-Traumatic Stress Diagnostic Scale (PDS)(Foa, 1995).*

The PDS is a self-report questionnaire which has been demonstrated to have high validity and reliability (Foa, 1995). It was utilised to assess whether participants met the full DSM-IV criteria for Post-Traumatic Stress Disorder.

2. *Impact of Event Scale (IES) (Horowitz, Wilner and Alvarez, 1979)*

The IES is a self-report measure of post-traumatic stress which has been demonstrated to have high internal consistency (Zilberg, Weis and Horowitz, 1992), high validity, and good reliability (Ferring and Filepp, 1994). It contains 15 items, which are subdivided

into two scales: intrusion and avoidance. This measure was utilised to give an index of the degree of avoidance behaviours engaged in.

3. *Post Traumatic Cognitions Inventory (PTCI) (Foa, Ehlers, Clark, Tolin, Orsillo, 1999)*

The PTCI is a 36 item self-report questionnaire designed to access the trauma-related cognitions of the individual. The PTCI has been demonstrated to have excellent internal consistency and high test-retest reliability (Foa, Ehlers, Clark, Tolin and Orsillo, 1999). Scores on the PTCI are subdivided into three scales: negative cognitions about the self, negative cognitions about the world, and self-blame. The PTCI was utilised to obtain a measure of both the number and the nature of trauma related cognitions experienced by the individual.

4. *The Dissociative Experiences Scale (DES) (Carlson and Putnam, 1993)*

Several cognitive models of PTSD have highlighted the use of dissociation as an active avoidance strategy. The DES takes the form of a 28 item self-report measure that gives an overall measure of dissociative experiences. It has been demonstrated to have high validity and reliability (Carson and Putnam, 1993). The DES was utilised as a measure of dissociation to supplement the general findings of the IES.

5. *The Level of Expressed Emotion (LEE) (Cole and Kazarian, 1988)*

The LEE is a self-report measure of perceived Expressed Emotion within the family. It has been demonstrated to have high internal validity and high test retest reliability (Cole

and Kazarian, 1988). It has also been demonstrated to have high correlation with the Camberwell Family Interview (Brown and Rutter, 1966), the original method of assessing EE (Kazarian, Cole, Malla, and Baker, 1990).

The LEE is subdivided into 4 subscales; intrusion, attitude towards illness, tolerance/expectation and emotional responsiveness. The subscales were devised from the correlates of EE calculated by Vaughn and Leff (Vaughn and Leff, 1981). The intrusion scale was designed to correspond with the emotional overinvolvement scale of the CFI and the other three subscales correspond with the criticism / hostility subscales of the CFI (Cole and Kazarian, 1988). The total score on the measure can be used to allocate individuals to an expressed emotion category (see Cole and Kazarian, 1988 for more details on the allocation method) or can be used as an index of perceived expressed emotion. It was utilised to give a measure of the level of Expressed Emotion displayed by the key relative identified by the participant

Assessment 2: Key Relative assessment

6. *Five-minute speech sample (FMSS)(Magana, Goldstein, Kamo and Miklowitz, 1986).*

The five-minute speech sample takes the form of an interview in which the key relative is encouraged to give their view of the participant. The FMSS has been demonstrated to have good reliability and validity (Malla, Kazarian, Barnes and Cole, 1991) and compares well to the Camberwell Family Interview (Moore and Kuipers, 1999). In this study, the interview was carried out over the telephone, a method demonstrated to have comparable validity to the face to face version originally utilised (Beck, Daley, Hastings

and Stevenson, in press). The key relative is encouraged to talk spontaneously and is given no feedback on their responses. Comments are coded for evidence of criticism, warmth, emotional over-involvement, and hostility. These codes are collated and the key relative is given high or low EE status, depending on the number and type of responses made. Relatives displaying more than one critical comment, a negative initial statement a negative relationship rating, self-sacrificing / overprotective behaviour, emotionality during the interview and / or excessive praise were allocated a rating of high Expressed Emotion (Magana, Goldstein, Kamo and Miklowitz, 1986).

RESULTS

Data Analysis

Stage 1: Validation of the EE categories allocated by the LEE score

The FMSS was utilised to corroborate the groupings of participants according to the perceived EE status of their relative as calculated in stage 1 of the data analysis. For those relatives who completed the FMSS, their EE categorisation as given by the FMSS was compared with their perceived EE categorisation as given by the LEE.

Stage 2: Exploration hypotheses using Pearson's r correlation

It was initially intended to investigate the hypotheses using ANOVAs. Individuals would be categorised into high and low EE groups, depending on the EE status of their relative as measure by the LEE. However, categorisation revealed that the numbers of individuals in each group were very small, minimising the validity of statistical analysis using ANOVAs. In addition, the validity of the categorisation methodology for the LEE (Cole and Kazarian, 1988) is also questionable as there is no theoretical justification for using a median split. The median was calculated from one study and thus it cannot be concluded that this median is representative of the population as a whole.

It was therefore decided to utilise a correlational to investigate the four hypotheses specified earlier in the study. Parametric statistics were utilised as KS tests demonstrated that all of the variables were normally distributed.

Results

Stage 1: Comparison of FMSS and LEE categorisation

Of the 6 individuals completing the FMSS, 66.67% (4/6) were rated as low in Expressed Emotion. Categorisation on the FMSS matched with categorisation on the LEE in all cases.

Stage 2: Correlational analysis

Pearson's r correlations were used to test each of the four hypotheses specified in the introduction. For all of the below correlations, n = 15 and therefore degrees of freedom = 14.

Hypothesis 1:

Participants living with relatives rated as high EE on the criticism / hostility scale will display significantly higher levels of / more severe cognitions regarding the trauma and its sequalae.

This hypothesis was tested by looking for an association between the three subscales of the LEE measuring criticism / hostility (Emotional responsiveness, tolerance / expectation and attitude towards illness) and cognitions as measured by the various subscales of the PTCI.

Table 1: Associations between scores on three criticism / hostility subscales of the LEE and cognitions as measured by the PTCI.

	LEE: Emotional Responsiveness		**LEE: Attitude towards illness**		**LEE: Tolerance/ Expectation**	
	r value	**P value**	**r value**	**P value**	**r value**	**P value**
PTCI: Beliefs about the world	0.694**	0.004**	0.580*	0.023*	0.665**	0.007**
PTCI: Beliefs about the self	0.284	0.304	0.245	0.378	0.355	0.195
PTCI: Self-blame	0.508	0.053	0.446	0.095	0.520*	0.047*
PTCI: Total	0.527*	0.044*	0.447	0.095	0.560*	0.030*

* = P<0.05
** = P<0.01

The results show positive correlations between the PTCI beliefs about the world subscale and all three of the LEE subscales; the LEE emotional responsiveness subscale ($r = 0.69$ $P<0.01$), the LEE tolerance/expectation subscale ($r = 0.67$, $P<0.01$), and the attitude towards illness subscale ($r = 0.58$, $P<0.05$).

Significant positive correlations were also found between the PTCI self-blame subscale and the LEE tolerance/expectation subscale ($r = 0.52$, $P<0.05$). There was also a positive trend between the emotional responsiveness subscale of the LEE and the PTCI self-blame subscale ($r = 0.51$, $P = 0.05$).

Finally, the total score on the PTCI correlated with both the emotional responsiveness subscale of the LEE ($r = 0.53$, $P<0.05$) and the tolerance/expectation subscale ($r = 0.56$, $P<0.05$).

These findings support the hypothesis that participants living with relatives rated as high EE on the criticism / hostility scale will display significantly higher levels of / more severe cognitions regarding the trauma and its sequalae. However, they also suggest that this is a simplistic picture of a more complex relationship between various aspects of criticism / hostility and different types of cognitions (see discussion for further comment).

Hypothesis 2:

Participants living with relatives rated as high EE on the criticism / hostility scale will display significantly higher levels of avoidance behaviours.

This hypothesis was tested by looking for an association between the three subscales of the LEE measuring criticism / hostility (emotional responsiveness, tolerance / expectation and attitude towards illness) and avoidance behaviours as measured by the IES (avoidance subscale and total score), PDS (severity and symptom scores) and the DES.

Table 2: Associations between scores on three criticism / hostility subscales of the LEE and avoidance behaviours.

	LEE: Emotional Responsiveness		**LEE: Attitude towards illness**		**LEE: Tolerance/ Expectation**	
	r value	**P value**	**r value**	**P value**	**r value**	**P value**
IES: Avoidance	0.032	0.909	-0.275	0.321	0.061	0.828
IES: Total score	0.031	0.913	-0.199	0.477	0.093	0.741
PDS: Severity	-0.112	0.691	-0.117	0.677	-0.077	0.785
PDS: Symptom	0.373	0.171	0.022	0.938	0.229	0.412
DES	0.227	0.415	0.068	0.810	0.223	0.425

No significant associations between any criticism / hostility subscale of the LEE and any measure of avoidance behaviours were found. Therefore hypothesis 2 was not supported.

Hypothesis 3:

Participants living with relatives rated as high EE on the emotional overinvolvement scale will display significantly higher levels of / more severe cognitions regarding the world.

This hypothesis was tested by looking for any associations between the intrusion subscale of the LEE and negative cognitions about the world, as measured by the PTCI.

Table 3: Associations between scores on the intrusion subscale of the LEE and cognitions about the world.

	LEE: Intrusion	
	r value	**P value**
PTCI: Beliefs about the world	0.364	0.182
PTCI: Total	0.308	0.264

No significant associations between the intrusion subscale of the LEE and beliefs about the world were found. This hypothesis was therefore not supported.

Hypothesis 4:

Participants living with relatives rated as high EE on the emotional overinvolvement scale will display significantly higher levels of avoidance behaviours

This hypothesis was tested by looking for any associations between the intrusion subscale of the LEE and avoidance behaviours as measured by the IES avoidance subscale, the IES total score, the PDS symptom and severity scores and the DES.

Table 4: Associations between scores on the intrusion subscale of the LEE and avoidance behaviours.

	LEE: Intrusion	
	r value	**P value**
IES: Avoidance	0.461	0.084
IES: Total score	0.557*	0.031*
PDS: Severity	-0.057	0.839
PDS: Symptom	-0.166	0.555
DES	0.473	0.075

* = P<0.05

A significant association between the intrusion subscale and the IES total score was found ($r = 0.557$, $p<0.05$). The intrusion avoidance scale also showed a trend towards significant ($r = 0.461$, $p = 0.084$). However, no other associations between measures of avoidance and the intrusion subscale of the LEE were seen. This hypothesis therefore remains unsupported. However, the trend towards significant suggests that further investigation of this area may be merited (see discussion for more details).

Summary of results

Analysis of the data using Pearson's r correlations showed that only hypothesis 1 was well supported by the data. No evidence was found in support of hypotheses 2 and 3 and only very weak and conflicting evidence was found to support hypothesis 4.

DISCUSSION

Significant findings

The results of this study show tentative support for hypothesis one; the findings suggest that there is a significant correlation between the PTCI and those subscales of the LEE corresponding to criticism / hostility. The findings also revealed that the negative thoughts about the world and self-blame subscales of the PTCI were more strongly correlated with the subscales of the LEE.

The findings suggest that the three subscales of the LEE representative of criticism / hostility are not equally correlated to the PTCI and its various subscale. Most notably, the tolerance / expectation and emotional responsiveness subscales of the LEE showed the strongest correlations ($P<0.01$) to the PTCI total score and to the PTCI self-blame and negative thoughts about the world subscale. The attitude towards illness subscale, the other subscale corresponding with criticism / hostility aspects of EE, showed only a trend towards significance when correlated with the PTCI total score.

The other three hypotheses postulated earlier in the study were not supported by the findings. No correlations were found between the criticism / hostility subscales of the LEE and avoidance behaviours, between the intrusion subscale of the LEE and the PTCI and between the intrusion subscale of the LEE and avoidance behaviours. However, correlational analysis of the relationship between the intrusion subscale and avoidance behaviours showed a trend towards significance, suggesting that this area might merit further investigation.

In their study, Tarrier et al. (1999) suggest that living with a high EE relative is a contributing factor to poor treatment outcome and thus persistence of PTSD. The current findings suggest that the poor treatment outcome seen in Tarrier et al.'s (1999) study may be associated with the cognitions held by the individual with PTSD. However, the results of the current study do not enable us to draw any conclusions as to causality. One might speculate that individuals with high levels of trauma related cognitions cause their relative to display high EE behaviours *or* that living with a high EE relative causes the individual to hold more trauma related cognitions. To investigate this issue further, an extensive mediational study would be necessary in order to establish the direction of causality (see Baron and Kenny, 1986, for models of mediation). Thus, all that can be concluded at this point is that there is an association between the perceived level of EE of a relative and the number and severity of trauma related cognitions held by the individual.

Limitations of the study

Caution is necessary in the interpretation of these results. The sample size used was very small, reflecting the difficulty of recruiting participants from a clinical population. For firm conclusions to be drawn, repetition of this study with larger participant numbers is essential.

The study does not allow the exploration of causality. This could be done through utilising mediational models (e.g. Baron and Kenny, 1986). This would require repetition of the study with a much larger number of participants and measures as, for a regression analysis to be possible, multiple measurements of each variable are necessary.

In addition, there are difficulties in the use of the LEE as a measure of expressed emotion. The LEE measures perceived EE and is less thoroughly researched than the CFI (Brown and Rutter, 1966; Moore and Kuipers, 1999) or the FMSS (Magana et al., 1986). Attempts were made to overcome this problem by supplementing the LEE with the FMSS for a small subsample of relatives who were willing to participate, in order to validate the categories allocated by the LEE. However, the group of relatives agreeing to participate in the study may not have been representative of the relatives as a whole. Of those agreeing to complete the FMSS, 66.67% (4 / 6) were categorised as low in expressed emotion and 33.33% (2 / 6) were categorised as high in expressed emotion (identical scorings were given by the LEE). This group therefore contained a disproportionately low number of low EE relatives when compared with the LEE scores of the total sample of the LEE. On the LEE, 26.67% (4 / 15) were judged as low in EE

whereas 73.33% (11 / 15) were categorised as high in EE. This difference in proportion might be explained in two ways. Firstly, the LEE may be over-inclusive in its categorisation of the high EE group or secondly, that low EE relatives are more willing to participate in this kind of research (possibly in order to help their relative). It is hard to establish which of the two possibilities is more likely from this data.

The study of Tarrier et al. (1999) found that 49% of relatives were categorised as high in EE and 51% as low. These figures fall between the FMSS and LEE scoring in the current study. It is possible that the scores on the FMSS are an underestimate of the true EE level of the whole sample and the LEE an overestimate. More substantive investigations with larger samples would be necessary to further explore differences between the FMSS and the LEE. However, these findings do suggest that its is possible that requiring relative participation may skew the sample as more supportive relatives, and thus low EE relatives, are likely to agree to participate in research. In contrast, utilising the LEE may lead to an overestimation of the level of EE of a relative as they may be *perceived* to be more critical and intrusive than their behaviours suggest.

A further criticism of this study, and probably of any EE research into disorders other than schizophrenia, is the origin of the EE measures, particularly the LEE. The LEE was developed from research with individuals diagnosed with schizophrenia. As such, its questions are most relevant to this population. Some aspects of the measure, particularly the subscale "attitude to illness", were designed to reflect the particular needs of individuals with schizophrenia and, as such, may not be relevant to individuals with

PTSD. This criticism is supported by anecdotal reports from those completing the questionnaire, which stated that relatives were not supportive but in different ways from those asked about. This may in part explain the finding that this subscale of the LEE was only correlated to one subscale of the PTCI and not to the total score, despite the high correlations of some of the other subscales of the LEE. It may therefore be necessary to modify existing EE measures to reflect the characteristics associated with psychological disorders other than schizophrenia.

Comment

Two interesting issues arise from the current study. Firstly, this study supports previous studies which suggest that high expressed emotion (criticism / hostility) differs in its method of impact from high expressed emotion (emotional overinvolvement). This study demonstrates a link between high levels of criticism / hostility (as measured by the emotional responsiveness, attitude towards illness and tolerance / expectation subscales of the LEE) and the cognitions held by the individual with PTSD. However, no similar link is seen between high levels of emotional overinvolvement (as measured by the intrusion subscale of the LEE) and cognitions. This finding is consistent with previous research (Tarrier and Barrowclough, 1987; Leff and Vaughn, 1985), which has also noted that the impact of living with relatives high in criticism/hostility differs from the impact of living with an emotionally overinvolved relative, despite both being classified as high EE. The noted difference between EOI and criticism/hostility is also supported by the theory; the attributional model of EE proposes that the effects of criticism/hostility are

mediated differently to EOI (see Introduction for a more detailed discussion of the attributions model explanation of EOI and criticism/hostility). Therefore the findings above suggest that EOI and criticism/hostility may impact upon PTSD in different ways. As this study was exploratory only, further, specific investigation of this difference is necessary. However, even without further research, this finding suggests that caution is needed when researching the impact of EE as a global concept.

The second issue relates to the particular pattern of cognitions demonstrated to be different in those living with a relative perceived to be high in EE. The PTCI is subdivided into three subscales; beliefs about the world, beliefs about the self and beliefs regarding self-blame. The results show that only beliefs about the world and beliefs regarding self-blame correlate with high levels of perceived EE. This suggests that individuals with PTSD show no higher levels of negative beliefs about the self, even when living with highly critical or highly intrusive relatives.

Earlier in this paper it was hypothesised that critical/hostile (and thus high EE) behaviour on the part of the relative may impact upon PTSD through its effects on the individual's appraisals of the trauma and its sequelae. The veracity of this hypothesis can not be determined from this study alone as the design included no attempt to ascertain causality. However, the findings of this study do suggest a modification of this hypothesis if it is to be utilised as the basis of future research; namely that perceiving a relative to be critical influences the individuals' appraisals *of the world and of their responsibility for the occurrence of the trauma.* This finding is supportive of the Ehlers and Clark (2000)

model of PTSD, which suggests that holding negative appraisals of the world and feeling responsible for the trauma can lead an individual to perceive ongoing threat, leading to greater levels of arousal and thus greater levels of PTSD symptoms. However, a mediational study (as discussed previously) would be needed to attempt to validate this hypothesis.

At this stage, one can only speculate why having a relative perceived as being high in EE is associated with holding more negative beliefs about the world. It is possible that perceiving one's relative to be highly critical results in the individual experiencing limited support and empathy from their immediate environment. This may leave the individual with a negative perspective of others and the world around them. Alternatively, it is possible that living with a relative perceived to be highly critical could increase the individual's sense of threat (their self-esteem being under threat from criticism by the relative). This could increase their levels of arousal, leaving them more hypervigilant and thus more likely to attend to threats in the world around them. Thus high levels of arousal may lead to an increase in negative cognitions, rather than the other way round. Studies which detail the exact cognitions regarding the world held by the individual as well as further studies of arousal levels and their links to particular cognitions may give us some insights into this issue.

The Ehlers and Clark (2000) model of PTSD offers a potential explanation for the association between self-blaming cognitions and living with a relative perceived to be highly critical. A survivor exposed to criticism may alter their appraisals of responsibility

for the trauma, or further, their appraisals of their response to the trauma. It is possible that self-blaming may be reinforced by a lack of support on the part of the relative; withholding emotional support may leave the individual feeling undeserving of support and lead to reappraisals of their role in or response to the trauma. This speculation merits further investigation as, if supported, there are implications for the maintenance of PTSD symptomology.

Three hypothetical links between EE and PTSD were posited which appear to be unsupported by the current findings. Firstly, it was speculated that being exposed to perceived criticism from a relative might lead to higher states of arousal, which might encourage an individual to engage in cognitive or behavioural avoidance. However, no significant associations between the subscales of the LEE corresponding with critical/hostile behaviour and avoidance behaviours (as measured by the IES) were found.

Secondly, it was posited that living with an intrusive (emotionally overprotective) relative might lead the individual to hold more negative beliefs about the world. It was hypothesised that attempts to protect the individual would reinforce their negative appraisals regarding the safety of the world. Again, no association between perceived intrusion and negative beliefs about the world was found.

Finally, it was suggested that living with a highly emotionally overinvolved relative may lead the individual to engage in higher levels of avoidance behaviours. Although, the findings did not clearly support this hypothesis, there was a trend towards a correlation

between high EOI (as measured by the intrusion subscale of the LEE) and avoidance (as measured by the IES). As this study has serious limitations, particularly as this study has such a small sample size, it is possible that repetition with a higher sample size may demonstrate more support for this hypothesis. Further research in this area is therefore necessary.

Conclusions

The results of this study show an association between the cognitions held by the individual with PTSD and the perceived EE rating of their identified key relative. This finding must be accepted somewhat cautiously, as there are several limitations to this study, most notably, the small sample size. However, this study gives us some further insights into the findings of Tarrier et al. (1999) that living with a high EE relative is associated with poorer treatment outcome in PTSD. Further, these findings fit well with the cognitive model of PTSD (Ehlers and Clark, 2000). The results suggest that holding more negative cognitions about the world and about responsibility for the trauma is associated with living with a relative perceived as high in EE. According to the Ehlers and Clark (2000) model, appraising the world and self negatively contributes to the maintenance of PTSD. Therefore, the findings suggest that the poorer outcome seen in individuals living with relatives high in EE (Tarrier et al., 1999) may be in part due to their cognitions.

Future research

Before expanding on the findings of this study, replication with larger numbers is essential. If possible, replication utilising direct measures of EE rather than perceived measures of EE should be completed in order to differentiate between direct measurement and perceptions of EE. Replication with direct measures of EE would also confirm that it is the relatives behaviour that is important not the individual's perception of the behaviour. However, as discussed previously, caution should be taken that a recruitment bias does not occur.

The findings suggest that further exploration of the link between the cognitions of individuals with PTSD and EE is merited. In particular, mediational studies would be helpful in establishing causality and in confirming a direct association between EE and cognitions. The variability accounted for may point to putative third factors causing both high EE behaviours in relatives and the presence of more trauma related cognitions in the individual. Finally, further exploration of the content and nature of cognitions held by individuals is essential to understanding how they may be linked with EE and increase our understanding of areas to be modified in treatment.

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APPENDICES

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APPENDIX 1 : Post Traumatic Cognitions Inventory

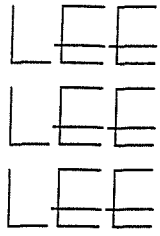
Post Traumatic Cognitions Inventory (PTCI)

We are interested in the kind of thoughts you may have had after a traumatic experience. Below are a number of statements that may or may not be representative of your thinking.
Please read each statement carefully and tell us how much you AGREE or DISAGREE with each statement.
People react to traumatic events in many different ways. There are no right or wrong answers to these statements.

	Totally Disagree	**Disagree very much**	**Disagree slightly**	**Neutral**	**Agree slightly**	**Agree very much**	**Totally agree**
1. The event happened because of the way I acted							
2. I can't trust that I will do the right thing							
3. I am a weak person							
4. I will not be able to control my anger and will do something terrible							
5. I can't deal with even the slightest upset							
6. I used to be a happy person but now I am always miserable							
7. People can't be trusted							
8. I have to be on guard all of the time							
9. I feel dead inside							
10. You can never know who will harm you							
11. I have to be especially careful because you can never know what can happen next							
12. I am inadequate							
13. I will not be able to control my emotions and something terrible will happen							
14. If I think about the event, I will not be able to handle it							
15. The event happened to me because of the sort of person I am							
16. My reactions since the event mean that I am going crazy							
17. I will never be able to feel normal emotions again							
18. The world is a dangerous place							
19. Somebody else would have stopped the event from happening							
20. I have permanently changed for the worse							

	Totally disagree	Disagree very much	Disagree slightly	Neutral	Agree slightly	Agree very much	Totally agree
21. I feel like an object, not like a person							
22. Somebody else would not have gotten into this situation							
23. I can't rely on other people							
24. I feel isolated and set apart from others							
25. I have no future							
26. I can't stop bad things from happening to me							
27. People are not what they seem							
28. My life has been destroyed by the trauma							
29. There is something wrong with me as a person							
30. My reactions since the event show that I am a lousy coper							
31.There is something about me that made the event happen							
32. I will not be able to tolerate my thoughts about the event, and I will fall apart							
33. I feel like I don't know myself any more							
34. You never know when something terrible will happen							
35. I can't rely on myself							
36. Nothing good can happen to me anymore							

APPENDIX 2 : The Levels of Expressed Emotion Scale



LEVEL OF EXPRESSED EMOTION SCALE

Client Version

John D. Cole, Ph.D.
Shahe S. Kazarian, Ph.D.

Instructions:

The following are a number of statements that describe the way in which someone may act towards you. Please identify the person who has been most influential in your life during the past three months. Examples of influential persons could be: mother, father, brother, sister, husband, wife, relative (e.g., aunt, grandfather) and friend. Then, read each statement and indicate whether this person has acted in these ways towards you over the past three months.

Mark your answers on the separate Answer Sheet provided. Simply circle the (T) box if you feel that the item is TRUE. Circle the (F) box if you feel the item is FALSE. It is important to make sure that the statement number agrees with the number of your response on the Answer Sheet.

1. Understands if sometimes I don't want to talk.
2. Calms me down when I'm upset.
3. Says I lack self-control.
4. Is tolerant with me even when I'm not meeting his/her expectations.
5. Doesn't butt into my conversations.
6. Doesn't make me nervous.
7. Says I just want attention when I say I'm not well.
8. Makes me feel guilty for not meeting his/her expectations.
9. Isn't overprotective with me.
10. Loses his/her temper when I'm not feeling well.
11. Is sympathetic towards me when I'm ill or upset.
12. Can see my point of view.
13. Is always interfering.
14. Doesn't panic when things start going wrong.
15. Encourages me to seek outside help when I'm not feeling well.
16. Doesn't feel that I'm causing him/her a lot of trouble.
17. Doesn't insist on doing things with me.
18. Can't think straight when things go wrong.
19. Doesn't help me when I'm upset or feeling unwell.
20. Puts me down if I don't live up to his/her expectations.
21. Doesn't insist on being with me all the time.
22. Blames me for things not going well.
23. Makes me feel valuable as a person.
24. Can't stand it when I'm upset.
25. Leaves me feeling overwhelmed.
26. Doesn't know how to handle my feelings when I'm not feeling well.
27. Says I cause my troubles to occur in order to get back at him/her.
28. Understands my limitations.
29. Often checks up on me to see what I'm doing.
30. Is able to be in control in stressful situations.
31. Tries to make me feel better when I'm upset or ill.
32. Is realistic about what I can and cannot do.
33. Is always nosing into my business.
34. Hears me out.
35. Says it's not OK to seek professional help.
36. Gets angry with me when things don't go right.

THE LEE SCALE (Client Version): ANSWER SHEET

YOUR NAME: _____ **AGE:** ____ **SEX: (circle one)** Male Female **DATE:** _____

MARITAL STATUS: (circle one)
Single Married/Common-Law Separated Divorced Widowed

Indicate who has been the most influential person in your life over the past three months: (circle one)
Mother Father Brother Sister Spouse
Other relative (e.g., Aunt, Grandfather) Friend
Other (Please Specify) _____

Have you been living with your influential person during the past three months? (circle one) Yes No

How many waking hours on a typical weekday have you been spending with your influential person during the past three months? ___ hours per week day

How many waking hours on a typical weekend have you been spending with your influential person during the past three months? ___ hours per weekend

Instructions for each item:
Circle the "T" box if you feel the item is TRUE
Circle the "F" box if you feel the item is FALSE

1	T	F	16	T	F	31	T	F	46	T	F				
2	T	F	17	T	F	32	T	F	47	T	F				
3	T	F	18	T	F	33	T	F	48	T	F				
4	T	F	19	T	F	34	T	F	49	T	F				
5	T	F	20	T	F	35	T	F	50	T	F				
6	T	F	21	T	F	36	T	F	51	T	F				
7	T	F	22	T	F	37	T	F	52	T	F				
8	T	F	23	T	F	38	T	F	53	T	F				
9	T	F	24	T	F	39	T	F	54	T	F				
10	T	F	25	T	F	40	T	F	55	T	F				
11	T	F	26	T	F	41	T	F	56	T	F				
12	T	F	27	T	F	42	T	F	57	T	F				
13	T	F	28	T	F	43	T	F	58	T	F				
14	T	F	29	T	F	44	T	F	59	T	F				
15	T	F	30	T	F	45	T	F	60	T	F				

APPENDIX 3 : Proof of Ethical Approval

Isle of Wight, Portsmouth & SE Hants Local Research Ethics Committee

Finchdean House
Milton Road
Portsmouth
PO3 6DP

Miss L Hodder
7 Ashby Road
Sholing
Southampton SO19 1DR

Direct Line: 023 9283 5139
Fax: 023 9285 5312
E-mail: anna.noble@portsha.swest.nhs.uk

19 September 2002

Dear Miss Hodder

REC Prop No: **07/02/1378**
Title: **An exploration of family communication style and its impact upon post traumatic stress disorder**

This is to inform you that the Chair of the Local Research Ethics Committee has approved the above study. Approval for the study is only granted until the end of **December 2003**. If your study continues after this date further Ethics Committee approval will be required.

The following documents were reviewed:

Protocol	not dated
Patient consent form	version 2, not dated
Patient/relative information letter	version 2, not dated
Relative consent form	version 2, not dated
University of Southampton ethical approval letter	dated 13/06/02
CV	Lindsay Hodder

The Ethics Committee will require a copy of the completed study for its records, you are therefore requested to submit a copy of the completed study to the address above.

The Committee must be informed of any untoward or adverse events which occur during the course of the study.

Please inform the Committee if the study is withdrawn, or does not take place.

The Ethics Committee must also be informed of, and approve, any proposed amendments to your initial application.

Please note it is the policy of the Committee NOT to deal direct with sponsoring companies. All correspondence (including telephone enquiries) MUST be from the first named researcher. Enquiries from other sources will be refused.

cont/...

Ethics Committee approval means that the proposal is ethically sound. It does not mean approval of resources, access to data or any other requirement relating to the project. These must be agreed with the organisation where the research / project is to take place.

If you have any further questions please do not hesitate to contact me quoting the Research Ethics Committee Proposal Number given above.

Yours sincerely

Anna Noble
Administrator to the Research Ethics Committee

cc: Clair Wright, Southampton & SW Hants LREC

NB: The Committee endorses the Royal College of Physicians Report on 'Fraud & Misconduct in Medical Research Practice 1991'. This states that all original data (eg questionnaires, lab books, hard copies of any computer data) are kept for a minimum of ten years in a retrievable form. If storage is to be outside either Portsmouth Hospitals or Portsmouth HealthCare NHS Trusts' premises, the Committee must be informed of the site of storage. It is a condition of any approval that such storage occurs.

University of Southampton

Department of Psychology

University of Southampton
Highfield
Southampton
SO17 1BJ
United Kingdom

Telephone +44 (0)23 8059 5000
Fax +44 (0)23 8059 4597
Email

13 June 2002

Lindsay Hodder
Department of Clinical Psychology
University of Southampton
Highfield, Southampton
SO17 1BJ

Dear Lindsay,

Re: An exploration of family communication style and its impact upon Post Traumatic Stress Disorder

The above titled application - which was recently submitted to the departmental ethics committee, has now been given approval.

Should you require any further information, please do not hesitate in contacting me on 023 8059 3995. Please quote reference CLIN/2002/21.

Yours sincerely,

KMSmith

Kathryn Smith
Ethical Secretary

cc. Janet Turner

SOUTHAMPTON & SOUTH WEST HANTS
LOCAL RESEARCH ETHICS COMMITTEE

Chairman: Dr Audrey Kermode

Manager: Mrs Clair Wright
Trust Management Offices
Mailpoint 18
Southampton General Hospital
Tremona Road
Southampton
SO16 6YD

Ref: CPW/

Tel: (023) 8079 4912
Fax: (023) 8079 8678

18 October 2002

Miss L Hodder
7 Ashby Road
Sholing
Southampton SO19 1DR

Dear Miss Hodder

Submission No: 284/02/t – An exploration of family communication style and its impact upon post traumatic stres disorder.

Following the conditional approval and in response to Professor Kingdon's letter dated 24 September 2002, I am pleased to confirm **full approval** having responded satisfactorily to the committees concerns. Our Vice-Chairman, Mr M Griffiths discussed the issues raised by the committee concerning the recruitment method with Dr J Eldridge, Chairman of the Portsmouth & IOW LREC and they are agreed that you intend to use an "Opt-in" method.

The following documents were re-considered:

- **Letter from Professor Kingdon dated 24 September.**

This approval was granted under Chairman's action by the Vice Chairman Mr M Griffiths, and will be recorded by the Committee at their meeting in November.

This committee is fully compliant with the International Committee on Harmonisation/Good Clinical Practice (ICH) Guidelines for the Conduct of Trials involving the participation of human subjects as they relate to the responsibilities, composition, function, operations and records of an independent Ethics Committee/Independent Review Board. To this end it undertakes to adhere as far as is consistent with its Constitution, to the relevant clauses of the ICH Harmonised Tripartite Guideline for Good Clinical Practice, adopted by the Commission of the European Union on 17 January 1997.

Yours sincerely

Mrs Clair Wright
Research Ethics Manager

Hampshire and Isle of Wight NHS
Strategic Health Authority

Ref: CPW/HH

SOUTHAMPTON & SOUTH WEST HAMPSHIRE
LOCAL RESEARCH ETHICS COMMITTEES
1ST Floor, Regents Park Surgery
Park Street, Shirley
Southampton
SO16 4 RJ

Tel: 023 8036 2466
023 8036 3462
Fax: 023 8036 4110

General Enquiries: temp1@gp-j82203.nhs.uk
clair.wright@gp-j82203.nhs.uk

31 October 2002

Miss Lindsay Hodder
7 Ashby Road
Sholing
Southampton
SO19 1DR

Dear Miss Hodder,

Submission No: 284/02/t – An exploration of family communication style and its impact upon Post Traumatic Stress Disorder.

In response your letters dated 9th October 2002, I am pleased to confirm ethical approval for the protocol amendment.

The following documents were reviewed:

- Letters from Miss Hodder dated 9th October 2002
- Draft Cover Letter signed by Ms Sue Ross, Consultant Clinical Psychologist on behalf of the Clinical Psychology Services, RSH

This approval has been granted under Chairman's action by the Chairman Dr David Briggs and will be recorded at the committee meeting in November.

Yours sincerely

Mrs Clair Wright
Research Ethics Manager

Isle of Wight, Portsmouth & SE Hants Local Research Ethics Committee

Finchdean House
Milton Road
Portsmouth
PO3 6DP

Miss L Hodder
7 Ashby Road
Sholing
Southampton SO19 1DR

Direct Line: 023 9283 5139
Fax: 023 9273 5073

13 February 2003

Dear Miss Hodder

REC Prop No: **07/02/1378**
Title: **An exploration of family communication style and its impact upon post traumatic stress disorder**

Thank you for your email of 6 February.

The Chair, acting under delegated authority, is happy to extend approval to include recruitment of patients from the Isle of Wight. You are reminded that the R&D department of the Trusts involved will need to be notified and give approval.

If I can be of any further assistance, please do not hesitate to contact me, quoting the Research Ethics Committee Proposal Number given above.

Yours sincerely

Anna Noble
Administrator to the Research Ethics Committee

E-mail: anna.noble@portsha.swest.nhs.uk

APPENDIX 4: Information letter for participants

Dear

As you know, a referral has been made on your behalf to the ..... Psychology service by..... I am aware that you have been currently placed on a waiting list for treatment.

My name is..... And I am currently working towards my Doctorate in Clinical Psychology. As part of this training, I am engaged in some research looking at how families influence the way in which we think about and deal with traumatic incident. I am writing to ask if you would be willing to take part in this research.

Why should I get involved?

There are two main reasons for taking part in this research.

Firstly, this kind of research is essential if we are to keep on improving treatments for psychological disorders. Many other willing volunteers have contributed to developing the effective treatment that you will receive.

Secondly, and perhaps most relevant to you, this session will not only be used to gather information. Time will be allowed (a minimum of half an hour) to answer and questions you have regarding Post Traumatic Stress Disorder (PTSD) or the therapy you are likely to receive. I will also be able to point you in the direction of useful self-help material and points of contact that may be useful to you whilst you are waiting for therapy to start.

If you do not wish to participate in the research but wish to find out more about PTSD, you are welcome to attend for half the session.

What would it involve?

Taking part would involve attending one session at . During this session, you will be asked to complete several short questionnaires which ask about the symptoms you are currently experiencing, the kind of thoughts you are having about the traumatic incident which you unfortunately experienced and the way in which your family communicates with each other. You will be asked if we can ask your relatives a few questions. You can choose to allow contact with your relatives or not – this will not affect you contribution to the study and will not affect your later treatment. If you agree to allow me to contact your relatives, no information about yourself will be shared with your relatives (and equally what your relatives tell me will be held confidentially).

Who will see this information about me?

A copy of the questionnaires will be given to the therapist with whom you will be working at a later date. This information will help them to better understand the difficulties you are experiencing. These copies will be kept with your medical notes, which are completely confidential.

Only this copy will contain any details that would allow you to be identified. The questionnaires will all be coded anonymously – names, addresses and dates of birth will *not* be included. Therefore nobody, excluding myself, will know who the questionnaires were completed by. No personal details will be included in the final research. Therefore the answers you give will be kept in the strictest of confidence.

What will happen to this information?

The information from participants will be collated and written up into a research paper (in summer 2003). Copies of that research paper will be available and you will be asked if you would like to be sent a copy if you agree to participate. I will be available to discuss the findings of the research if you have any questions upon seeing the research paper.

What do I have to do now?

Please complete the attached form and return it in the stamped addressed envelope enclosed. If you indicate that you are willing to take part in this research, I will send you an appointment on receiving the form.

If you have any questions or concerns about the research or any difficulties attending the appointment offered, please don't hesitate to contact me on
I would like to take the opportunity to thank you for reading this rather lengthy letter and for considering taking part in this research.

I look forward to hearing from you and I am sorry to trouble you once again.

Yours truly,

Trainee Clinical Psychologist

APPENDIX 5 : Consent form for participants

An exploration of family communication style and its impact upon Post Traumatic Stress Disorder

Consent form

I would be willing to take part in this study and I fully understand that :

*this will involve completing several questionnaires
*One copy of my questionnaires will be kept with my medical notes for my therapists use. All other copies will be anonymised and kept confidentially.
*choosing not to take part in this study will not affect my care/service provision in any way
*I have the right to pull out of the study at any point I choose
*I am willing for my relatives to be approached Yes / No
(please delete as appropriate)

Signed---------- Date----------

Name----------

Researcher's signature----------

Researcher's name----------

Convenient times for appointment would be :.....

My Contact number is .....

I am happy to be contacted by telephone : Yes / No*

*Please delete as appropriate

APPENDIX 6 : Information letter for relatives

Dear

My name is and I am currently working towards my Doctorate in Clinical Psychology. As part of this training, I am engaged in some research looking at how families influence the way in which we think about and deal with traumatic incidents. Your..... has agreed to take part in this research and there fore I am writing to ask if you would also be willing to take part in this study.

Why should I get involved?

This kind of research is essential if we are to keep on improving treatments for psychological disorders. It is thanks to many other willing volunteers that the therapy which your relative will receive is so effective. .....has already agreed to take part in the research. If you decide not to get involved, ...'s contribution will still be useful to us. However, in order to make this research a success we do need some relatives to agree to take part.

What would it involve?

Your part in the research would simply involve answering a telephone call, lasting approximately 10 minutes, during which we would talk generally about your relationship with..... The phone conversation will be taped in order to allow me to review our conversation at a later date.

Who will see this information?

This information will be kept confidential and will only be seen by myself and my supervisor (who will also review the conversation). Exact details of the content of the phone conversation will not be utilized in the final research – only the general gist of the conversation. Therefore all personal details and comments will be removed before submission for publication. Your name and the name of your relative will not be used anywhere in the research or shown to anyone other than myself and all details which could potentially identify either of you will be removed.

Yours relative will not be given access to the conversation. All details will remain confidential between you, myself, and my supervisor (who needs access to the conversations to check the quality of my work).

What will happen to this information?

The information from participants will be collated and written up into a research paper (in summer 2003). Copies of that research paper will be available and you will be asked if you would like to be sent a copy if you agree to participate. I will be available to discuss the findings of the research if you have any questions upon seeing the research paper.

What do I have to do now?

Please complete the attached form and return it in the stamped addressed envelope enclosed. If you indicate that you are willing to take part in this research, I will contact you by phone at a time which you have indicated to be convenient.

APPENDIX 7 : Consent form for relatives

An exploration of family communication style and its impact upon Post Traumatic Stress Disorder

Consent form for relatives

I would be willing to take part in this study and I fully understand that :

*this will involve holding a 10 minute taped phone conversation with the researcher
* All copies of this telephone conversation will be kept confidentially and my name and details will be removed. Only the researcher and her supervisor will have access to the conversation. My relative will not have access to this information.
*choosing not to take part in this study will not affect the care/service provision received by my relative in any way
*I have the right to pull out of the study at any point I choose

Signed---------- Date----------

Name----------

Researcher's signature----------

Researcher's name----------