Using a mixed methods sequential explanatory approach to identify the roles of social and cognitive factors in the development and maintenance of cancer-related PTSD in cancer survivors.

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Abstract

Aims: Identify the mean prevalence of CR-PTSD, factors related to trauma, and the development and maintenance of PTSD, in cancer survivors. **Background:** Systematic reviews reveal that CR-PTSD is uncommon, and it is unclear a) what makes this experience traumatic, and b) what factors are implicated in the development, and maintenance of PTSD in this population. **Methods:** A mixed-methods sequential explanatory approach was used. Phase 1 consisted of three studies: a) random-effects meta-analysis of PTSD prevalence statistics and moderating factors in cancer survivors (k=25, n=4189); b) a cross-sectional analysis of PTSD and contributing factors in a PTSD Clinic for cancer survivors (n=60); and c) a prospective analysis of the role of emotion schemas and processing styles and how they predict adaptation to stress in a sample of students (n=24). Phase 2 was conducted to find follow-up explanations for Phase 1 results. Study 4 (Phase 2) consisted of two clinical case studies from the PTSD Clinic – one with adjustment disorder, and the other with severe chronic CR-PTSD. **Results:** Study 1 revealed that PTSD prevalence in breast cancer survivors was 5.8% (95% CI=3.3-10%), and that there were no significant study-level moderators that predicted differences in prevalence. Similar results were found for Study 2, although when adjusted for age, those with CR-PTSD suffered from more impoverished emotional experiences than those without CR-PTSD. These differences were rendered non-significant when depression symptoms were added as a covariate. Study 3 revealed that increases in anxiety during a stressor were best predicted by emotion schemas related to the lack of comprehensibility of emotions. Findings from Study 4 suggested that aspects of the cancer experience was very traumatic for both patients, but that the course/development of disorder was influenced by the social-cognitive processes involving the interaction of the patient's emotion schemas and coping strategies, with the quality of their support system. **Conclusions:** Cancer can be traumatic under certain conditions, and PTSD is uncommon in cancer survivors, but clinical samples of cancer survivors with and without PTSD suggest that CR-PTSD is characterised by severe problems experiencing, linking, and labelling emotions. Preliminary evidence from case studies reveal that the combination of a) an appraisal of the cancer as traumatic, b) an invalidating social network, *and* c) emotionally avoidant coping styles throughout the cancer treatment, may predispose traumatised cancer survivors to PTSD.

Preface

This research was undertaken to begin the process of running a randomized clinical trial (RCT) for an experimental trauma-focused therapeutic approach with adjunctive emotional processing interventions (Baker, 2011). This was to be conducted in a sample of breast cancer survivors with PTSD related to the experience of cancer diagnosis and treatment. As such, this work serves to investigate many of the hidden suppositions behind PTSD and trauma in this population.

Before I started this research journey, I was working as a psychological wellbeing practitioner (PWP) in NHS Adult Primary Care Mental Health. I successfully treated mild-moderate anxiety and depression, and also assessed for depression and a range of anxiety disorders (including generalized anxiety, panic, social phobia, simple phobia, OCD, and PTSD). During this period, I was also committed to research on the role of emotional processing in anxiety disorders (Baker, et al., 2012). I was working voluntarily with a consultant clinical psychologist and research assistant to develop and publish a trauma-focused treatment protocol for PTSD that had the potential to address engagement issues in treatment non-responders – otherwise known as emotional processing therapy for PTSD (Baker, Gale, Abbey, & Thomas, 2013). I learned from experience that one of the privileges of working clinically with vulnerable people was becoming a partner in their journey of achieving physical and emotional wellbeing. As caring professionals, we achieve this through the holistic assessment of individual need, and based on subsequent formulations, prescribe evidence-based interventions to suit those needs. As such, caring professionals base their practice on the best-available evidence. In the National Health Service (NHS), the best evidence-based practice is determined in the guidelines written by The National Institute for Health and Care Excellence (NICE). The best evidence for PTSD (the focus of this thesis) shows that trauma-focused therapies such as Prolonged Exposure, Trauma-Focused Cognitive-Behavioural Therapy (TF-CBT), and Eye-Movement Desensitisation and Re-processing (EMDR) are equally efficacious and effective treatments that can aid the processing of trauma memories and engender recovery (NICE, 2005).

Following this, I was invited to apply for a PhD to develop an RCT that would provide evidence for the efficacy of our new approach (Baker et al., 2013) in a cancer sample with PTSD. Specifically, these patients were cancer *survivors* (those who completed treatment and were cancer-free; DoH, 2007), who presented with symptoms of PTSD anchored to aspects of the cancer experience. In order to prepare for a future RCT, I conducted a systematic review of the literature, which revealed a range of practical, conceptual, and diagnostic issues that needed to be addressed to justify a trial in this population.

The first issue was whether the cancer experience is traumatic. Medical illnesses can be endorsed as traumatic events according to the Diagnostic and Statistical Manual 4th Edition (DSM-IV; APA, 1994), and systematic reviews have highlighted the traumatic nature of a range of medical problems (Tedstone & Tarier, 2003). However, whether cancer itself is generally traumatic has been open for debate since the publication of DSM-IV, and was recently declassified as such under DSM-5, although medical events that are sudden and catastrophic, would still meet trauma criteria (APA, 2013). Research-to-date has not accounted for this, but rather rests on the supposition that cancer is a traumatic event (PTSD Criterion A, DSM-IV; APA, 1994). But if cancer is not *typically* traumatic, then PTSD cannot be diagnosed.

The second issue is one of diagnostic accuracy. Just because an individual survives a traumatic event does not mean they will actually develop PTSD. If one supposes that cancer is indeed a Criterion A event,

there remains the issue that many side-effects of cancer treatment mimic PTSD hyperarousal symptoms, and thatcognitive intrusions used to endorse re-experiencing criteria actually reflect future worries rather than intrusive memories. This could result in misdiagnosis, which would then introduce systematic error into a clinical trial.

The third issue is one of application. Supposing that a) cancer can and does meet PTSD Criterion A; and b) that cancer-related PTSD (CR-PTSD) can be reliably screened and diagnosed in cancer survivors, the issue remains that PTSD is extremely uncommon in very common cancers (Thompson, Eccleston, & Hickish, 2011). If the prevalence of CR-PTSD is low, recruiting for a suitably-powered clinical trial would be difficult for three reasons: 1) A minimum sample of n=40 CR-PTSD patients (20 treatment, 20 control) would require screening hundreds patients based on the range of prevalence statistics elicited by structured clinical interviews; 2) the cancer literature (chapter 1.5) is also characterised by samples skewed towards less severe cancer, and low-medium response rates, warranting an initial sample greater than suggested by prevalence statistics and 3) given the lack of clarity in the literature about whether PTSD is a suitable diagnosis for a traumatised cancer survivor, general screening methods may not be suitable, but excluded in favour of structured clinical interviews by oncology specialists and consultant clinical psychologists. The combination of these three points posits an unfeasible, expensive, and time-consuming effort for oncology services.

This PhD represents an effort to address the limiting issues, but one must first consider the chief end of any research endeavour in this field: to improve assessment and interventions as to improve the quality of life of service users. In the context of cancer survivors, it is important to improve the understanding of how their PTSD develops. For example, what parts of the cancer experience are endorsed as traumatic?; what factors are implicated in stalling the course of natural adjustment?; and given the issues surrounding detection of CR-PTSD, what patient factors distinguish CR-PTSD from the subsyndromal symptoms and adjustment disorders that characterise many cancer survivors? (Kangas, 2013). Answering these questions will enable oncology specialists to better screen for CR-PTSD in survivors of cancer and its treatment.

In order to achieve these research aims, I have conducted an extensive literature review. This first section (chapter 1.1.) examines the epidemiology of PTSD, and highlights epidemiological trends and broad factors implicated in the likelihood of developing PTSD after a traumatic event. The second section (chapter 1.2.) examines and critically evaluates the risk factor research for PTSD, identifying weaknesses and methodological limitations of the research, highlighting areas which need additional focus. The third section (chapter 1.3.) moves to the clinical theories that inform our understanding of PTSD, and how it is treated successfully. This section also critically evaluates the theories in the context of their ability to explain and predict the PTSD syndrome and its major course diversions. As the reader will notice, the clinical theories are less able to predict the role of factors that are inextricably linked to the issues in the PTSD risk factor research. The fourth section (chapter 1.4.) synthesises the findings from chapter 1.2 and 1.3., and investigates their contribution to the maintenance of anxiety disorder and PTSD, and how that also factors into treatment resistance. Using the findings from chapters 1.1 – 1.4., the fifth and final section (chapter 1.5.) is a review of the literature that focuses on the prevalence of CR-PTSD, and the factors implicated in the severity of symptoms. The literature is evaluated in the context of what is already known about PTSD and the contribution of heterogeneous study methods, samples, and assessment measures to the lack of clarity over CR-PTSD.

This thesis employs a mixed-methods, sequential explanatory approach to achieve the research aims, and consists of four studies, of which three are quantitative (Studies 1-3), and one a series of qualitative case studies (Study 4). The quantitative phase seeks to identify 1) the general prevalence of CR-PTSD in cancer populations and broad factors that moderate the prevalence of PTSD; 2) the factors that distinguish CR-PTSD from non PTSD samples, and 3) whether these variables can be used to predict levels of distress in response to an external demand. The qualitative phase seeks to provide follow-up explanations for why the variables in the quantitative phase are implicated in the differential diagnosis of CR-PTSD and subsyndromal adjustment responses.

Though the superlative aim of this thesis is to improve the detection and differential diagnosis of CR-PTSD, there are individual aspects of this thesis that will provide independent contributions to knowledge. Study 1 is the first of its kind. There has never been a published empirical synthesis of prevalence data for CR-PTSD – this study will inform oncology specialists of how much CR-PTSD they can expect. Also, Study 2 is arguably the first of its kind to examine CR-PTSD solely in a group of post-treatment and disease-free cancer survivors, and explore clinical features that distinguish CR-PTSD from its related, but nevertheless sub-threshold, diagnoses. Although this thesis does have clinical and service-based implications for the cancer population, it also provides an excellent opportunity to examine the factors that contribute to chronic PTSD after protracted traumatic stressors. Investigating these factors that are implicated in chronic PTSD courses may inspire research questions for other populations characterised by protracted traumas such as survivors of domestic violence, and repatriated war veterans.

2.1. The Epidemiology of PTSD

Epidemiology is the study of the factors related to the occurrence rates and distribution of disease, in specific populations. The rates of disease are measured in terms of incidence and/or prevalence. Incidence is the number of new cases of a specific condition in a specific sample over a specified time, whereas prevalence is the proportion of a population presenting with a specific condition at a particular time point. Prevalence therefore includes incidence (new cases), as well as people who have had the condition long before the epidemiological survey (old, but active cases). Epidemiological surveys provide essential data on disease trends, both historic and current. In this sense, the rates of disease are seen to rise and fall in relation to population characteristics, and have clinical and service-based implications for the detection and treatment of a range of medical or psychological problems (Saracci, 2010).

The epidemiology of trauma and mental disorder across populations has been an integral part of the development of the DSM diagnostic criteria for PTSD (APA, 1994; 2000; Table 1). It provides data on the prevalence of traumatic experiences in the population (both general and specific), and the lifetime prevalence of the PTSD syndrome - both in the population in general, and in those who have been traumatized. Data collected from whole populations and large representative samples provides preliminary data on socio-demographics, interpersonal, and systemic variables that may be predictive factors in PTSD development and severity (Norris & Slone, 2007).

Generally, the role of trauma intensity and exposure is indexed by an increase in PTSD prevalence rates increasing with trauma intensity. Conversely, vulnerability factors predicting the PTSD syndrome are determined by an over-representation of socio-demographic or intra-personal moderator variables in those who develop PTSD compared to those who do not (McFarlane & De Girolamo, 2007). However, studies of PTSD epidemiology vary greatly in terms of the specific diagnostic criteria, and the measures used, be they screening tools that measure symptom severity, or assessment measures that measure all criteria in question. For this reason, we will examine PTSD prevalence rates for DSM-III and DSM-IV separately in each section.

1.1.1. Trauma Exposure in the General Population

The earliest epidemiological investigations of trauma exposure were conducted in the US population using DSM-III PTSD criteria. A seminal survey by Breslau, Davis, Andreski, & Peterson (1991) found that in a US urban sample of young adults (n = 1007) 39.1% were exposed to at least one trauma, with 67.3% of the exposed group experiencing one, 23.3% experiencing two, and 9.4% three, traumas over their lifetime. The high prevalence of traumatic events was also reflected in Norris' (1992) study, where in a US adult sample (n = 1000), 69% had experienced traumatic events, with 4.4% experiencing sexual assault, and 30.2% experiencing a tragic death of a loved one. The experience of tragic accidents was also common. Similarly, Resnick Kilpatrick, Dansky, Saunders, & Best (1993), in their sample of US adult women (n = 4008), found a lifetime trauma exposure prevalence of 68%. Victimisation from criminal activity was prevalent (35.6%), followed by non-crime related disasters (33%), sexual assault (14.3%) completed rape (12.7%), homicide of a family member (13.4%), and physical assault (10.3%). Though the prevalence of trauma fluctuated depending on the sample, there was an emerging trend where some

traumas were more widely experienced than others in the general population. However, subsequent surveys revealed additional gender trends. Kessler, Sonnega, Bromet, Hughes, & Nelson (1995), in the National Co-morbidity Survey (NCS; *n* = 5877) found that 60.7% of men and 51.2% of women had experienced at least one DSM-III criterion A traumatic event. This is similar to the results of Resnick et al. (1993), who found that the majority of their sample has experienced A1 events. Furthermore, Kessler et al., (1995) replicated the findings that the majority those who experienced one trauma, actually experienced more than one. The NCS also revealed that the most commonly reported A1 event was the witnessing of death or serious injury (35.6% men; 14.5% women), followed by the experiencing of disasters (18.9% men; 15.2% women) and also life-threatening accidents (25% men; 13.8 women). What was evident from these epidemiological surveys was that experiencing one or more traumatic events was common, and was contrary to the supposition that a traumatic event was outside the range of normal experience (APA, 1980).

With the publication of the DSM-IV (APA, 1994), PTSD Criterion A was spilt into two components: A1 (the traumatic event), and A2 (the individual's emotional reaction). Criterion A1 was expanded to include distressing experiences previously omitted from the DSM-III (APA, 1980), such as life-threatening illness. Breslau, Kessler, Chilcoat, Schultz, Davis, & Andreski (1998), in their US sample from the Detroit Area Survey (*n* = 2181), revealed that 89.6% of the sample experienced DSM-IV criterion A1 events, Those who experienced one A1 event also experienced a mean of five A1 events. The most prevalent event experienced in this sample was the sudden unexpected death of a loved one (60%), replicating similar findings from the NCS (Kessler et al., 1995). In terms of gender differences, Breslau et al., (1998) demonstrate more women than men reported sexual assault and rape, whereas men reported physical assaults and accidents. Of special interest is that the use of DSM-V Criterion A1 did increase the prevalence of traumatic events compared to the NCS (Kessler et al., 1995), but produced similar statistics for the prevalence of traumas included in DSM-III Criterion A such as rape, natural disaster, and witnessing death or injury.

Epidemiological studies conducted in Central / South America, Europe / UK, and Australia, revealed variable rates of trauma. Zlotnick, Johnson, & Kohn (2006), in their Chilean sample (*n* = 2345) found a lifetime trauma prevalence of 39.7% (46.7% men, 33.2%, women). Much more severe results were found in the Mexican general population (*n* = 2509) by Norris et al. (2003), with a 76% lifetime trauma prevalence (83% men, 71% women). Perkonigg, Kessler, Storz, & Wittchen (2000), in their sample from the Munich population (*n* = 3021), found a lifetime trauma prevalence of 21.4% (26% men; 17.7% women), with 17% of sample meeting criterion A1 and A2. The opposite was found by Frans, Rimmö, Åberg, & Fredrikson (2005), who using their Swedish sample (*n* = 1824), found a lifetime trauma prevalence of 80%. Similarly, Creamer, Burgess, & McFarlane (2001) in their sample from the Australian National Survey of Mental Health and Well-being (*n* = 10641) yielded a lifetime prevalence of at least one traumatic event of 64.6% for men and 49.5% for women. Witnessing death or injury was the most commonly reported traumatic event (37.8% men; 16.1% women), followed by life-threatening accidents (28.3% men; 13.6% women) and natural disasters (19.9% men; 12.7% women). Men were more likely

Table 1: DSM-IV-TR Diagnostic Criteria for PTSD (APA, 2000)

Component	Criterion	Explanation
Trauma	A	First, the presenting individual must have experienced (or witnessed) an event that threatens their (or another's) psychological and/or physical integrity (Criterion A1), and they themselves must react with helplessness or horror (Criterion A2). Traumatic experiences that can generate this response can range from (but are not necessarily limited to) prolonged combat exposure, natural disasters, and physical/sexual assault, to receiving a diagnosis of a life-threatening illness.
Syndrome	B	This refers to the automatic triggering of intrusive recollections of the traumatic event after it has passed (B1), or recurrent nightmares either of the event or are thematically related to the event (B2). When the trauma is more severe, presenting individuals may dissociate for seconds, hours, or days, when the whole or certain parts of the trauma memory are re-experienced and replayed in the present (B3). Emotional distress (B4) and physiological arousal (B5) present at the time of the trauma are often re-experienced at this time. At least one of these re-experiencing symptoms must be endorsed for the duration of the disturbance, and not present before the traumatic event.
	C	This refers to systematic efforts to evade reminders of the trauma, be they internal, such as thoughts and feelings (C1); or external, such as conversations (C1) activities, situations, or people (C2). Avoidance might also include selective amnesia for 'hotspots' in the trauma memory (C3), or a diminished emotional responsiveness, which can manifest in a reduced interest and participation in hobbies that were once enjoyed (C4), or of feeling distant from others (C5), or an impairment in feeling loving emotions (C6). The sufferer may also feel like their life is cut short, in terms of their lifespan, or their chances of having fulfilling life experiences such a marriage, children, and a career (C7). At least three of these avoidance symptoms must be endorsed for the duration of the disturbance, and not present before the traumatic event.
	D	This refers to increased physiological reactivity such as difficulty sleeping (D1), increased irritability and anger episodes (D2), decreased concentration and subsequent impairment in completing tasks (D3), hyper-vigilance due to perception of threat (D4), and an exaggerated startle response (D5), and which was not present before the traumatic event.
Course	E	The PTSD syndrome (B-D) will be present within a month post-trauma as a normal adjustment response (see acute stress disorder, APA, 2000), and is not necessarily pathological. However, if the full symptom profile is present for longer than one-month after the traumatic event has passed, then it is considered to have continued into a post-traumatic stress disorder. Finally, the PTSD syndrome can follow one of three courses: 1) *acute*, where the duration of symptoms, assuming the endorsement of Criterion E, is less than three months; and 2) *chronic*, where the symptom profile has been present for three months or more. Finally, it must be specified whether or not the symptoms have a *delayed onset*, that is, the PTSD syndrome begins to appear at least six months after the traumatic event.
Functioning	F	This refers to the effect of the post-traumatic stress syndrome on the sufferer's personal functioning. In order for PTSD to be diagnosed, the combination of re-experiencing, avoidance, and hyper-arousal symptoms must be severe enough to impair the sufferer's ability to engage meaningfully in their social, occupational, and relational arenas, or cause severe and clinically significant distress.

than women to experience these relatively non-interpersonal traumas, whereas women more likely to report sexual assault and rape (3.8% men; 12.9% women). Multiple traumatic events were far more common in this sample than not for those who experienced at least one traumatic event. This replicates the findings from earlier studies that the experiencing of multiple traumatic events over a lifetime is more common than not (Breslau et al. 1991; Breslau et al., 1998; Kessler et al., 1995).

In the UK, McManus Meltzer, Brugha, Bebbington, & Jenkins (2009) conducted the Adult Psychiatric Morbidity Survey (APMS; *n* = 7461), and found a lifetime trauma prevalence of 42% (44.1% men, 30.1% women) in their sample. This is lower than the estimates for several US and Swedish samples, although comparable to that of the Australian sample, and also to the study conducted by Ferry et al (2008) in Northern Ireland (*n* = 1095), who found a lifetime trauma prevalence of 66% (men 68.8%, women, 64.3%). This is twice the prevalence of the rest of the UK population, but given the age range of the sample (18-93), this may be explained by the sample having been exposed to IRA bombings. All the previous studies were conducted on the general population, and revealed that men reported more traumatic events than women.

However, of special interest is the study conducted by Liebschutz, Saitz, Brower, Keane, Lloyd-Travaglini, Averbuch, & Samet (2007). They conducted an epidemiological study on a US urban primary care sample (*n* = 509). They found a trauma prevalence of 79%, of which 65% had been exposed to more than one event. It is revealed in their sample that multiple exposures to traumatic events was true for a significant majority of the sample. Of particular note was that 46% of the traumatised sample experienced the witnessing of death or serious injury. This reflected the findings from Breslau et al., (1991), Kessler et al., (1995), and Creamer et al., (2001) that lower magnitude stressors (such as indirect exposure to death/injury) were of higher frequency. Breslau & Kessler (2001) used the DAS data from their earlier study (Breslau et al., 1998) and applied a larger list of A1 events (DSM-IV; APA, 2000) and found that 77% met Criterion A2, with 33% meeting A2 for military combat, to 93% for rape, and 94% for life-threatening illnesses, therefore increasing the prevalence of traumatogenic events that can be used to diagnose PTSD by 59%.

Overall, the prevalence statistics of trauma exposure were heterogeneous across studies, but this can be explained by sampling different populations (US / European / Australasian), and also by variations in screening and assessment instruments, as partially determined by different diagnostic criteria. There were emerging trends that the experience of traumatic events (be they accidents, disasters, victimisation from criminal activity, or vicarious traumatisation from witnessing or hearing about death or injury) were not just common, but statistically probable occurrences over the lifespan, and that the experiencing of multiple traumas given an initial exposure was just as likely. However, this does not necessarily mean that those who experience these events suffer chronic psychological injuries. The experience of a traumatic event is necessary, but insufficient on its own to generate PTSD. The next section reviews the data regarding the risks of developing PTSD given the experience of general and specific traumatic events.

1.1.2. PTSD prevalence in the General Population

In one of the earliest epidemiological studies using DSM-III PTSD Criteria (APA, 1980), Helzer, Robins & McEvoy (1987) studied the general US population (*n* = 2493), and yielded a 1% lifetime prevalence for PTSD. The conditional risk of PTSD from physical assault in the civilian population was 3%, and being wounded in Vietnam was 20%. This study revealed that full PTSD according to DSM-III was very uncommon, although the conditional risk was significantly higher for those who survived wartime combat. Breslau et al., (1991) revealed different results. They found a 9.2% lifetime prevalence

of PTSD (6% for men and 10.4% for women). Of those who experienced DSM-III traumatogenic events, 23.6% developed PTSD. In terms of conditional risk [CR] following specific traumas, rape yielded the highest CR (80%) followed by threat to life (24%), the witnessing of injury or death (23.6%), assault (22.6%), receiving news of a loved one's death (21.1.%), and accident/injury (11.6%). Norris (1992) found that sexual assault lead to the highest PTSD rates (13.6%). Similarly, Resnick et al., (1993) found that out of the whole sample, 12.3% had a history of lifetime PTSD, with 4.6% current PTSD. Of those exposed to trauma, 17.9% had lifetime PTSD, and 6.7% had current PTSD. The conditional risk of PTSD differed depending on the trauma, with physical assault (38.5% lifetime, 17.8% current) and rape (32% lifetime, 12.4% current) yielding the highest risks. The lifetime prevalence of PTSD over the whole sample was comparable to the figures yielded by Breslau et al., (1991). Kessler et al., (1995) in the NCS found a lifetime PTSD prevalence of 7.8% when using strict DSM-III criterion A, and 8.4% when non-criterion events were included. As with previous studies, the experience of rape yielded one of the highest conditional risks (men, 65%, women 45.9%), followed by childhood physical abuse (48.5% women), combat (38.8%), and assault (21.3%). This seminal study demonstrated the high prevalence and chronicity of PTSD symptomatology, and was direct challenge to the DSM-III-R PTSD Criteria (APA, 1980) used in this study, as the criteria assumed the statistical abnormality, and uniformly overwhelming nature of traumatogenic events. Even more so as the methodology employed lower-bound estimates of lifetime trauma and PTSD prevalence by only recording each participant's most upsetting trauma, rather than their whole trauma history.

As seen in the previous section, the amendment of DSM-IV PTSD Criterion A (APA, 1994) substantially increased the prevalence of traumatogenic events that could be used to generate PTSD, but the following studies demonstrate that it did not significantly affected PTSD prevalence (Breslau & Kessler, 2001; Kilpatrick, Resnick, & Acierno, 2009). In the DAS, Breslau et al., (1998) found the conditional risk of PTSD to be 9.2% in their sample (13% mean, 6.2% men), with captivity and torture yielding the highest CR (53.8%), followed by rape (49%) and assaultive violence (20.9%). Perkonigg et al., (2000) found a lifetime PTSD prevalence of 0.7%, and a conditional risk of 7.8%, with rape having the highest CR at 44%, and childhood sexual abuse at 28%. Despite the very low prevalence of traumatic experiences in this population, the conditional probability of developing PTSD following trauma is consistent with the other populations studied. Similarly, Creamer et al., (2001) found 12-month prevalence of current PTSD at 1.3% which was substantially lower than Kessler et al's., (1999) calculation from the NCS (3.9%). However, the highest CRs for PTSD were associated with rape and molestation for both genders. Natural disasters and witnessing death and injury were associated with the least conditional probability for both men and women, respectively.

Similarly, McManus et al., (2009) found in their UK sample that 3.0% of adults in England screened positive for current PTSD (2.6% men; 3.3% women). The conditional risk of PTSD given trauma exposure was 8.9% for the whole population, with younger cohorts (range 16-24 years) having a much higher CR of 19.8%. Ferry et al., (2008) in their Northern Ireland sample (n = 1095), in addition to having twice the trauma exposure rate of the rest of the UK, also yielded a lifetime PTSD prevalence of 8.5% (men, 5.2%, women, 11.1%) three times that of McManus' (2009) sample. In the Swedish population, Frans et al., (2005) found a lifetime PTSD prevalence of 5.6% (7.4% men, 3.6% women) with a conditional risk of 6.9% (men 4.2%, women 9.6%). These figures are comparable to those in the NCS

(Kessler et al., 1995). Conditional risk was elevated in those with multiple traumas (mean 8.5%; men, 8.5%; women, 12.2%) and lower in those with singular traumas (mean 2.6%; men, 1.2%; women, 3.6%). The highest conditional risks (like Creamer et al., 2001) were also associated with sexual and physical assaults. This demonstrates like other studies (Breslau et al., 1998) that though trauma is common, it does not predictably lead to PTSD. But when it does, the risk of PTSD is elevated by multiple trauma, and/or acts of interpersonal violence. In the USA, Kessler, et al., (2005) replicated the National Co-morbidity Survey using DSM-IV diagnostic criteria, and found a lifetime PTSD prevalence of 6.8%. This is consistent with Frans et al's (2005) findings in their Swedish sample, and also earlier studies by Kessler et al., (1995), and close to the estimates of Breslau et al., (1991). Studies in Central and South America have revealed comparable trends. Zlotnick et al., (2006) in their Chilean sample (n = 2345) found a lifetime PTSD prevalence of 4.4% (men, 2.5%, women, 6.2%), whereas Norris et al., (2003) in their Mexican sample (n = 2509) found a prevalence of 11.2% (men, 7.2%, women, 14.5%). Though the previous studies have been conducted in the general population, the following have been conducted on specific populations.

1.1.3. PTSD in Specific Populations

Though the previous discussion focused on broad populations it is also prudent to focus on larger samples experiencing specific traumas. According to the NCS (Kessler et al., 1995), the experience of natural disasters, accidents, and witnessing Criterion A events were associated with lower conditional probabilities compared to interpersonal trauma (sexual/physical assault) and combat exposure. Some meta-analyses have revealed the contrary: that natural disaster resulted in greater conditional probability of PTSD (Rubonis & Bickman, 1991). Green, Lindy, Grace & Leonard (1992), in their study of the Buffalo Creek Dam disaster, found a 59% lifetime PTSD prevalence within the survivors, with 25% of those still meeting diagnostic criteria 14 years later. A possible reason for this is that is that the high CR of PTSD in this sample was proportional to the loss of relationship and resources. For example, it has been argued that individuals experiencing natural disasters in developing countries were at greater risk of PTSD due to the loss of already scant resources as a result of the exposure (Norris & Slone, 2007). In another study, Perrin, DiGrande, Wheeler, Thorpe, Farfel, & Brackbill (2007) found in their sample of rescue and recovery workers (n = 28962) two-three years after the World Trade Center disaster, a mean lifetime PTSD prevalence of 12.4%. The groups most at risk of PTSD where those who worked in construction, sanitation, engineering, or volunteer workers who were performing tasks inconsistent with their occupation, and did not have disaster training or experience. From these studies there is evidence to demonstrate that the level of exposure and traumatisation is related to the degree of resource loss and the emotional preparedness and resilience of individuals or people-groups when confronting distressing events.

The NCS (Kessler et al., 1995) also established that combat exposure was a comparatively uncommon trauma in the general population, with 6.4% lifetime prevalence, but with a high condition probability of PTSD of 38.8% (Kessler et al., 1995). The National Vietnam Veteran Readjustment Study (NVVRS; Kulka et al., 1990) revealed markedly higher current PTSD prevalence rates for men (15%) and for women (9%) than general civilian trauma. But this was contrasted by significantly lower rates for men

(2.5%) and for women (1.1%) who were non-theatre veterans. Dohrenwend et al. (2006), using data from this same sample, found that the PTSD prevalence among veterans increased with combat exposure (low, 10.3%; moderate, 24.9%; high, 38.6%). Similar trends were found in contemporary military samples from the UK armed forces. Fear et al., (2010) found that in military personnel deployed to Iraq and Afghanistan, the prevalence of PTSD was 4%. But, military personnel who were in combat roles were twice as likely to report probable PTSD as those who were not in combat roles. De Jong, Komproe, & Ommeren (2003) examined the prevalence of current PTSD and the conditional risk of PTSD development by level of combat exposure in different cohorts who survived four different conflicts. De Jong et al sub-grouped each cohort by whether or not they were exposed to armed conflict. They found the mean PTSD prevalence ranged from 15.8% in Ethiopia, 17.8% in Palestine, 28.4% in Cambodia, and 37.4% in Algeria. However, those who were not exposed to armed conflict yielded markedly lower PTSD rates compared to those who were exposed (3.9% *v* 19% in Ethiopia, 29% *v* 28% in Palestine, 69.% *v* 33% in Cambodia, and 13.2% *v* 39.5% in Algeria). In an earlier study by Snow, Stellman & Sommer (1988) it was found that PTSD prevalence for those exposed to comparatively moderate levels of combat was 28%, whereas those who had been exposed to much higher intensities, the prevalence was 65%. This provides evidence that the severity of combat exposure is mediated by the length of time in combat and closeness to the combat zone. These figures are also relatively consistent with Kessler et al.,s (1995) estimate of CR for combat exposure, but also introduces the concept of an exposure continuum within different categories of traumatic events. It is arguable that there is a relationship between the intensity of exposure and conditional risk of subsequent PTSD, both in terms of the amount of trauma experienced and the type of trauma experienced. Another example of this 'dose-response' relationship is revealed in US civilian samples, which have revealed that 45.9% of female rape victims (and 65% of men) develop PTSD, and is higher than all the other events experienced in the sample (Kessler et al., 1995). These figures have been replicated in other samples, but have also revealed that while a completed rape on women is associated with higher lifetime PTSD prevalence (27.8%; Kilpatrick et al., 1987), if the completed rape also resulted in physical injury, the lifetime PTSD prevalence is even higher (80%; Kilpatrick et al., 1989), a finding also replicated by Breslau et al., (1991), and Resnick et al., (1993). This suggests that some traumas are generally more toxic than others, and that the level of toxicity is worsened by the intensity of exposure. This conclusion is well supported by several clinicians and researchers (van der Kolk, Weisaeth, & van der Hart, 2007).

1.1.4. Discussion

It is evident from 20 years of epidemiological research that the prevalence rates for trauma exposure and conditional risk of PTSD differ based on population, sampling, and assessment methods, making it hard to draw conclusions from the figures (McFarlane & De Girolamo, 2007). Frequently cited confounds in measurement lie not just in the assessment tool, but the nature of the disorder itself and latent factors within the patient - for example, accurate recall influenced by the disorganised and poorly elaborated trauma memory (c.f. Brewin et al., 1996; Ehlers & Clark, 2000), as well as mood at time of assessment affecting retrospective recall. In terms of sampling, epidemiological studies need to be representative of the population of interest, and this is primarily established by a large *n* that is selected via a truly random

selection process where participants have an equal chance of selection independent of the selection of another participant. However, even when this is done, the levels of response rates affect the representativeness in that the sample becomes more self-selecting; for example those who have experienced more severe trauma and more severe PTSD may avoid selection due to protracted avoidance symptoms.

Results from epidemiological studies are highly dependent on the methods used. In terms of assessment, the use of structured interviews is normal, and reliability of the administration of these interviews is often established in the studies. However, cross-sectional designs may confound an otherwise well-conducted interview due to specific features of the disorder affecting the accuracy of retrospective reporting, and do not capture the influence of a) the effect of specific traumas on the fluctuating course of PTSD's, and the effect of this fluctuating course on the individual (McFarlane, 1997), or whether or not the person has sufficiently recovered (or not) from a prior trauma to reliably capture said variable as a moderator. The second issue is whether the measures used are validated for use in the culture or populations of interest such as specific trauma populations, different age groups, or the physically ill (Herschbach et al., 2005).

In addition to measurement characteristics, diagnostic criteria have been a substantial influence. The PTSD criteria have changed, but so have the assessment measures. This is seen in earlier studies using DSM-III criteria that were inherently restrictive on events that could qualify as traumatic (Kessler et al., 1995), yet said criteria have been empirically demonstrated to underestimate the prevalence of trauma, and over-estimate conditional risk of PTSD development (Davidson & Foa, 1991). For example, the DSM-III trauma definition was intended to capture relatively uncommon events with which a PTSD reaction might be expected, whilst simultaneously eliminating distressing but otherwise common events. Conversely, the DSM-IV definition was expanded to include these events given epidemiological data revealing the DSM-III A criterion was unrepresentative of the data (c.f. Davidson & Foa, 1991), but was also appended by a caveat stating that the experiencer had to have experienced helplessness or horror to acknowledge that intrapersonal factors related to distress affect the level of distress and traumatic toxicity of the event. Furthermore, the addition of functional impairment made the criteria more stringent, and consequently, epidemiological studies revealed a higher prevalence of trauma in the population, but lower lifetime prevalence of PTSD, and lower estimates of conditional risk (Breslau, 2001).

1.1.5. Conclusions

Overall, the literature suggests that lifetime trauma exposure in the general population is more common than not (Breslau et al., 1998; Creamer et al., 2001; Frans et al., 2005; Kessler et al., 1995; Resnick et al., 1993), although there are studies in other Western populations have not replicated this, the conditional risk of PTSD development was comparable to US populations (Perkonigg et al., 2000). This, however, may be the result of society, as the DAS (Kessler et al., 1995) was conducted in a population where criminal activity was prevalent. In clinical and military samples, there is evidence that trauma exposure is even higher than population estimates (de Jong, et al., 2003; Liebschutz et al., 2007). Also, those who have already experienced one trauma are more likely to experience subsequent traumatic events (Breslau et al., 1991; Breslau et al., 1998; Creamer et al., 2001; Kessler et al., 1995). The use of

an expanded DSM-IV A1 criterion has, in some studies, yielded an increased prevalence of low-threshold traumatic events. In terms of conditional risk, despite the DSM-IV criterion A1 being expanded to include additional stressors, there is evidence that the lifetime PTSD prevalence in the general population has not increased substantially (Breslau & Kessler, 2001; Kilpatrick, Resnick, & Acierno, 2009). There is also evidence to show that multiple traumatic events are associated with an increased risk of PTSD (Frans et al., 2005; c.f. Klein & Alexander, 2009); that some lower frequency, higher-magnitude traumas generally carry a greater conditional probability for PTSD development (e.g. rape and sexual assault, Frans et al., 2005; combat exposure, de Jong et al., 2003; Kessler et al., 1995; c.f. Klein & Alexander, 2006; 2009; Norris, 1992), and that these higher magnitude events may operate on an exposure continuum which is associated with even higher PTSD prevalence rates and thus increased conditional probability (de Jong et al., 2003; Dohrenwend et al., 2006; Kilpatrick et al., 1987; Kilpatrick et al., 1989; Norris, 1992), and conversely, that higher frequency events (e.g. witnessing death or injury, unexpected death of a loved one) carry a lower conditional risk, but can still trigger PTSD nonetheless (Breslau et al., 1998; Breslau & Kessler, 2001; Frans et al., 2005). Finally, even if trauma prevalence is low in a population, and the lifetime PTSD prevalence is low, the conditional risks of developing PTSD from traumatogenic events may still be comparable to the CR's yielded from populations where traumatic events are more common (Creamer et al., 2001; Perkonigg et al., 2000).

1.2. Risk Factors for PTSD

Evidence over the past 20 years has revealed that not only are traumatic events common, but not all those who go through traumatic events will develop PTSD (Yehuda, 2004). Of those that do, the repeatedly observed dose-response relationship between trauma exposure and PTSD is not uniformly predictive of severity (King et al., 1999). This highlights that trauma exposure is a necessary but insufficient condition for PTSD development (McNally, 2001). If the full syndrome is not a normal response, then there may be intrapersonal and environmental factors that increase the risk of PTSD.

In order to understand how PTSD develops, it is necessary to consider the assumptions that underpin, and hence define, the concept of trauma. Early theorists posited that the experience of stress is the result of a discrepancy between an environmental demand and the individual's coping capacity (both real and perceived), where failure to meet the demand has serious consequences to the individual's physical, psychological, social, and occupational well-being (McGrath, 1976; Lazarus & Folkman, 1984). Therefore, greater differences between environmental demand and coping capacity exert a greater strain on the individual's resources, and generate a greater experience of stress. This experience of stress is arguably neutral, but can be divided into two subtypes: *eustress* and *distress*. Eustress is when the environmental demand is within an individual's coping capacities, and facilitates adaptive coping, whereas distress occurs when the strain of the environmental demand is outside the individual's coping capacities. This disrupts adaptive responding, triggering feelings of helplessness, anxious avoidance, and depressive withdrawal. The implications of this argument are that individual differences in coping resources and the size of the stressor interact, and ultimately determine, the relative degree of stress.

Generally speaking, this framework describes the process of adjustment *during* a stressful event, whereas trauma (in the context of PTSD), is used to describe the psychological damage *after* an extremely stressful event. In keeping with McGrath (1976) and Lazarus & Folkman (1984), a traumatic event can then be described as an experience of *extreme* stress; a serious imminent threat to life or physical integrity to which the individual reacts with helplessness or horror (APA, 2000), and thus feels powerless to effect control over the stressor. As such, trauma reflects "…people's inability to come to terms with real experiences that have overwhelmed their capacity to cope" (van der Kolk & McFarlane, 2007, p.4). However, the capacity to cope and adapt to imminent stressors is known to be moderated by a range of variables implicated in the development of PTSD, which typically fall into a diathesis-stress framework. In psychological terms, the diathesis-stress framework states that individual differences (such as genetic or psychological vulnerabilities) can remain latent and undetected until activated by a sufficient stressor. These vulnerabilities may increase their vulnerability to distress and maladjustment when faced with an overwhelming stressor and interferes with individual functioning, leading to the development of psychopathology (Ingram & Price, 2001). The experience of distress is exacerbated and made chronic due to concomitant and enduring information-processing biases, reduced performance, impaired coping, and disturbances in emotion regulation (Elwood et al., 2009).

Diathesis-stress models, while inherently trans-diagnostic, have been applied to PTSD. Though the traumatic event serves as the stressor that instigates the PTSD syndrome, vulnerabilities present before and during the stressor increase the risk of PTSD compared to those who do not possess those traits. McKeever & Huff (2003) present three diathesis pathways to PTSD development. The first is *residual stress*, which refers to the immediate and enduring stress experienced following a traumatic event. The second is *ecological*, and consists of pre and post trauma factors related to social support and interpersonal relationships styles. And the third is *biological*, which reflects neurocognitive vulnerabilities developed during childhood that predisposes trauma survivors to hyperarousal and dissociation. McKeever & Huff (2003) argue that the contribution of these three pathways varies on an individual basis and factors within and between these pathways will interact to generate vulnerability to PTSD if a criterion A stressor is experienced.

Like McKeever & Huff (2003), Vogt, King, & King. (2007) argue that there are multiple pathways to PTSD vulnerability. But, constructing a systemic, integrated, diathesis stress model of PTSD development is problematic because vulnerability factors identified in some studies have not been identified in others, and those studies in which there is agreement over what variables constitute stable vulnerability factors, vary in their measurement of that factor's predictive power. It has also been argued that the results and data trends are the result of the population of interest, and often an artefact of study design and measurement (Elwood et al., 2009; Vogt et al., 2007). Furthermore, Vogt et al., (2007) have argued that the literature exploring vulnerability (or risk) factors for PTSD development is not consistent in its approach to identifying what qualifies as a risk factor, and therefore use Kraemer et al's (2001) risk factor framework to critically evaluate the issues surrounding this research. This section will also evaluate the status of risk factors for PTSD according to Kraemer et al's (2001) framework. Given that many of these variables have not been categorised this way, this chapter will refer to them as vulnerability factors.

1.2.1. Kraemer et al.,s (2001) Risk Factor Framework**.**

In this framework, *risk* can be defined as the probability of the outcome (developing PTSD) after a traumatic event. A *correlate* is a variable that is associated with that outcome, but does not bear any temporal or causal properties that characterise its relationship to the outcome. A *risk factor* is a correlate which is demonstrated to have temporal precedence over the outcome to which it is associated. Finally, a *causal risk factor* is a correlate, which in addition to having demonstrated temporal precedence to the outcome, if it is experimentally manipulated, is able to effect change in the outcome. Variables that may act as *correlates* or *risk factors* fall into one of two categories – those of *moderators* or *mediators*. Moderators are fixed variables that set the conditions by which independent variables (IVs) cause change in their dependants (DVs), and thus modify the effect of the IV on the DV (c.f. Baron & Kenny, 1986). Mediators, however, are intervening variables that are caused to vary by the IV, but also effect change on the DV. As such, mediators are variables that can be manipulated to effect outcome. Being manipulable, they also have the quality of sharing causality with the IV, allowing for total or partial mediation of an effect. Given the nature of risk factor research for PTSD, it is essential to understand the different types of relationships between potential risk factors and PTSD development and how they manifest statistically. Kraemer et al., (2001) identify several different relationships between two variables *A* and *B*, and an outcome measure *O*.

The first relationship is when *B acts as a proxy for A:* One characteristic of epidemiological and risk factor research in PTSD is its use of overarching variables such as socio-economic status [SES], trauma and psychiatric history (both personal and familial), as a means of capturing broad characteristics within a population that are related to PTSD development. The ease of measuring these variables is evident, but unfortunately sacrifices specificity for sensitivity. Kraemer et al., argue that variable *B* is a proxy for *A*'s relationship with *O* if neither *A* nor *B* have temporal precedence, both *A* and *B* are correlated with each other, and with *O*, but *A* is a greater predictor of *O* than *B*. An example in PTSD literature would be a hypothetical relationship between lower SES and risk of PTSD, where SES may hypothetically be a proxy variable for the effect of lack of resources on stress and coping. Kraemer et al., (2001) suggest that this reveals the aggregate effect of overarching variables and encourages their disaggregation to elucidate the true risk factors. Essentially, these variables, while capturing some essence of the true risk factor, also capture a lot of error variance that might contribute to the heterogeneity of results found between studies.

The second relationship is when *B acts as a mediator of A:* This relationship is there to explain how variable *A* effects change in outcome *O*. In research, one would establish the temporal precedence of measurement *A* (such as in longitudinal or prospective designs), find a correlation between *A* and *B*, with the quality of that correlation identifying total mediation (*B* has greater power to predict *O* over *A*), or partial mediation (equal power between *A* and *B* in predicting *O*). The nature of this relationship provides preliminary evidence for a causal chain between *A*, *B*, and *O*, but does not begin to establish cause and effect unless used in experimental designs.

The third relationship is when *A acts as a moderator of B:* Kraemer et al. (2001) note that any variable can be used as a moderator depending on choice of linear model and sample size, so they re-operationalize the moderator concept in the following way: If *A* has temporal precedence over *B* (either in terms of either concept or measurement), *A* may identify one or more sub-populations where the distribution of *B* is homogeneous, but the effect of *B* on *O* is dependent on the conditions set by *A*. In this

scenario, *A* and *B* are uncorrelated, but there share comparable predictive power. The value of this relationship in epidemiological and risk factor research is that it suggests that moderators can create different chains of causality. In the case of PTSD risk factor research, *A* (history of child abuse) and *B* (present-day sexual/physical assault) are hypothetical vulnerability factors for *O* (subsequent PTSD), but the evidence demonstrates that *A* moderates the effect of *B* on *O*. As moderators are fixed values, they can often be seen as vulnerability or resiliency factors that change the effect of the variable of interest.

Overall, Kraemer et al's (2001) framework states that if a measurement of a PTSD correlate does not precede the manifestation of the syndrome, it is a concomitant. If temporal precedence can be determined through longitudinal and experimental designs, then it is a risk factor. If it is a risk factor, then it is one of these types: 1) moderators, which do not change over time in an individual (dichotomous variables), and are considered antecedents to the syndrome, and 2) variable risk factors (or mediators) change over time and can be manipulated. If it can be truly manipulated then one can assert that it is a causal risk factor. Finally, given that all of these conditions are met, the nature of these variables necessitates viewing them as potential proxies of underlying constructs, The implications of this framework are, however, that what researchers currently identify as risk factors, may not be statistically justifiable given the use of cross-sectional designs in which the conditions required to establish risk factor status are not available.

1.2.2. Risk Factors in the General Population

The large epidemiological studies reviewed in the previous section identified several factors that were significantly correlated with PTSD development, namely the experience of prior trauma, and the continually replicated dose-response relationship between trauma type, exposure, and likelihood of PTSD. Additionally, Breslau et al's (1991) study of young adults in Michigan identified early separation from caregivers, a family history of anxiety, and pre-morbid anxiety and depression as variables that were associated with an increased likelihood of developing PTSD following a traumatic event. However, these factors were indicated by a statistically significant over-representation of the demographic factor in the sample of those with PTSD, and lacked the specificity to demonstrate their contribution in the aetiology of PTSD (Brewin et al., 2000). Klein & Alexander (2006; 2009) compiled a summary of vulnerability factors. They identified significant *pre-trauma* moderating factors such as interpersonal violence, personal or familial history of mental illness, female gender, lower SES or a minority status (such as ethnicity), and childhood abuse. Identified *peri-trauma* factors involved trauma severity, dissociation or intense negative emotions during or immediately after the experience, and the perception of life-threat. The most significant *post-trauma* factors were identified as concurrent life stressors and the absence of emotional support.

However, like epidemiological studies, risk-factor research suffers with the same methodological weaknesses. The variety of experimental designs (cross-sectional/retrospective *v* prospective), the type of measures used (screening tools *v* diagnostic interviews) and their inherent limitations, are arguably significantly responsible for the heterogeneity of results found in risk factor studies. This limits the generalisation of results from a systematic review alone, and thus calls for an empirical analysis of the true effect value of these variables in a weighted meta-analysis. The meta-analyses conducted by Brewin et al., (2000) and Ozer et al., (2003) are influential works that identified risk factors across a range of

studies that have sampled from a broad range of trauma populations, and thus have been widely cited, so it is necessary to elaborate on their results here.

Brewin et al., (2000) conducted one of the first meta-analyses of risk factors for PTSD development, spanning several trauma populations. They studied 14 separate variables thought to influence PTSD. Moderating demographics such as age at trauma, gender, and ethnicity, was significant PTSD predictors, but not consistently across populations, whereas moderators like education and trauma prior to the index event, were significant predictors across the whole analysis, but varied in their predictive power according to population and methodology. The most stable predictors were moderating pre-trauma factors relating to the experience of childhood abuse or a history of psychiatric problems within the immediate family. The most powerful of the predictors were peri-traumatic, such as severity of exposure, or post-traumatic mediators such as additional life stress, or a lack of social support. Nevertheless, the predictive power for all of these variables was low to moderate at best. Brewin et al., (2000) concluded that even though several key variables have been identified as significant risk factors for PTSD, the heterogeneity of their predictive power points to both a heterogeneous disorder that, while being largely the same syndrome, manifests flexibly depending on population, trauma, and intrapersonal effects, and also a simultaneous artefact of experimental design. As such, they warned against using risk factor research to form a universal vulnerability model for PTSD, but encourage the further exploration of these intrapersonal and trauma characteristics

Ozer et al., (2003) explored this in their meta-analysis, concentrating on pre and peri trauma personality characteristics, trauma features, and post-traumatic sequelae. They meta-analysed seven predictors in three groups: *pre-trauma* factors such as trauma history, prior psychological adjustment problems, and family history of mental illness (like Brewin et al., 2000); *peri-trauma* factors such as dissociation, perceived threat to life, and emotional responses; and finally, *post-trauma* social support. In terms of *pre-trauma* variables, prior trauma yielded significant but weak power (r = .17), but the strength of this relationship was influenced by exposure to civilian (r = .27) rather than combat-related (r = .18) interpersonal violence, or accidents (r = .12). Prior psychological adjustment problems yielded a greater predictive value, and were yet again moderated by the experiencing of civilian (r = .31) and combat-related (r = .28) interpersonal violence. The same was also true of a family history of psychological problems, which although was a relatively weak predictor overall (r = .17), was substantially more correlated when the current index trauma was civilian interpersonal violence (r = .31) compared to combat (r = .12), or accidents (r = .08). *Peri-traumatic* variables such as perceived threat to life yielded a weighted mean of r = .26 and were significantly predictive of PTSD diagnosis and symptom severity, with relationships between diagnosis and perceived threat being stronger by the experiencing of civilian interpersonal violence (r = .36), and less so with the experience of accidents (r = .20). Emotional responses at time of trauma was a significant predictor of PTSD diagnosis (r = .26), as was dissociation (r = .35), but this varied depending on assessment methods and time elapsed since the index trauma. Finally, *post-trauma* social support was also a significant predictor (r = -.28), where less perceived support is predictive of PTSD symptoms. However, this relationship was influenced by the time elapsed between trauma and assessment which was longer than three years (r = -.42).

Though dissociation and social support have been identified as moderately powerful correlates of PTSD symptoms, several studies on military veterans reveal that a vulnerability model for PTSD is better

accounted for by a cluster of pre, peri, and post-trauma variables. King et al., (1998) used data from the National Vietnam Veterans Readjustment Study (NVVRS; Kulka et al., 1990) to assess the influence of recovery and resilience factors. Measures of hardiness, warzone stressors, post war life stressors, post-war social support, and subsequent PTSD were used. King et al found that those who exhibited greater hardiness (similar to a high 'internal locus of control'), presented with fewer PTSD symptoms, and was a direct predictor of PTSD (r = -.25 men; r = -.28 women). They also report that this was similar to findings of the effect of hardiness on physical and mental health. Similarly the lack of functional post-war social support yielded the greatest direct relationship with PTSD (r = -.42 men; r = -.47, women). Interestingly, the experience of traditional combat only yielded indirect relationships with future PTSD (r = .62 men; r = .42 women). Of great interest is that their structural equation model (SEM) revealed that the strength of hardiness as a predictor was associated with its indirect effect shared with functional social support, which they attribute to notion that 'hardy' persons are likely to have greater ability to elicit support effectively. Conversely, the model showed no significant link between hardiness and combat exposure. However, this could be because hardiness might not reduce the experience of traumatic stress, but may rather act to mobilise resources to process and promote recovery. Overall, King et al. found that their SEM was optimal when post-war factors were added as mediators between warzone stressors and PTSD. Though the data used was collected retrospectively in the original NVVRS and therefore introduces artefacts of traumatic stress symptoms into the measurements, the model clearly demonstrates that personal resilience during the experience of traumatic events does not reduce the experience of stress; but that it may improve the mobilisation of resources to process trauma and promote recovery. The implication of this conclusion is that chronic PTSD is not necessarily just the result of being traumatised, but influenced by a failure to mobilise resources central to the course of processing trauma. A particular strength of this study is that it acknowledges the role of intrapersonal factors (hardiness), environmental factors (the traumatic stressor), and systemic factors (social support) as interactive events that mediate and predict the occurrence of PTSD.

Similar findings regarding the interaction or pre-trauma vulnerability, and recovery variables were reported by Koenen et al., (2003), who conducted a 14-year longitudinal study on an independent sample of Vietnam veterans (n = 1377). Higher levels of combat exposure, depressive symptoms, and perceived negative attitudes from the community upon repatriation at Time 1 predicted PTSD chronicity at Time 2. Conversely, community involvement at Time 1 was associated with decreased PTSD risk at Time 2, suggesting that levels of social support were positively correlated with PTSD development. This apparent mediating effect of post-trauma support was also demonstrated by Frazier et al., (2011) in a civilian prospective study of students (n = 264) who completed two online surveys of pre-trauma risk and resilience factors. Those who experienced criterion A events were assessed for PTSD. It was found that the predictive relationship of risk/vulnerability factors was significantly partially mediated by unsupportive social interactions, highlighting the influence of the post-trauma environment on symptoms and recovery.

In an earlier study, King et al., (1999), found that their SEM including pre-trauma, warzone, and post-trauma recovery variables accounted for 72% of the variance, which is an unusually high yield for risk-factor research. For men, peri-trauma factors contributed the most variance, followed by post-trauma support, and finally by pre-trauma factors. For women, post-trauma factors were superordinate, followed

by peri then pre trauma factors. King et al., (1999) also argue that pre-trauma variables such as family instability (including abuse) mediate resource mobility both during traumatic stress and also during the recovery environment. The implication here was that PTSD development is largely predicted by this complex interplay of factors over a lifetime, and is not solely predicted by the trauma *per se*, which has been traditionally considered the central aetiological agent (APA, 2000; Davidson & Foa, 1991). The fact that combat exposure is protracted introduces courses of variables that modulate the severity of the stressor, but also introduces the homeostatic effect of resource mobilisation on the ability to withstand traumatic stress. King et al., (1999) argued that the corollary of these findings is that a broader view of traumatic stress be taken from brief, singular traumatic events, and extended to protracted traumatic episodes that deplete coping resources. This includes protracted events that may include experiences that are arguably lower in their level of traumatic intensity, but nevertheless cumulative in their traumatic effect.

1.2.3. Discussion

These two meta-analyses identified moderating demographics such as younger age and socioeconomic status that serve as mild vulnerability factors. Childhood sexual abuse, family problems and psychiatric history are also identified as reliable vulnerability factors, and also that these variables demonstrate a higher relationship with PTSD across studies compared to their socio-demographic counterparts. According to Kraemer et al.,s (2001) framework, this may be because socio-demographics are broad constructs which aggregate subtle but nevertheless influential social variables, whereas the clinical variables are more specific in nature. One thing that is apparent and expected is the heterogeneity in relationship strength between variable and outcome for these two meta-analyses. For example, social support is the strongest predictor in Brewin et al., (2000), but is markedly weaker in Ozer et al., (2003). Nevertheless, subsequent studies have exposited this relationship, showing that negative social interactions increase the risk of PTSD (Adams & Boscarino, 2006), whereas positive and increasing levels of social support decrease risk (Ozer & Weiss, 2004), providing preliminary evidence to suggest that social support is a factor that mediates PTSD chronicity.

Furthermore, these two meta-analyses both identify significant *pre trauma* (trauma history, family psychiatric history, prior mental health problems and adjustment), and *post-trauma* variables (social support) that exhibit moderating and mediating relationships with PTSD. The universal finding that the experience of prior trauma is a significant predictor of PTSD (Brewin et al., 2000; King et al., 1999; Ozer et al., 2003; van der Kolk & Greenberg, 1987), and may even sensitize one to future traumatic events (Dougal et al., 2000). Brewin et al., (2000) provide strong evidence for a dose-response relationship between degree of trauma severity and PTSD, which is supported by other studies (Dowrenwend, 2006). This is further explored by Ozer et al., (2003) who revealed the interactive effects of all the pre-trauma variables with specific trauma characteristics. For example, the experience of civilian interpersonal violence yielded the greatest relationships between pre-trauma vulnerabilities and PTSD diagnosis compared to accidents of combat exposure. Not only is this indicative of a dose-response relationship between trauma and outcome, but it also reveals a potential proxy nature for these variables. One difference between combat and civilian violence may be one of psychological/physical preparedness and

resource access, whereas the low risk associated with the experience of accidents compared to assault may be indicative of the lack of malicious intent.

There are also methodological considerations. With reference to the Kraemer et al. (2001) framework, other issues that become evident are that the types of methodologies used to identify vulnerability factors are limited in their ability to establish the true temporal precedence and manipulability required of a causal risk factor. Therefore, most variables identified as risk factors cannot be considered causal, but are more likely concomitants. While this is true of retrospective and cross-sectional designs, even prospective studies require retrospective assessment of some diagnostic features. For example, *peri-traumatic* variables such as dissociation, perceived threat to life, and even the description of the event itself and the level of traumatic exposure, are measured post-traumatically, and are therefore confounded by diagnostic features of PTSD, especially if the disorder is severe. For example, dissociation can create the paradoxical phenomenon of vivid but incomplete memory, but often with the separation of both severe negative affect and experience at the time of the event (Brewin et al., 1996; Ehlers & Clark, 2000). This may lead to inaccurate reporting of duration, distress, and level of exposure. Perceived threat to life during the trauma is an unclear concept in itself. Some traumas are protracted and facilitate more cognitive evaluations of the event (Brewin & Holmes, 2003), others are so quick (e.g. RTA's; accidents resulting in traumatic brain injury) that the victim may be unaware they have been traumatised until after the trauma's occurrence, yet nevertheless still present with the PTSD syndrome. Furthermore, asking someone to rate their perception of threat to life post-trauma may confound the issue: those whose functionality has been more severely affected by trauma may rate it as a greater threat to their life as a result.

Also, the types of measurements used to quantify the contribution of these vulnerability factors often assume broad, socio-demographic or moderator-type qualities which may not be accurate. For example, the vast majority of risk-factor research is conducted using populations where many have already undergone a trauma prior to the index event, and have since developed the PTSD syndrome and either remitted or remained chronic. This means that pre and peri trauma vulnerability factors are measured as moderators, when the quality of that variable is likely that of a mediator. For example, *pre-trauma* factors such as the presence of a familial psychiatric history *A*, are treated as moderators of the effect of the index trauma *B*, on the risk of PTSD development *O*. However, the moderating power of *A*, may depend less on the presence of family or personal psychiatric history, but more on its interactive effect on the family system, and the resulting relational styles that the family uses to manage this effect. This system has to then further model to the dependant how to deal with distress and conflict as means of maintaining the stability of their caregiving attachments, which the dependant has to then put into action. All of these variables are then of course moderated by the developmental stage of the dependant, which may well predict the rigidity of her or her attachment styles that were learned from an unhealthy family system. This resulting level of rigidity may well then act as a predictor of schema rigidity, which is considered a factor in PTSD chronicity (Janoff-Bulman, 1989; 1992). Nevertheless, this complex interplay of events is operationalized under the misleading label of a 'family history of psychiatric problems'. This is not due to negligence, however. It is impossible to assess the relative contribution of all of these systemic factors post-hoc. However, it does highlight that what are often captured as moderators, are really mediating events that follow a protracted, reflexive, course that are characterised by the repeated interaction of variables within and between events.

Similarly, the experience of trauma prior to the index event, which epidemiology has identified as being more common than not, reveals this to be a significant vulnerability factor. Yet according to these meta-analyses, the relationship between prior trauma and PTSD after the index event is somewhat weaker than would be predicted from its high prevalence in epidemiological studies. An implication from the use of this variable is that the experiencing of a prior traumatic event *per se* is a predictor of future traumatisation and PTSD. Not only that, but it also assumes chronic, unresolved, psychological vulnerability, and this is not always the case. Some research reveals an inoculation effect from prior trauma (Norris & Murrell, 1988), a concept allowed by current PTSD theory, and the ultimate goal of therapeutic change (Baker, 2010), providing that there are sufficient resources to process and overcome the trauma, and the person being more flexible and resourced as a result (c.f. Janoff-Bulman 1989; 1992). In the context of prior childhood sexual abuse as a significant vulnerability factor, the predictive power of such an experience may be less to do with the experience of trauma as a child, but as experiencing it as part of a relational system where survival may be dependent on conformity to that abusive system, and therefore that trauma is non-resolvable.

However, the measurement of post-trauma variables may take on different challenges. Brewin et al (2000) and Ozer et al., (2003) identified a lack of post-trauma social support as a significant predictor of PTSD. This variable requires significant attention, as in military samples, it has been demonstrated that upon repatriation from the war theatre, a perception of lack of emotional support was highly predictive of PTSD (Koenen et al., 2003). However it is unclear whether the presence or absence of emotional support is a predictor because of lack of opportunity to process traumatic memories when emotional resources are already depleted, whether the lack of empathy from others results in secondary wounding, or because trauma-specific adaptive responses preclude the effective engagement of emotional support. In keeping with PTSD theory, it may be that emotional depletion, PTSD symptoms, and trauma-specific coping strategies moderate the sufferer's ability to engage emotional support, which may in itself partially mediate the chronicity and severity of the disorder. The implication here is that a true vulnerability model for PTSD is multifactorial, systemic, interactive, and integrative, and with a range of residual, ecological, and biological factors contributing to PTSD vulnerability (McKeever & Huff, 2003).

1.2.4. Conclusions

Overall, the research suggests that while, as predicted, widely accepted pre, peri, and post trauma factors (prior trauma, dissociation, social support) all contribute to the risk of PTSD development, their individual level of predictive power is low. Additional evidence from military and civilian samples suggests that their combined interactive effects account for more variance in the relationship than the individual predictors alone, further re-enforcing the hypothesis that PTSD has a multivariate aetiology. Furthermore, the moderating effects of prior trauma on the index traumas likelihood of generating PTSD are mediated by the post-trauma recovery environment. The implications of these findings are that the individual risk factors are less important than their systemic and interactive effects both in terms of the experience of traumatic events, and the subsequent recovery environment, pointing toward a complex diathesis-stress model for PTSD.

In terms of Kraemer et al.,s (2001) framework, the central requirement of a causal risk factor is that it has temporal precedence over the trauma and that it is manipulable. If both conditions are met, then from a clinical perspective, at-risk individuals can be identified and interventions put in place to buffer these individuals against chronic PTSD. This would require an increased use of prospective research designs which measure mediators in process and course, and utilise control groups to control moderating effects (Baron & Kenny, 1986). Brewin et al. (2000) included many high quality prospective designs in their analysis, but even these are unable to measure essential features of the syndrome. However, the requirement of temporal precedence for a causal risk factor may not be strictly relevant to PTSD. It is only true if one adopts the supposition that temporally precedent factors are singularly responsible for trauma responses – as well as their maintenance. From the research cited above, post-trauma mediators of PTSD chronicity break away from this definition, but this lack of temporal precedence also challenges the notion that pure PTSD is solely the result of criterion A and pre trauma vulnerability.

The concept of course within a syndrome extends not just to the experience of post-traumatic symptoms, but also to the course of coping within a traumatic event. The course of coping within a trauma may affect the event's relationship with its vulnerability factor, and this may become more apparent the more protracted a traumatic episode becomes. Prolonged combat exposure and domestic abuse scenarios are prime examples of protracted traumatic events that are characterised by dynamic changes in immediate threat and comparative safety, but also characterised by unpredictability, chronic hyper-vigilance, and the requirement to immediately mobilise depleted emotional resources (Smith et al. 1999). These fluctuating courses of events may interact with vulnerability factors differently from more immediate traumas, and this complex interaction may also be a predictor of post-traumatic symptoms. However, comparatively little research has been done to capture the course of adjustment and adaptation within a traumatic episode. In summary, the influence of risk factors in PTSD may be multifactorial and interactive both during and after traumatic episodes. This may affect the way in which we consider the process of traumatisation and the concept of the traumatic event being the major aetiological agent.

1.3. Clinical Theories of PTSD

Thus far, previous chapters have established that pre peri and post trauma factors are implicated in the development of PTSD. As such, these variables will also be accounted for in clinical theory. Over the past thirty years, several clinical theories of PTSD aetiology, development, and maintenance, have been proposed with a view to explaining the PTSD syndrome, its course, establishing the focus of treatment, and identifying what makes treatment effective. These are the schema theories (Epstein, 1985; Horowitz, 1986; Janoff-Bulman, 1992), emotional processing (Foa & Kozak, 1986; Foa & Rothbaum, 1998), dual representation (Brewin, Dalgleish, & Joseph, 1996), and cognitive (Ehlers & Clark, 2000) approaches. In recent years, critiques of these theories have emerged, in particular, Brewin & Holmes (2003), Cahill & Foa (2007), and Dalgleish (2004), but have not used the same evaluative criteria. This is important, because criteria vary according to the focus of the review. For example, Cahill & Foa (2007) have a clinical focus, which necessitates an explanatory account of psychiatric symptoms and associated

phenomenology such as cognitions, changes in core beliefs following trauma, typical courses following trauma, and how therapeutic interventions improve outcomes. Dalgleish (2004), however, focuses on the internal consistency of clinical theory, and its emergent ability to generate both correct and erroneous predictions. This means that a good theory of PTSD must be able to correctly explain and predict the role of key variables in the development and maintenance of PTSD. But given that vulnerability to chronic PTSD is better explained by interactive relationships between variables rather than singular factors (chapter 1.2.), it is necessary to question whether this finding is also reflected in the limitations of PTSD theory. With reference to these reviews, the aim of this chapter is to describe and critically evaluate, clinical theory related to the aetiology, development, and maintenance, of PTSD. The final discussion will synthesise these evaluations, and highlight common weaknesses in explanatory and predictive power that need to be addressed in future research.

1.3.1. An Evaluative Framework

This evaluation was conducted using a framework that focused on criteria related to the internal and external validity of theory. Dalgleish (2004) argued that a theory's internal validity is established by its parsimony, specificity, and power to generate specific, testable, predictions. A good theory would have the minimum number of components required to describe all variations and dimensions of data for the phenomenon of interest, and should be able to explain interactions between components which will also be empirically verifiable in the data. Thus, internal validity of a theoretical construct is ultimately tested in its ability to predict (rather than just explain) patterns in future data. This approach is primarily concerned with evaluating the internal structure of a theory as a technology, and is therefore a framework that can be applied to theories in general. Cahill & Foa (2007), however, argue that a PTSD theory must also be able to provide comprehensive accounts of psychopathology, course, individual differences that affect presentation and course, and a model in which to conceptualise treatment efficacy. These criteria are more concerned with a theory's ability to explain PSTD symptomatology (a function of external validity), and using the theories inner workings to predict the course of disorder, both with, and without treatment. As such, these criteria will be applied to investigate the weaknesses in clinical theory. Table 2 provides an overview.

Table 2: A synthesised criteria set for the evaluation of theories regarding PTSD psychopathology

General Theoretical Power (see Dalgliesh, 2004)	PTSD-specific Explanatory Power (see Cahill & Foa, 2007)
Parsimony The minimum amount of components necessary to model the active processes in the theory, with the maximum amount of theoretical abstraction. *Specificity* Can the theory account for specific datasets rather than just broad empirical findings? *Predictive Power* Does the system of theoretical components generate empirically testable predictions? Does the interaction of components generate any erroneous predictions?	*Psychopathology* Accounts of: 1. the PTSD syndrome (Criteria B-D; APA, 2000) 2. primary (automatic) and secondary (appraisal-driven) emotions (Brewin et al., 1996; Andrews et al., 2000; Pitman et al., 1991) 3. trauma-related cognitions (Ehlers & Clark, 2000; Foa & Rothbaum, 1998) 4. the transformation of meaning (Horowitz, 1986; Janoff-Bulman, 1992). *Course of the Syndrome* 1. An understanding that the PTSD syndrome is a common post-trauma presentation from which the majority of experiencers recover and do not develop chronic PTSD (Criteria E-F; APA, 2000) 2. A working model of factors that facilitate and/or preclude recovery *Individual Differences Affecting Presentation and Course* Accounts of: 1. pre, peri, and post-trauma variables (Brewin et al., 2000; Ozer et al., 2003) 2. cognitive processing styles (Brewin et al., 1996) 3. traumatic exposure intensity (Breslau et al., 1889; Kessler et al., 1995; Kilpatrick et al., 1987). *Treatment Efficacy* An account of how therapeutic interventions manipulate the proposed psychological mechanisms to generate therapeutic change (Cahill & Foa, 2007; Dalgliesh, 2004).

1.3.2. Schema Theories

According to Fiske & Linville (1980), the term *schema* refers to how the human mind cognitively represents knowledge gathered throughout the human lifespan. Such schemas categorize and organise this information in a meaningful way by categorising relationships between concomitant stimuli and experiences, and thus provide a cognitive framework to which new experiences can be compared and processed. As such, a schema serves as a psychological heuristic that provides a stable model for predicting how the world operates, and how the social self relates to the world (Bartlett, 1932). This need for stability means that the processing of life experience is biased towards maintaining these schemas (Cantor & Mischel, 1997; Langer & Abelson, 1974), and as such, information that is congruent with these schemas is more easily processed and recalled than information that is not (Swann & Read, 1981). Consequently, schemas are hard to change, but do so with the slow but steady integration of new information over time (Fiske & Taylor, 1991). This allows the synthesis of old and new information into a modified schema without replacing the schema altogether (Janoff-Bulman, 1989). The psychological imperative to resolve experiences within existing schemata is known as the 'completion tendency' (Horowitz, 1986).

1.3.2.1. Stress-Response Theory Applied to PTSD

However, this psychological imperative to process experience is put under significant strain when experiences during the life-cycle challenge one's deepest-held beliefs. According to Stress Response Theory (SRT; Horowitz, 1986), those who experience trauma find the influx and intensity of new information and emotion so sudden and overwhelming that the experience cannot be integrated. The tendency for 'slow and steady' schema modification collapses under the strain, and the individual experiences a failure to 'complete' this process. After the initial traumatisation, there is a period of shock where psychological defences are activated, and the survivor experiences emotional numbing and denial. However, the completion tendency eventually compels attempts to process this new information, which manifests in the form of re-experiencing symptoms. Horowitz (1986) suggested that under these circumstances, the completion tendency is achieved via a recurring cycle that regulates the flow of trauma information using the psychological defence from traumatic memories and re-experiencing symptoms. SRT states that it is this cycle that is responsible for PTSD syndrome.

1.3.2.2. Schemas

Though SRT accounts for the process of schema modification, other theorists address their contents. Epstein's (1985) cognitive-experiential theory posited that the experience of trauma affects a person's personality, their theory of self, and the world, by challenging (or shattering; Janoff-Bulman, 1992) four core beliefs: 1) *the world is benign*, 2) *the world is meaningful*, 3) *the self is worthy*, and 4) *people are trustworthy*. This forces the trauma survivor to either accommodate new modified beliefs, or assimilate the trauma into the old beliefs. The common view between these three theorists (Epstein, 1985; Horowitz, 1986; Janoff-Bulman, 1992) is that traumatic memories violate previously held beliefs and assumptions, and that the integration of these new memories requires a modification of existing core beliefs.

1.3.2.3. Shared Suppositions and Predictions

Stress response theory - and by extension, the works of Epstein (1985) and Janoff-Bulman (1992) - rest on these suppositions: 1) traumatic experiences themselves are discordant with schemas that govern the initial perception and appraisals of incoming sensory information; 2) The integration of this new information may require modifying existing schemas; and 3), the PTSD syndrome is experienced as part of an adaptive avoidance/re-experiencing cycle which operates to bring gradual homeostasis. This theory also makes several predictions: 1) if cycle continues, the affective intensity of re-experiencing decreases until the PTSD symptoms are no longer present and homeostasis is achieved; 2) the process has failed if the re-experiencing and avoidance continues indefinitely; 3) the re-experiencing-avoidance cycle will start after a period of psychological numbing; and 4) the degree of discrepancy between schemas and incoming trauma experiences predicts the degree of PTSD severity.

1.3.2.4. Critical Analysis

SRT (Horowitz, 1986) provides a foundational account of PTSD *psychopathology* by introducing the concept that the PTSD syndrome is due to the active processing of non-integrated trauma information into schemas. This theory also accounts for the transformation of meaning as it explains the fundamental experience of PTSD sufferers as overwhelming and disrupting of the self. It does not, however, provide an account of trauma-related cognitions or emotions.

SRT can differentiate between the two major *courses* of symptoms: 1) the natural, normal, course of these symptoms is a dynamic cycle of avoidance and re-experiencing that gradually declines in frequency and severity as new information is integrated; and 2), the chronic, pathological, course of these symptoms occurs if the defensive avoidance mechanisms are above or below optimal functioning. These two courses generate testable predictions: 1) if the defences are under-optimal, it will result in chronic PTSD; and 2) if over-optimal, will result in avoidance strategies like substance abuse and protracted situational/experiential avoidance, which inhibit the intrusive symptoms. When these strategies can no longer be maintained, delayed-onset PTSD may occur. Prediction 2 is not supported in the light of evidence that delayed-onset is arguably the rarest course (Freuh et al., 2009), and of those who do exhibit this course, the vast majority are either experiencing exacerbations of prior PTSD (Andrews et al., 2007), or a progression from sub-syndromal symptoms (Smid et al., 2009).

SRT is also very weak in its predictions of how *individual differences* affect the emergence and course of the syndrome. Closer examination reveals more predictions that diverge from the empirical literature. For example, SRT cannot explain why it is that a minority of trauma survivors develop chronic PTSD, or why some traumas are more likely to generate PTSD than others (Kessler et al., 1995; Norris, 1992). SRT (and by extension, the works of Epstein [1985] and Janoff-Bulman [1992]) also predict implicitly that the more discrepant with trauma individual schemas actually are, the more severe the PTSD syndrome. It has been noted by several other commentators that if this were true, then those individuals with less dissonance between the two would experience less severe symptoms and a quicker recovery (Brewin & Holmes, 2003; Cahill & Foa, 2007). This is not supported. Populations with previous psychiatric histories

(Kilpatrick et al., 1985) or multiple traumas (Brewin, et al., 2000; Kessler et al., 1995; Ozer, et al., 2003; Resnick, et al., 1993), are consistently more vulnerable to future chronic PTSD. It has been argued that such populations are likely to have negative schemas of the self, the world, and others, which are more likely to be confirmed rather than challenged by traumatic experience (Brewin et al., 1996). Present-day clinical knowledge is clear that previous trauma is not only a vulnerability factor in future PTSD, but can also be 'reactivated' and merged with present day PTSD symptoms (Nishith, et al., 2000), in which circumstances it may have never achieved an effective synthesis, leaving the survivor in a state of perpetual traumatisation. Therefore a trauma history may serve as a proxy for those who have yet to recover a stability of self (cf. Janoff-Bulman, 1992). But, given Horowitz's (1986) suppositions, SRT is only able to make reliable predictions for individuals who have never been traumatised, cannot discriminate between the effects of present and prior events, and is thus unable to account for a full clinical history.

In addition to diverging predictions, Brewin & Holmes (2003) have criticised SRT for its inability to predict *treatment efficacy* because it is empirically unable to distinguish between symptom reductions due to recovery or elaborate avoidance. Schema approaches state that schemas interact with the influx of current experience, but SRT also fails to elaborate on the processes required to transfer new trauma information from flashbacks into existing schemas. This is a central weakness, as despite its simplicity, SRT does not account for processes involved the interaction of its components (see Table 1, general theoretical power). Neither does it explain how these flashbacks differ from ordinary memories, or how peri-traumatic emotional responses such as dissociation (Ozer, et al., 2003) impact the experience of these flashbacks. Furthermore, SRT says nothing about individual appraisals of trauma and how they mediate the processing of experience, or how post-trauma factors such as social support may interact with traumatic experience and personal resiliency (King et al., 1998; King et al., 1999) to decrease the risk of PTSD (Ozer & Weiss, 2004), or how the lack of such support can increase the risk (Adams & Boscarino, 2006; Brewin et al., 2000). Therefore, SRT cannot provide sensitive predictions about course and prognosis because it fails to account for individual differences that mediate and moderate course.

Finally, SRT as a social-cognitive approach provides a basic theoretical pathway for schema change, but it is low in abstraction (see Table 1). It does not specify the interaction effect of individual beliefs residing in schemas with trauma, and how that affects PTSD presentation course, or recovery. By extension, SRT cannot predict how the experiencing of specific emotions (which are influenced by beliefs; Beck, 1995) during and after trauma, affects presentation and course. Overall, SRT's diverging predictions and lack of discriminative power threaten the theory's ability to make sensitive predictions regarding patient course and prognosis and thus affects its *specificity* and *generalizability*, highlighting the structural weakness and incompleteness of this theory. However, the information processing theories excel at addressing some of these issues.

1.3.3. Information-Processing Theories

The previous approach was concerned with how the experience of trauma challenges, or 'shatters' a trauma survivor's schemas, and the process of re-adjustment required to create stable, more flexible, and realistic schemas. The information-processing approach, however, is focused on *how* trauma-related

information is represented, stored, and processed, and how therapy can facilitate this natural course in PTSD sufferers.

1.3.3.1. Emotional Processing Theory

Information processing theories such as those by Lang (1977) and Foa & Kozak (1986) suppose that anxiety disorders are maintained by unconscious associative networks. Lang (1977; 1984) proposed that the experience of pathological anxiety is represented in 'fear structures'. Fear structures are associative memories that contain stimulus representations, and the cognitive, behavioural, and physiological reactions to these stimuli. These structures act as programs implemented to avoid danger. Therefore, fear structures that truly represent present danger will facilitate effective action.

However, not all fear structures elicit adaptive responding. According to emotional processing theory (Foa & Kozak, 1986), anxiety disorders too are the manifestation of information networks in memory (fear structures) which contain representations of feared stimuli, fear responses (behaviours and physiological arousal states), and cognitive appraisals associated with the rest of the structure. However, these structures do not accurately represent imminent danger. They are linked with, and activated by, non-threatening stimuli, which then activate unnecessary safety behaviours, interfering with adaptive responding.

1.3.3.2. Suppositions

Emotional processing theory rests on these suppositions: 1) the fear structure is activated when the individual encounters triggering stimuli; 2) this trigger elicits a cascading activation to the all other elements of the fear structure, generating the cluster of cognitive, behavioural, and physiological reactions; and 3), the fear network is activated as an adaptive response in order to avoid perceived danger.

1.3.3.3. Emotional Processing Theory for PTSD

The PTSD syndrome (Criteria B-D) and impact on functioning (Criterion F) are therefore the result of strong, maladaptive fear structures, with a greater number of powerful associations that may not reflect reality. PTSD fear structures contain a remarkable number of non-threatening stimuli that by virtue of them being thematically or sensory-related to the initial trauma, are associated with present-day threat, and have a very low threshold of activation. This can result in the frequent activation of the structure. Developments in the conceptualisation of PTSD within this framework include a synthesis with the schema approach (Foa & Rothbaum, 1998). In this approach, the frequent and rapid activations of trauma-related fear structures - due to thematically-related, but otherwise innocuous, stimuli - lead the sufferer to appraise the world as *"entirely dangerous"*. The present-day re-experiencing of the activated structure, combined with remembering powerlessness during the trauma, often becomes associated with appraisals of "*present-day self-incompetence"* (Foa & Rothbaum, 1998; Rauch & Foa, 2006). These cognitive appraisals of the PTSD syndrome act as maintenance factors –reinforcing fear, and the tendency to avoid that which is believed to be too threatening to confront.

1.3.3.4. Predictions

EP theory predicts that emotional and cognitive engagement by talking through the trauma memories with others promotes a natural recovery from PTSD. This is achieved by exposure to triggers, which inhibits negative reinforcement of avoidance, facilitates activation of the fear structure, and natural processing (Rauch & Foa, 2006). If this continues, natural recovery from PTSD is indicated by a steady decline in symptoms within and between exposures to triggers over time (Foa & Cahill, 2001; cf. Horowitz, 1986; c.f. Rachman, 2001). The act of re-living the trauma prevents the negative reinforcement of avoidance patterns by exposing oneself to related triggers, while simultaneously refraining from the use of avoidance behaviours. This provides the opportunity for beliefs regarding a) the permanence of anxiety, b) their inability to cope with the feelings, c) one's in-ability to overcome the trauma, and d) that thinking about the trauma is dangerous, to be corrected experientially via the extinction of anxiety from within-session habituation (Rauch & Foa, 2006). The nature of the therapeutic relationship provides experiential confirmation of present-day personal safety that can be integrated into the fear network. Finally, the processing of this material into long-term memory allows the individual to see the experience in context - as an isolated incident, and not necessarily a threat to their sense of a comparatively safe world, or personal competency.

EP theory also predicts that emotional processing is impaired by over and under-engagement during exposures. Under-engagement leads to insufficient activation of the fear structure (Foa & Kozak, 1986), whereas over-engagement instigates information processing biases that prevent the integration of new, corrective information (Rauch & Foa, 2006). As such, failure to recover/chronic PTSD is measured by no change in symptoms, and related to failure to engage emotionally with the memory. Therefore, the clinical application for PTSD is to re-live the trauma by using a) triggers to elicit a suitable level of arousal to fully activate the fear structure, and b) use the process of extinction via within and between-session habituation to systematically reduce the intensity of anxiety associated with the trauma memory. EP theory for PTSD also predicts the effect of present-day trauma on pre-trauma beliefs about the safeness of the world and the competency of the self via two routes to traumatisation (Foa & Rothbaum, 1998): the violation of positive assumptions of safety (also known as 'shattered assumptions'; *"I am competent, the world is safe"* (c.f. Janoff-Bulman, 1992); and the positive reinforcement of negative schemas representing one's own competence and safety (*"I am incompetent, the world is unsafe"*). For example, old fear structures related to personal safety and competency that were generated during prior traumatic events will be re-activated and reinforced due to newer traumatic events thematically and semantically resembling the old. Furthermore, those individuals with less flexible beliefs and assumptions, are thought to a) be more vulnerable to the PTSD syndrome (cf. Janoff-Bulman, 1992); and b), appraise the subsequent symptom clusters, their effect on functioning, and the responses of their support network, in such a way that may interact and maintain the disorder.

1.3.3.5. Critical analysis:

Foa & Kozak (1986) approach accounts for the *psychopathology* of the PTSD syndrome (criteria B-D), conceptualising the symptoms as fear structure components that are activated by triggering stimuli. It also accounts for meta-cognitions regarding PTSD symptoms by seeing them as a function of experiencing low-threshold of activation via thematically, but loosely-related triggers. The addition of schema levels or representation allow for both primary and secondary emotions due to appraisal processes, and also accounts for transformation of meaning in the shattering or re-enforcing of core beliefs.

The synthesis of associative networks and schemas can account for *individual differences* in trauma history that can lead to the syndrome via two routes - shattered assumptions and reinforcement. As EP theory for PTSD synthesises a schema approach (Foa & Rothbaum, 1998), it is arguable that trauma information consistent with the schema will be easily integrated as it is consistent with previous learning. Either route will ultimately affect the strength of the fear network as a whole and its various components, leading to a low threshold of activation and severe anxiety. Essentially, the reasons for traumatisation are different, but the resulting syndrome is the same. EP theory also accounts for the *course* of symptoms by stating that behavioural avoidance due to the fear structure as a mechanism precludes normal processing and thus continuing the course of the disorder.

This approach is strong in its account of recovery and *treatment efficacy*. Emotional processing is facilitated via the exposure to triggers, the suppression of safety behaviours, subsequent insertion of corrective information, and emotional engagement by talking with others. This therapeutic approach is able to represent anxiety disorders in general, and can thus accommodate different types of structure for each disorder, providing high levels of *specificity*. EP theory sports significant predictive power as unlike the previous schema approach (Horowitz, 1986), it has been designed to account for the treatment efficacy of prolonged exposure therapy (NCCMH, 2005) and has empirically testable therapeutic outcomes. Exposure has demonstrated considerable effectiveness for simple phobia (Marks, 1979), and PTSD (Foa & Rothbaum, 1998; Taylor et al., 2003; Van Etten & Taylor, 1998). But while the approach explains the efficacy of exposure, the use of associative networks cannot account for the success of cognitive therapy (Ehlers, Clark, Hackmann, McManus, & Fennell, 2005), or explain whether it is between-session habituation (behavioural route) or reappraisal (cognitive route) that produces symptom reduction (Brewin & Holmes, 2003). It may be that between-session habituation and re-appraisal are mutual concomitants, and that the greater the time between sessions, the greater the chance to implicate re-appraisal as the mechanism of action.

There are some additional problems with the suppositions behind the theory: EP postulates that new 'corrective' information is integrated into memory. This is challenged with accumulating evidence that trauma memories remain unchanged in favour of laying down *new*, qualitatively distinct, and complete, long-term memories with neural retrieval advantages (Brewin & Holmes, 2003). Also, it is unclear how the degree of emotional engagement (a comparatively covert behaviour) facilitates processing within this approach.

In addition to this problem, EP theory is focused primarily on fear, possibly at the detriment of other post-traumatic emotions such as anger, guilt, and shame. It has been argued that associative networks could accommodate additional emotions and concomitant appraisals in separate networks (Cahill & Foa, 2007). However an additional interaction between networks is not a feature of the theory. Though another

network may explain the presence of other emotions, it will also generate unprecedented predictions, and reduce its parsimony. Clinically speaking, an additional network that operates the same as a fear structure rests on the supposition that all emotions respond to the same therapeutic approach, whereas research has demonstrated that emotions such as anger (Jaycox & Foa, 1996) or shame (Kubany & Manke, 1995; Lee, Scragg, & Tumer, 2001) do not necessarily habituate, but can impair emotional processing.

It is perhaps not coincidental that other emotion-related aspects of PTSD are not covered in great detail. One key feature of PTSD is the effect of emotional numbing (Criterion C4-6) and dissociation (Criterion B3) on post-traumatic processing and recovery (Ozer et al., 2003). The puzzling nature of individual trauma memories being both vivid and vague (e.g. hotspots vs. dissociative amnesia), is inadequately represented in the associative network approach. Associative networks predict that all the data in the fear structure would be activated when exposed to a trigger, whereas dissociative symptoms dis-integrate aspects of the trauma memory. The recounting of traumatic memories in dissociated manner vs. a fully-engaged manner, and the effect on therapeutic reliving, remains inadequately explained. This lessened ability to account for the effect individual differences on course reduces the model's power to predict the course of treatment at this level of representation.

1.3.4. Synthesised Approaches

1.3.4.1.. Dual Representation Theory (1996)

Brewin, Dalgliesh, & Joseph (1996) posited that human memory uses two simultaneously-operating systems, known as “verbally accessible memory” (VAM), and “situationally accessible memory” (SAM). VAMs are verbal and narrative in structure, can be consciously and deliberately retrieved, and are integrated within the broader temporal and semantic contexts of autobiographical memory. The nature of these memories makes them communicable, but limits them to containing information that has been processed consciously. SAMs, however, contain lower-level sensory perceptions, physiological, and affective reactions. Unlike VAMS, these are non-verbal, and activated involuntarily. As they are situationally triggered, they are neither deliberately retrievable, nor directly communicable. The presence of two different types of memory system has been consistently observed by other researchers (Bucci, 1997, 2001; Epstein, 1994; Leventhal, 1979).

1.3.4.1.1. Dual Representation Theory for PTSD:

In PTSD, the complex nature of VAM memories allow them to contain primary peri-traumatic emotions (helplessness, horror), and secondary post-traumatic emotions/evaluations of both the trauma, and its perceived effect on the self and their world (including anger/rage, shame, and grief). SAMs, however, are responsible for involuntary flashbacks that are triggered by internal or external reminders of the trauma. They often contain basic perceptions of the traumatic scene, somatic sensations of fear, peri-traumatic emotions (and if the trauma is more protracted, evaluations of the self), and are comparatively unprocessed. As such, they are not retrieved verbally, are not linked in with long-term memory, and are therefore not retrieved within semantic and temporal context. The flashbacks are therefore experienced as happening now, and independent of previously acquired knowledge. These distinctions makes the traumatic memories comparatively incommunicable, non-contextual, and therefore very distressing.

1.3.4.1.2. Suppositions

PTSD can be seen as a problem of memory integration and flashback management (Brewin & Holmes, 2003). For integration to occur, SAM's are triggered, experienced, and all information in the engrams receives deliberate, conscious attention. The SAM is recounted verbally in order to facilitate the generation of a VAM counterpart. If all of the SAM memory receives deliberate attention and verbal processing, the VAM will hypothetically have a neural retrieval advantage over the original trauma memory stored in SAM, provided that all sensory data from SAM is better represented as a VAM. This VAM counterpart gives the memory a semantic, spatial, and temporal context, resulting in the memory no longer being experienced 'in the now', providing the experience that the danger is no longer present (and thus place the trauma and the associated emotions and behavioural responses in the past).

1.3.4.1.3. Predictions

Brewin et al.,s (1996) model allows for three different types of emotional processing that promotes either 1) recovery, 2) chronic emotional processing, or 3) the premature inhibition of processing. Recovery is explained by the neural retrieval advantage of VAMs (Brewin & Holmes, 2003) as achieved by the procedure above, and like all previous approaches, is indexed by the amelioration of symptoms. Chronicity of the syndrome, however, is indexed by the recurring retrieval of SAMs with no symptom amelioration, and therefore no reduction in distress. Factors that may affect chronicity are when traumas interact with previous negative schemas, there are competing demands for emotional coping resources, a lack of social support, or in cases of prolonged traumatisation, and an inability to prevent or process intrusions due to on-going threat. Premature inhibition, however, occurs when trauma survivors develop avoidance strategies to limit the activation of the syndrome, and may result in fewer PTSD symptoms, but significant memory impairments, hyper-vigilance to threat cues, and an increased vulnerability to the syndrome if re-traumatised in the future.

1.3.4.1.4. Critical Analysis

DRT is able to explain the *psychopathology* of PTSD as the result of triggered SAMs, which contain sensory, somatic, and emotional information that are characteristic of flashbacks. The subjective experience of these symptoms representing current danger, rather than traumas passed, is a function of these memory engrams being temporally dis-integrated. It follows that the most obvious development in this approach is that, unlike emotional processing theory (Foa & Kozak, 1986; Foa & Rothbaum, 1998; Rauch & Foa, 2006), it proposes a dual, rather than single, memory system. Emotional processing theory suggests that recovery is due to the systematic insertion of corrective information (e.g. present-day personal safety) into a single structure, DRT, however, theorizes that recovery is due to successful processing (or copying) of SAM-stored information into a VAM (Brewin et al., 1996; Brewin & Holmes, 2003). Thus, the update of safety information is achieved not by inserting new information into the trauma memory, but by inserting the trauma memory itself into a long-term memory VAM, which results in corrective information being added to the trauma memory by virtue of context. This provides an excellent account of the transformation of meaning and appraisal-driven emotions that were not adequately represented in previous approaches (Foa & Kozak, 1986; Horowitz, 1986).

Another key strength in this approach is how this theory postulates three *courses of the syndrome* (recovery, chronicity, and inhibition), and can therefore differentiate between symptom amelioration due to recovery or avoidance, (a problem unaccounted for in SRT; Horowitz, 1986), and chronic PTSD symptoms with no recovery (as accounted for by EP theory). These three symptom courses reflect

individual differences in cognitive/emotional processing styles for regulating symptoms that influence course and prevent recovery, but also that those strategies are affected by systemic factors such as social support, and prolonged traumatisation.

This also provides additional clinical applications that may improve *treatment efficacy*. Assuming that these three processing strategies are one of three broad coping mechanisms that PTSD sufferers use, this additional focus may aid the assessment and formulation of an intervention that addresses these factors, improving emotional processing. Furthermore, DRT suggests that emotional processing is more than the systematic insertion of corrective information. It involves an active emotional processing mechanism where the traumatic events and associated bodily/affective states are consciously attended to, experienced, and manipulated in working memory (Brewin et al., 1996) with the aim of processing and copying one memory engram into another. This is supported by neurological evidence for more than one memory system (see Brewin & Holmes, 2003, for a review). This is in contrast to SRT (Horowitz, 1986) which exposited a fundamental psychological mechanism but did not account for other mediating variables. This is also true (albeit to a lesser extent) of Foa & Kozak's (1986) earlier work.

The *theoretical power* of DRT is a result of Brewin et al., (1996), like Foa & Rothbaum (1998), using a synthesised approach. The use of SAM's as the memory engram responsible for re-experiencing symptoms is similar in representation level to fear structures (Foa & Kozak, 1986), and therefore inherits its explanatory and predictive power. Additionally, DRT provides an emotional processing mechanism by which SAMs (or VAMs) are consolidated. This acts as the bridge between SAM transfer and VAM creation – or from the perspectives of the previous two approaches, the method by which a) the completion tendency is achieved, or b) corrective information is inserted.

This is a significant addition: First, it provides a mechanism by which two different components of a theory interact (see Dalgleish, 2004), which is something emotional processing theory and stress response theory did not clarify. Second, the use of VAMs as a site for more complex reactions to trauma and the target of processing SAM-based memory engrams accounts for PTSD and treatment-related phenomena in a way which the early associative networks approach could not: This particular strength of the DRT, despite having more functional components in the theory, is due to the interaction of these components. For example, the use of VAMs explains and predicts the presence of cognition in PTSD symptomatology, and accounts for and predicts the efficacy of cognitive therapy, where associative networks could not (Foa & Kozak, 1986). The account of therapeutic efficacy for cognitive therapy as well as behavioural exposure is parsimonious: DRT states that behavioural exposure works by enabling access to, and the cognitive processing of, SAMs in working memory in order to create new SAMs and also create a context-specific VAM- both with a neural retrieval advantage. Cognitive therapy, alternatively, manipulates VAMs already present to create/modify in such way that they have a neural retrieval advantage over old SAMs. Both achieve the neural suppression of incomplete and under-elaborated memories by more complex narratives, which by virtue of having more connections, possess a retrieval advantage. This is arguably the superlative strength of this theory.

However, several *individual differences that affect presentation and course* remain unaccounted for: the role of schemas in the experience of trauma and the PTSD syndrome is not clear in DRT, with the main impetus of the theory coming from giving trauma memories autobiographical context. However, DRT, like associative networks, also does not explain the role of peri-traumatic dissociation and its

impact on memory. A recurring criticism is that the intrinsic complexity of DRT, while generating excellent explanatory power and new ideas for research (cf. Baker, Gale, Abbey, & Thomas, 2013), has not necessarily yielded an increase in specificity of predictions (Brewin & Holmes, 2003; Dalgleish, 2004), or added anything new to therapeutic efficacy (Cahill & Foa, 2007).

1.3.4.2. The Cognitive Model

Cognitive approaches to mental disorders rest on two suppositions: 1) That it is the cognitive appraisal of events that govern emotional responses, and 2) that specific emotions relate to particular beliefs. In broad terms, thoughts related to anger revolve around the perception of something being done to you that is considered unjust; those related to guilt revolve around you having done something unjust to another; those related to sadness revolve around the perception of loss; and fear, the perception of immediate danger (Beck, 1995).

1.3.4.2.1. The Cognitive Model Applied to PTSD:

The cognitive approach of Ehlers & Clark (2000) postulates that it is common for people who have experienced a traumatic event to then exhibit the PTSD syndrome. In the majority of cases, these memories are processed gradually, and the re-experiencing and hyperarousal symptoms decline over a short period of time. However, a minority of traumatised individuals continue to experience the syndrome indefinitely. Ehlers & Clark suggest that those who do (and thus develop full PTSD) process the memories in a way that leads them to experience a sense of current threat. The perception of current threat is a result of 1) maladaptive negative appraisals of the traumatic experience and the ensuing symptoms and experiences; and 2) a failure to sufficiently elaborate on and integrate the memory within autobiographical context due to "strong associative memory and perceptual priming" (Ehlers & Clark, 2000, p.319). Ehlers & Clark account for this by stating that the stimulus-stimulus (initial trauma – thematically related triggers) and stimulus-response (trigger – affective, cognitive behavioural response) associations generate a very low threshold for retrieval by exposure to external triggers. This means that while the relationship between triggers and initial trauma is often not immediately apparent, the probability of re-experiencing is very high (c.f. Foa & Kozak, 1986). This means that sufferers may lack awareness of why the experience is being triggered, and this prevents them from learning that the triggers are not dangerous. When the memories are activated, symptoms of re-experiencing, hyper-arousal, and trauma-specific emotions occur with the sense of current threat, activating associated cognitions and avoidant behaviour patterns that are executed to eliminate the threat, and therefore the distress, but unfortunately prevents any cognitive processing, maintaining the symptoms.

1.3.4.2.3. Critical Analysis

Ehlers & Clark (2000) provide an excellent account of PTSD *psychopathology* by fully explaining the symptoms, the source and role of cognition, how meanings are generated or changed, and also specifies the role of beliefs and coping systems of the individual on their cognitive processing of trauma, which, in turn, allows for the presence of peri-traumatic emotions, and secondary emotions. In keeping with the cognitive tradition, Ehlers & Clark's approach facilitates assessment and formulation of *individual differences* and needs in a treatment plan. Individual vulnerability factors, peri and post-traumatic cognitions/ appraisals (including mental defeat) are accounted for in the intervention. More specifically, Ehlers & Clark elaborate on the effect of cognitions on processing and provide many idiosyncratic

examples that lead to persistent PTSD, including the function of poor support networks as a maintainer of PTSD symptoms.

Ehlers & Clark's (2000) provides a strong account of the *course* of the PTSD syndrome as a normal response to trauma, but that actual PTSD is characterised by this syndrome being processed (and re-lived) in a way that is systematically different from those who recover normally. What determines the course taken is whether PTSD sufferers process the trauma in a way that appraises the symptoms as dangerous and then use avoidance strategies to systematically ward off the symptoms, which then prevent them being processed. Thus, the course of persistent PTSD symptoms is more than adequately explained in this approach by the *individual differences* in appraisals and strategies used to ward off the experience of PTSD.

Ehlers & Clark's (2000) approach, like Foa & Rothbaum's (1998), is also linked to treatment protocols, which, as a consequence, lends a substantial account *of treatment efficacy*. In broad terms, both emotional processing theory and cognitive theory share the view that the PTSD syndrome is maintained by the perception of current threat and resulting strategies to control the threat and the symptoms. The main distinction is that the strategies covered by Foa & Kozak (1986) are overt behavioural strategies, whereas Ehlers & Clark (2000) focus on covert cognitive strategies. Due to the emphasis on cognitive procedures, this model is primarily a 'maintenance cycle breaker' regarding the reflexive relationship between symptoms, appraisals, and maladaptive coping strategies. But this approach has been criticised for conceptualising avoidance as the result, rather than cause, of distress (Cahill & Foa, 2007). In terms of *theoretical power*, the cognitive model is similar to DRT in that it acknowledges two different types of memory, albeit under different but related terminology (data-driven [situationally-accessible] / conceptual-evaluative [verbally-accessible]), and therefore inherits the explanatory power of that approach. However, it also builds on that power by introducing two interactive processes (representation and appraisal) which are not fully represented in other approaches. DRT does explain appraisal being a result of lack of autobiographical context, but the cognitive approach enriches this and conceptualises appraisals as having a similar cause, but also mediated by the pre-trauma history and coping strategies of the individual. This gives the theory a remarkable flexibility that is lacking in the other approaches. However, the cognitive approach does not seem to specify the role of schemas or the dual representation in memory that the emotional processing theory (Foa & Rothbaum, 1998) and dual representation theory (Brewin et al., 1996) do. The *theoretical power* of this model is unclear in places, as even though the cognitive approach acknowledges its evolution from the previously discussed approaches (Ehlers & Clark, 2000), it has been argued elsewhere that they actually provide different predictions (Brewin & Holmes, 2003): in cognitive theory, the processing of trauma in a data-driven fashion is a predictor of persistent PTSD, yet according to DRT, data-driven processing of trauma (from the conceptually-identical SAM representation) is said to facilitate recovery. However, it is arguable that the mechanisms and resulting predictions are the same provided that the processing is done with autobiographical context in mind: Ehlers & Clark (2000) predict that processing trauma data per se only reinforces the nature of the memory by strengthening stimulus-stimulus-response associations and perceptual priming, but that processing the data in relation to the self improves autobiographical integration. Brewin et al., (1996) predict the same outcome by processing SAM's verbally in working memory to facilitate integration.

1.3.5. Discussion

1.3.5.1. Psychopathology

All theories can account for the symptoms, but via several routes and/or different levels of cognitive representation. SRT conceptualises the core PTSD presentation as the operation of a homeostatic mechanism that slowly integrates traumatic experiences into discordant schemas. The associative networks approach, however, conceptualises the PTSD presentation as the result of highly associated stimulus-response elements with very low thresholds for retrieval that activate escape-avoidance behaviours. DRT explains PTSD symptoms as the result of distinct associative memories that have not been processed into autobiographical memory, and the cognitive model, has adopted a synthesised view of emotional processing and dual representation theories. All theories agree that re-experiencing is central to processing, which is also prevented by avoidance. But the theories' view the role of avoidance as either being adaptive (Horowitz, 1986), or maladaptive (Foa & Kozak, 1986; Ehlers & Clark, 2000). However, subtle commonalities between each theory suggest that avoidance is primarily an adaptive response to real danger, but the symptoms are not dangerous. In SRT, avoidance is a finely balanced mechanism that regulates the rate of processing, and it is only when overactive avoidance is used that PTSD ensues. The role of re-experiencing is seen differently in each theory. The re-experiencing in SRT is a function of steadily incorporating discordant information into working models of relating to the world and the self, whereas emotional processing theory sees the re-experiencing as being the function of an especially strong and overactive network of stimulus-response pairs. Similarly, DRT and cognitive theory see the re-experiencing symptoms as belonging to a specific form of associative memory structure that needs to be processed in relation to the self. The common theme between these theories is that re-experiencing symptoms are stimulus-triggered, low threshold, and need to be processed into autobiographical context. However, the account of cognitions is only robust in later editions of EP theory (Foa & Rauch, 2006; Foa & Rothbaum, 1998), and in cognitive theory (Ehlers & Clark, 2000), but these theories are linked in with specific therapies, whereas the others are focuses on providing explanatory power to the mechanisms thought to be active during the emotional processing of traumatic events. The role of emotions other than fear has only been captured in EP theory (Foa & Rauch, 2006; Foa & Rothbaum, 1998), dual representation (Brewin et al., 1996), and cognitive theory (Ehlers & Clark, 2000). However, although their presence is explained within the broad canvas of the trauma experience (such as primary and secondary emotions in the respective SAM/VAM counterparts [Brewin et al., 1996] or how appraisals of trauma, the self, and one's relationships affect emotions [Ehlers & Clark, 2000]), it is less clear how these emotions (or their avoidance) relate to the development and maintenance of the syndrome. As discussed in earlier sections, fear (while clearly a central feature of PTSD) is not the only emotion connected to flashbacks. These theories can provide exceptionally robust accounts of how fear generates flashbacks, but not of other contributing emotions.

1.3.5.2. Course of the Syndrome

In terms of how course is explained, predicted, and represented, all models express the notions that 1) recovery is the norm as demonstrated by a gradual state of declining emotional disturbance, with an associated increase in functionality (c.f. Rachman, 1980; 2001); and that 2), avoidance of this process acts

as a maintainer of distress. As a corollary, dual representation, emotional processing, and cognitive approaches all share the view that recovery is achieved by inhibiting avoidant strategies to facilitate processing. Similarly, these approaches assert that recovery is achieved not when old associations are eliminated, but when new associations override the old. Therefore, the shared clinical implications are that all trauma-related information contained in the fear structure, SAM, or associative memory, must be better organised and elaborated in order to suppress old associations. Any trauma engrams that remain unprocessed put the sufferer at risk of future PTSD due to the reactivation of unprocessed trauma. This is both a powerful generalisation and predictor of therapeutic change, as it places precedence on full emotional engagement in order to process all material into context, and protect the trauma survivor against relapse. All trauma-focused therapy is centred on this approach. This is revealing, in that it follows that less obvious individual factors which impair their capacity to engage should be a central focus of treatment.

1.3.5.3. Individual Differences Affecting Presentation & Course

It is apparent from these theories that the PTSD syndrome is considered a healthy adaptive response when re-experiencing is associated with a decrease in symptoms and an increase in functionality – but this is not full PTSD. All the approaches deal with broad fundamental strategies which initiate non-normal course diversions and emotional processing (e.g. behavioural avoidance, [Horowitz, 1986; Foa & Kozak, 1986]; cognitive appraisal, Ehlers & Clark; chronic and inhibitive processing [Brewin & Holmes, 2003]). Therefore, there are other contributory factors that prevent the normative course of emotional processing. Factors such as the role of 1) pre-trauma schemas as vulnerability, or resilience, factors; 2) the role of an individual's appraisal of symptoms on their severity and chronicity; 3), the effect of the sufferer's support network on their appraisals, have largely been accounted for the cognitive model (Ehlers & Clark, 2000). However, while the cognitive approach is designed to provide idiosyncratic formulations designed to break individual maintenance cycles, like the other theories, it provides no robust account, or predictions, for the role of peri-traumatic (or even post-traumatic) dissociation on PTSD chronicity. Similarly, there are few specific predictions about the role of other emotions or experiential avoidance strategies.

Similarly, it is evident that these theories do not sufficiently account for the subtle differences in strategy or interpersonal circumstances that can hinder emotional processing, and have less power to predict the effect of intrapersonal and interpersonal factors on prognosis. Given that emotional processing is a fundamental part of exposure for anxiety disorder, and that emotional engagement is key to facilitating this process, it is interesting that cognitive therapy for anxiety disorder has been criticised for not sufficiently addressing the role of experiential avoidance in therapy (Leahy, 2007; Newman et al., 2008). Several papers document the role of emotion schemas (Leahy, 2002; 2007), shame (Lee, Scragg, & Turner, 2001), and fear of severe anxiety (Falsetti et al., 2008) in the maintenance and treatment resistance of anxiety disorders, particularly PTSD.

1.3.5.4. Theoretical Power

However, cognitively representing the role of emotion schemas and predicting their impact on PTSD presentation and course may be extremely difficult. The most powerful PTSD theories contain multiple levels or representations: 1) SAMs, 2) VAMs and 3) conscious cognitive processes. SAMS represent unprocessed, non-verbal sensory memories, VAMS represent processed, verbal, temporally-contextual

and autobiographical memories, and cognitions represent conscious appraisals of trauma and symptoms. Emotion schemas do not appear to fit in any one level of representation. They cannot be merely SAMs, because, beliefs about emotion can be verbally communicated, and changed, yet they cannot merely be VAMs, because schemas operate out of conscious awareness, and as in the case of persistent depression, complex PTSD, and personality disorders, are very hard to change. If research-clinicians do not know where schemas sit in the theoretical model, then their effect on emotions, and strategies used to control them, cannot generate meaningful predictions.

This does not, however, imply the theories are impoverished. Dalgleish (2004) argued that increasing the levels of mental representation improve explanatory power, but also reduce parsimony, and require that the increasing number of interactions between elements of the representational model be demonstrated empirically. This presents a conundrum when attempting to generate complete theories, as traumatic experience, the PTSD syndrome, its course, and the mediating effect of intrapersonal/interpersonal variables and socio-economic systems on course and prognosis is inordinately complex. Attempts to generate a universal model of schematic, associative, and verbal representations have been put forward (Dalgleish & Power, 2004), but are difficult to apply in clinical practice. McKeever & Huff (2003) highlighted similar issues surrounding the intrinsic difficulty of introducing systemic variables into a diathesis-stress framework for PTSD. There are many mediating and moderating variables that qualify as individual differences affecting presentation, course and prognosis; adding these components, while increasing predictive power, would make theories inordinately complex and non-parsimonious, without taking into account that these variables may be proxies of underlying constructs (Kraemer et al., 2001). A complete theory of PTSD should be able to explain and predict the effect of key individual differences on presentation, course, and recovery.

1.3.6. Conclusions

All theories readily account for the psychopathology of the PTSD syndrome, its course, the efficacy of trauma-focused treatment, and provide some reasonable explanatory power for individual differences in variables moderating or mediating recovery and chronicity, and they do it in a complementary fashion at different levels of cognitive representation: from shattered or reinforced core beliefs residing in latent schemas about personal safety and competency (Epstein, 1985; Horowitz, 1986; Janoff-Bulman, 1992), to subsequent over-activated cognitive-behavioural-affective networks employed to deal with danger related to personal competency and safety (Foa & Kozak, 1986; Foa & Rothbaum, 1998; Rauch & Foa, 2006), to the processing of raw, under-elaborated and un-communicated trauma memories from a sensory-affective level of representation to a cognitive-linguistic-autobiographical level of representation (Brewin, et al., 1996), to the higher-level cognitive appraisals of PTSD symptoms that maintain and propagate the syndrome (Ehlers & Clark, 2000). However, the role of schemas in PTSD vulnerability and maintenance appears restricted to beliefs regarding the world being universally dangerous and the self being powerless, and the resulting emotion of fear. Though this may predict how people respond to danger, and how they also react to their own experience of fear (by using basic avoidance behaviours), it does not account for the subtle evasion of additional emotional experience that occurs at a cognitive level (be it via rumination,

worry, or cognitive suppression), and how this itself may predispose trauma survivors to PTSD and contribute to its maintenance.

1.4. The Role of Emotion Schemas and Avoidance in the Inhibition of Emotional Processing

Previous research has shown that individual variables provide very little power to predict who will develop PTSD following a traumatic event. However, diathesis-stress frameworks for PTSD (McKeever & Huff, 2003) point to the pivotal role of ecological diatheses where the traumatic event, post-trauma social support, and pre-trauma cognitive vulnerability, interact to generate the risk of subsequent PTSD. It is well documented that an individual's adjustment during stressful events is related to cognitive processing styles and appraisals of their experience. These styles can act as protective or predisposing factors towards PTSD. Cognitive theories suppose that cognitive processing biases affect attention, memory, and affect what information is processed (Buckley, Blanchard, & Neill, 2000). Cognitive vulnerability to PTSD is central to its maintenance due to the way that PTSD sufferers chronically appraise and process their distress compared to those without PTSD (Ehlers & Clark, 2000). It is this cognitive vulnerability that is central to the clinical theories for PTSD. These theories can account for the distinguishing PTSD symptom profile (intrusions/cognitions, avoidance, hyperarousal), the major course diversions of the syndrome, and inform the evidence-based trauma-focused interventions which have generally proven effective at facilitating the course of memory processing. However, they are less robust in their ability to represent and predict the role individual of beliefs and cognitive strategies *about the experience and control of emotions* in the maintenance of the PTSD, and how they may negatively influence the ability of a patient to emotionally process their trauma. It has been argued that cognitive-behavioural approaches focus on changing, rather than embracing, internal experiences, and as such, have substituted maladaptive experiential avoidance for evidence-based emotion-regulation strategies (Hayes et al., 1996). Though emotion regulation is certainly helpful in discrete circumstances, variables centred on control of emotional experience can become problematic when they are controlled and avoided to the point that they do not enter awareness and thus cannot inform action and change (Greenberg & Safran, 1989). However, this is becoming increasingly recognised as a trans-diagnostic problem that has implications for effective therapy.

The aim of this chapter is to explore and review the evidence for how a person's beliefs regarding the experience and expression of negative emotions, and the strategies used to regulate them, may prevent engagement, stall processing, and thus predispose and maintain anxiety disorders, including PTSD. In order to understand this process, this chapter will define emotional processing and emotion schemas, and explore how they may influence the effectiveness of prolonged exposure therapy.

1.4.1. Emotional Processing: Definition & Hypothesised Mechanisms

Generally speaking, emotional processing is a forward-moving cycle that occurs when individuals go through distressing life events and experience the personal meanings and concomitant emotions related to the event. Eventually, the experience is absorbed and declines in frequency and intensity, and the

individual moves towards normal levels of functioning (Rachman, 1980). While the majority of individuals will fully process their distressing experiences, a minority will not complete the process and continue to suffer the intrusions (e.g. nightmares/flashbacks/intrusive imagery), and hyper-arousal symptoms (e.g. restlessness and irritability) that should have otherwise been absorbed (Rachman, 2001). This processing failure can manifest in unresolved grief, anger, depression, and anxiety disorders.

Many theorists have converged on a conceptualisation of emotional processing that involves two systems. These have been described as schematic and conceptual (Leventhal, 1979), experiential and rational (Epstein, 1994), or subsymbolic and symbolic (Bucci, 1997, 2001). Recently, theories on PTSD have described situationally-accessible and verbally-accessible memories (Brewin et al., 2003), or data-driven and conceptual-evaluative systems (Ehlers & Clark, 2000). The common distinction between these systems is that they store information differently. The first system is automatic, associative, and non-verbal, is experienced via bodily sensations, processes vast quantities of information using priming and spreading activation, and it is triggered by internal/external sensory stimuli. In contrast, the second system is verbal, declarative, temporally contextual, and is wilfully accessed via higher executive functions. All theories that refer to these two systems imply that the emotional processing cycle is the manifestation of transferring (or copying) experiential information from one system to the other (Brewin et al., 1996; Bucci, 1997; 2001; Gendlin, 1982), and that full emotional engagement is required to complete the process (Rauch & Foa, 2006). This process, while necessary, is also unpleasant and overwhelming, and contains homeostatic fail-safes to manage the level of arousal to facilitate the steady processing of distress (Horowitz, 1986). The rationale for exposure therapy is that facilitating this process promotes recovery (Hunt, 1998; Whelton, 2004) because it serves as a consistent predictor of improvement in therapy (Castonguay, Goldfried, Wiser, & Hayes, 1996; Greenberg & Safran, 1987; Orlinsky & Howard, 1986). However, initial problems experiencing and processing emotional states predict a lack of engagement and therapeutic change (Klein et al., 1969; Watson & Bedard, 2006).

Emotional processing mechanisms have received the most attention in the theory behind the aetiology, symptomatology, course, and treatment of PTSD (Foa, Hembree, & Rothbaum, 2007; Feeny, Zoellner, & Foa, 2002; Rauch & Foa, 2006). Though the normative course is one of decreasing psychological distress and increasing personal functionality (Rachman, 2001), those who are clinically distressed and suffer the symptoms of incomplete emotional processing are being impaired in their ability to process these experiences - and therefore cannot engage in therapy.

1.4.2. Emotion Schemas

According to Greenberg & Safran (1987), the therapeutic process "…requires both the activation of existing emotion schemas and the generation of new information with which to reorganise the existing emotional processing network" (p.265). In addition to the definition of schemas in chapter 1.3., Leahy (2002) states emotion-schemas in particular are a subconscious cognitive program consisting of a set of beliefs, emotions, and behaviours, that are used to inform and conduct one's relationship to their own, and another's, emotions. Similarly, Gottman et al., (1997) suggests that out of emotion schemas emerge several different philosophies: 1) *emotion coaching*, which facilitates expression, investigation, problem solving, and is validating, and intimacy-generating; 2) *dismissive*, where emotions are seen as harmful,

often unhelpful, and therefore should be avoided, and 3) *disapproving*, where emotions are viewed as inappropriate and invalid, and as such should be reprimanded if expressed. These philosophies promote either the approach or avoidance emotional experience, and as such may have an impact on therapeutic engagement. Leahy (2007) argued that fearful attitudes towards emotional experiences are the overarching feature of all anxiety disorders, and influence the degree of therapeutic engagement. Those who have positive attitudes toward emotion will approach them and engage, whereas those who do not will avoid and disengage. To that end, working on an individual's emotion schemas may be essential to effective exposure therapy for anxiety disorders.

1.4.3. Exposure Therapy

Exposure therapy (as explained in Chapter 1.3.) is a core therapeutic approach that is used to treat all anxiety disorders. It is highly effective at treating simple phobias (Marks, 1979), panic disorder (Baker, 2011), and for treating PTSD (Foa & Rothbaum, 1998; Taylor et al., 2003; Van Etten & Taylor, 1998), and has been used with repeated success (Foa & Meadows, 1997). But despite the efficacy of exposure for PTSD specifically, a significant proportion of patients do not respond to treatment (53%; Resick et al., 2002). A number of studies call into question its effectiveness in practice, including problems with therapeutic engagement (Foa, Riggs, Massie, & Yarczower, 1995; Perconte & Groger, 1991; Pitman et al., 1991; Tarrier, Sommerfield, Pilgrim, & Faragher, 2000), and drop-out rates between 14% and 27% (Hembree et al., 2003**;** Resick et al., 2002). This may be because the presence of some factors are barriers to effective exposure, such as emotional numbing and extreme anxiety (Jaycox & Foa, 1996), and reluctance to re-experience trauma memories due to beliefs that exposure will increase the severity of PTSD symptoms (Cook, Schnurr, & Foa, 2004). This does not mean that exposure is inappropriate in these cases (Feeny, 2003), but rather that it is being applied to a particular sub-population of patients who cannot engage with the therapeutic approach (Baker, Gale, Abbey, & Thomas, 2013).

1.4.4. Emotional Processing in PTSD with Co-Morbid Disorders

The DSM-IV-TR diagnostic criteria for PTSD (APA, 2000) is known for its co-morbidity with other disorders, having been shown to overlap with panic disorder (PD), generalized anxiety disorder (GAD; Friedman & Yehuda, 1995), and major depression (Brady et al., 2000; Campbell et al., 2007; Shalev et al., 1998). The numbing symptoms of PTSD (Criterion C4) can reflect avoidant symptoms in major depression (Criterion A2). Panic disorder, while not specifically reflecting PTSD criteria, does clinically present with overlapping features as over 50% of trauma survivors experienced peri-traumatic panic attacks, and re-experiencing the memories of these traumas can cause panic attacks to return **(**Bryant & Panasetis, 2001; Nixon & Byant, 2003).

Falsetti, Resnick, & Davis, (2008) argued that the presence of co-morbid panic disorder impaired the ability of the individual to engage in trauma-focused therapy. Panic attacks are generally triggered by 'false alarms'. But a 'true alarm' in PTSD (such as a traumatic event) can trigger a peri-traumatic panic attack. The chronic hyper-arousal intrinsic to PTSD increases their vulnerability to panic as they closer to the arousal threshold. An additional feature of panic disorder is that the individual does not link the panic

attack to internal cues, which makes the sensations incomprehensible and frightening. This may result in common cognitions such as *"the attack came out of nowhere"* and *"I am going crazy"*, which reinforce the perceived unpredictability, incomprehensibility, and catastrophic consequences of experiencing fear. This leads those with panic to control it by avoidance, which may interfere with the cognitive processing of the trauma (Falsetti et al., 2008). However, additional research shows that panic sufferers may attempt to control other emotions as well. Baker et al., (2004) compared a panic disorder sample (n = 48), with two groups of healthy controls (n = 531) on how they respond to anxiety, sadness, and fear, and also the strategies used to control them. They found that those with panic disorder were more consciously aware of their feelings, and had a greater tendency to control anger, sadness, and anxiety, especially through the use of cognitive suppression - but also had much greater difficulties identifying their emotions.

Similar features of emotional control and poorer awareness have been recognised in individuals with GAD. GAD is typically conceptualised as a disorder of persistent 'free-floating' worry (APA, 2000; 2013) - a cognitive strategy that is used to avoid emotional arousal triggered by external stimuli (Behar et al., 2009). Avoidance models postulate that people with GAD typically find emotions unpleasant and attempt to avoid them (Borkovec, 1994; Borkovec & Newman, 1998), or control them (Turk, Heimberg, Luterek, Mennin, & Fresco, 2005), by using worry as a distraction (Borkovec & Roemer, 1995). This reduces the experience of anxiety, but in doing so, prevents the experience and subsequent processing of the underlying fear (Borkovec, 1994). However, the initial worry approach is often ineffective, and becomes debilitating, leading GAD sufferers to worry about the experience of worry - this is otherwise known as the meta-cognitive model (Wells, 1995). Similar to those with panic, people with GAD begin appraising the worry as *"uncontrollable"* and *"dangerous"*, and seek to avoid worry via thought-suppression and re-assurance seeking, and it is this 'meta-worry' that distinguishes GAD from other sub-clinical presentations.

1.4.5. The Clinical Implications of Covert Emotion Avoidance on the Effectiveness of Exposure

The implications of such covert emotion avoidance are trans-diagnostic in nature. Cognitive Behavioural Therapy (CBT) for GAD is far less effective than for the other anxiety disorders (Borkovec & Ruscio, 2001), possibly because the CBT paradigm does not treat the subtle emotional avoidance factors that maintain GAD (Newman et al., 2008). Similarly, exposure for PTSD may be less effective if panic disorder is present due to the presence of beliefs relating to the control of anxiety (Falsetti, et al., 2008). Generally speaking, the cognitive model states that beliefs influence the emotions a person feels, and that emotions predict cognitive biases that affect information processing and memories that are activated, but much less is known about the effect of meta-cognitive beliefs regarding how people process the experience of aversive events (Leahy, 2002; 2007). If the fear of unpleasant psychological experiences (and the attempts to control/avoid them) predisposes people to panic attacks or worry, which can then interfere with emotional processing, then this presents a problem for any exposure-based treatment. The effectiveness of exposure therapy rests on the prolonged presentation of stimuli that activates 'fear structures'. The use of physiological habituation, and the systematic insertion of new, corrective experiences, introduces new information to the structure, reduces its ease of activation, and the intensity and duration of concomitant fear (Foa & Kozak, 1986; Lang, 1977). This process requires full emotional engagement.

Consequently, the effectiveness of exposure is compromised under a number of conditions: 1) failure to engage *emotionally* with the memory by using avoidance strategies (Foa & Cahill, 2001); 2), failure to process fear due to under-engagement, and therefore insufficient activation of the fear network (Foa & Kozak, 1986); or 3), over-engagement, which generates extreme fear, and instigates information-processing biases which inhibit the integration of corrective information (Rauch & Foa, 2006). PTSD patients with undetected co-morbid panic features may enter into exposure and become over-engaged, and therefore disengage without experiencing the necessary habituation. Similarly, it is possible that PTSD patients who use cognitive avoidance strategies (such as worry or rumination) may actually be insufficiently engaged as to begin emotional processing at all. Taking these conditions into account, exposure may be unfeasible if the individual becomes fearful of the physiological sensations, and focuses on the experience of fear rather than the memories. Therefore, a clinician would have to treat the phobia of unpleasant emotions, before commencing trauma-focused therapy.

1.4.6. The Effect of Emotion-Regulating Behaviour on Psychiatric Distress

A common feature between GAD and PD is the tendency to fear the experience of anxiety and its concomitant sensations, otherwise known as anxiety sensitivity (Reis, 1991). The fear of emotions has long been implicated in general distress and the effectiveness of therapy (Gendlin, 1982; Leahy, 2007). If people hold negative beliefs about the consequences of experiencing or expressing emotions, they may employ idiosyncratic emotion-regulation strategies that minimise the threat associated with that experience. Consequently, the role of beliefs surrounding the experience and expression of emotions, and the subsequent strategies used to control them - be they behavioural (avoidance, safety behaviours), or cognitive (e.g. rumination, suppression), may be jointly implicated in the development and duration of distress.

Strategies such as rumination involve the chronic, repetitive, and negative, focus on the causes and consequences of psychological symptoms (Nolen-Hoeksema, 1991), whereas looming cognitive style is an attention and cognitive appraisal bias whereby an individual generates, maintains and cognitively attends to mental representations of escalating danger and risk, and is a cognitive vulnerability specific to anxiety (Riskind, 1997; Riskind, Williams, & Joiner, 2006). Aldao, Nolen-Hoeksema & Schweizer (2010) examined the relationship between six dispositional cognitive strategies (rumination, avoidance, cognitive suppression, acceptance, cognitive appraisal, and problem solving), and psychiatric symptoms of depression, anxiety, eating disorders, and substance abuse. After conducting a meta-analysis of 241 effect sizes from k=114 studies, they found that the largest effect size was for rumination, with medial effect sizes for avoidance and suppression, and the smallest effect sizes for reappraisal of acceptance and problem solving. Aldao & Nolen-Hoeksema (2010) later used factor-analysis to examine the relationship between four of these cognitive emotion regulation strategies (rumination cognitive suppression cognitive appraisal and problem solving), and psychiatric symptoms of depression, anxiety, and eating disorders, in a sample of undergraduate students (n=252). They found that the maladaptive strategies (rumination and suppression) were more highly correlated with psychiatric symptoms than were the adaptive strategies (re-appraisal and problem-solving), and were also significantly correlated with all three disorders. Among their most interesting observations were that maladaptive cognitive strategies carried a far greater

variance contribution across psychological disorders then the use of adaptive strategies (Aldao et al., 2010). These are important findings given that cognitive therapy typically emphasises practising new adaptive strategies and implies that emotion regulation strategies have trans-diagnostic value, and are an appropriate target for intervention (Aldao & Nolen-Hoeksema, 2010).

1.4.7. Emotion-Regulation Training as an Adjunct to Therapy

Emotion regulation strategies have since been investigated as a target for intervention. A key study by Berking et al., (2008) aimed to establish the clinical utility of emotion regulation skills training prior to commencing CBT. Using the Integrative Training of Emotional Competencies [ITEC] program (which trains participants in positive regulation strategies such as non-judgemental awareness, acceptance, tolerance, self-compassion), they used a sample of 289 patients who were set to receive cognitive-behavioural treatment for a range of disorders including dysthymia (2%), major (25%) and recurrent (23%) depression, panic disorder (3%), adjustment disorders (22%), and PTSD (3%). The clinical sample was randomised into ITEC and non-ITEC groups, and compared to 246 nonclinical controls. The results showed that accepting and tolerating emotions was a significant component of treatment outcome and that replacing parts of the CBT protocol with emotion regulation actually augmented its effects.

1.4.8. Emotion Regulation in PTSD

Very recent studies reveal a positive relationship between PTSD symptom severity and a fear of the consequences of experiencing and expressing emotions themselves, including a lack of emotional acceptance, emotional clarity, and emotional dysregulation that impairs goal-directed behaviour during distress (Farnsworth & Sewell, 2011; Price et al., 2006; Tull, Barrett, McMillian, & Roemer, 2007). In one study, individuals who were exposed to Criterion A level traumas and who had PTSD, had significantly more difficulty regulating and accepting their emotions compared to those who a) did not experience criterion A traumas, and b) those who did meet criterion A but did not have PTSD (Weiss et al., 2012). In addition, difficulties regulating emotions, and the absence of social support, have been shown to mediate the link between childhood abuse and adult PTSD (Stevens et al., 2013). An earlier study also revealed that PTSD re-experiencing symptoms were positively correlated with a) difficulties labelling emotions, and b) depression and emotional numbing symptoms (Monson et al., 2004). This suggested that problems identifying emotions may be the result of emotional numbing symptoms that reduce the ability to label and regulate these emotions, and that clinicians should be encouraged to formulate adjuvant emotional processing therapies to address these inhibitive emotional states (Monson et al., 2004). Since then, adjuvant interventions such as Multiple-Channel Exposure Therapy (M-CET) for PTSD with co-morbid Panic Disorder (Falsetti et al., 2008), Acceptance and Commitment Therapy (ACT) for PTSD (Varra & Follette, 2004), and emotional regulation/preparation stages for complex PTSD (Cloitre et al., 2002; Cloitre et al., 2010), have also been added to trauma-focused treatment packages to improve engagement and treatment efficacy.

In recent years, this has led to a synthesis of emotion processing research that accounts for the role of emotion schemas in predicting emotion avoidance/regulation styles and how their use prevents the

processing of emotional distress, predispose people towards alexithymia, and hinders therapeutic engagement (Baker, 2007). This model has been used successfully in the assessment and formulation of treatment for panic (Baker, 2011) and been used to restructure prolonged exposure therapy into an emotional framework that, like Cloitre's et al. (2002; 2010), and Berking et al.,s (2008) work, is preceded by pre-therapy preparation stages. This new model of emotional processing, and approach to prolonged exposure, has been applied to PTSD to adapt trauma-focused for those individuals who may not be able to fully engage (Baker, Gale, Abbey, & Thomas, 2013).

1.4.9. The Emotional Processing Model

In Baker's (2007) model, the onset of an emotional experience starts with a precipitating input event. This event has to be registered, either consciously, or unconsciously. This event may be a minor event, or a major traumatic event such as a road-traffic accident, or a protracted stressful event. In this model, the cognitive appraisal (that is, what the event means to the 'experiencer') is what determines the emotion experienced. Several factors affect processing at this stage – for example: failure to register the event (whether one is conscious of the event, or not); misinterpretation of the event due to incorrect appraisal, or appraisal influenced by past memories of similar 'aversive' experiences; or active avoidance of any potentially threatening event (such as avoiding thinking about, or being in the presence of, the 'aversive' trigger).

After the input event, the individual experiences the emotion elicited by the appraisal of the precipitating event. However, the use of experiential avoidance through suppression (the conscious attempts to smother out the emotional experience itself) and avoidance (re-directing attention from the 'aversive' emotional response, or from stimuli/situations that can elicit that response) can prevent a fully integrated emotional experience. Deficits in emotional experience include: the failure to experience the emotion as a psychological whole; deficits in the awareness of emotional experience; and difficulties in labelling the emotion, which can make it difficult to link the emotion to the triggering event. During, and after the emotion is experienced, the emotion is often expressed. Difficulties that arise in emotion expression are over-control by the suppression of expression, or the failure to regulate emotions; this failure to regulate emotions is often a sign of unprocessed emotional experiences.

According to Rachman's (1980, 2001) conceptual framework, unprocessed emotional experiences manifest through the presence of persistent and intrusive emotional experiences. An unprocessed emotional experience may manifest as "…obsessions, flashbacks, nightmares, pressure of talk, inappropriate expressions or experiences of emotions that are out of context… [or] …proportion…" (Rachman, 2001, p.165). Maladaptive emotional control mechanisms (such as avoidance and suppression), deficits in control (dysregulation), emotional experience, and signs of incomplete processing are factors in Baker's (2007; 2010) emotional processing model, and are measured in the most recent version of the Emotional Processing Scale (EPS-25; Baker et al., 2010), which has demonstrated sensitivity to therapeutic change in clinical samples undergoing CBT (Baker et al., 2012).

1.4.10. Emotional Processing Therapy for PTSD

Emotional Processing Therapy [EPT] was developed from Baker's emotional processing model (2007), which draws on and synthesises concepts of emotional processing (Rachman, 1980; 2001) physiological habituation (Marks, 1979), emotional processing theory (Foa & Kozak, 1986) experiential focusing (Gendlin, 1982) and multiple code theory (Bucci, 2001). It's mechanisms of action are based on emotional processing theory, and therefore also involve all the elements of prolonged exposure, and is thus similar to other NICE-recommended trauma-focused therapies to date (NCCH, 2005). However, EPT employs a richer approach to exposure by drawing from experiential focusing and multiple code theory to understand how unprocessed emotional information is represented in the body sub-symbolically (that is, a non-verbal felt meaning with no referent) and how to facilitate processing it to a conscious symbolic level (verbal).

EPT approaches the practice of prolonged exposure from the context of pre-existing, and unhelpful, emotional processing and coping styles that according to Baker not only are a contributing vulnerability factor in the risk of developing PTSD, but also preclude the processing of distressing memories and the associated affect, maintaining the disorder. The aim of emotional processing therapy is therefore not only to enable the processing of current trauma, but (in agreement with Leahy, 2007) to also modify emotional processing styles to facilitate full, effective, engagement in the exposure, and carry this new strategy into their lives after therapy. Several sessions are dedicated towards implementing ways to adapt and change these styles for the ease of processing current and future emotional distress. EPT operates on the assumption that prolonged exposure therapy is, at its core, exposure to emotional experience, rather than a purely behavioural exposure, because the act of confronting previously avoided distressing memories allows the patient to be exposed to powerful and distressing emotional experiences. At this point, memories can then be processed effectively to the point that the recall of memories no longer disrupts day-to-day functioning.

1.4.11. Conclusions

The course of emotional processing is a central feature in the psychological adjustment to distressing experiences. However, disruption of this process can result in, and contribute to the maintenance of, anxiety disorders. A person's beliefs regarding the experience and expression of negative emotions can motivate them to use strategies that facilitate emotional avoidance, which can impair their therapeutic engagement. Research to-date reveals that emotion schemas (and resulting processing styles) are central to anxiety disorders, including PTSD, and that they may be responsible for a significant degree of treatment resistance (Baker, 2007; Baker, Gale, Abbey, & Thomas, 2013; 2010; Leahy, 2007).

1.5. Cancer-related PTSD (CR-PTSD): Prevalence and Predictive Factors

In Table 1, it is explained that according to DSM-IV-TR criteria (APA, 2000), PTSD occurs when the individual experiences (or witnesses) a traumatic event that threatens their psychological and/or physical integrity (Criterion A1), and they react with helplessness or horror (Criterion A2). Later, they re-experience the memories, sensations, and emotions from the trauma through sensory flashbacks, and/or

nightmares (Criterion B). The sufferer avoids reminders of the trauma, can be emotionally numb (Criterion C), anxious, irritable, and hyper-vigilant (Criterion D). While not intrinsically pathological, this becomes problematic when the duration of the distress is more than one month post-trauma (Criterion E), and causes serious socio-occupational impairments (Criterion F). Traumatic experiences that generate these responses involve, but are not limited to, prolonged combat exposure, natural disasters, physical/sexual assault, and medical illness. The traumatic nature of medical events has been the focus of clinical attention and researched extensively (Tedstone & Tarrier, 2003), but much of the research has been dedicated to cancer. Cancer itself is common, with 33% of the UK population receiving a cancer diagnosis within their lifetime; and of this population, 50% will reach five-year survivorship (Cancer Research UK. 2013).

Cancer is a disease of rapid, uncontrolled, and abnormal, cell division that causes tumours, and progresses through several stages of growth and spread. The severity of the cancer is determined by the size of the primary tumour (T), the degree of progression into neighbouring lymph nodes (N), and the presence/absence of secondary tumours (or metastasis) in other parts of the body (M). These three factors comprise the TNM staging system, and varying combinations of TNM inform the overall stage of cancer progression. Generally, at Stage I, the tumour is restricted to the initial site, but this tumour will have begun to damage adjacent cells within the tissue. By Stages II-III, the single tumour has advanced to overtake a significant area or the majority of the affected tissue, and by Stage IV, it has gained the ability to metastasize, either within the originating organ, or into other organs via transmission through the bloodstream or the lymph nodes (Bruce, 2006; Kangas, Henry, & Bryant, 2002; Klein, 2008). It is at Stage IV that the cancer is often widespread, and prognosis is poor.

There are several different cancers which vary between them in terms of growth rate, affected cell type, symptomatology, and response to specific medical treatments. The available medical treatments for cancer include chemotherapy, radiotherapy, immunotherapy, hormone therapy, and surgery (both tissue removing and tissue conserving), and are often combined depending on cancer type and growth rate. Surgery can have permanent side effects (Steinlin, et al., 2003; Turner et al., 2009), as can chemotherapy, which induces hair loss, nausea, fatigue, mood disturbances, lymph oedemas around injection sites (Kangas et al., 2002; Thompson, 2011), sensations of heat, sweating, and palpitations (Lipov et al., 2008; Sturdee, 2008) and cognitive deficits in executive function, processing speed, and memory (Argyriou et al., 2011; Wefel et al., 2004). These side effects can also persist beyond the completion of treatment, and can be unpleasant and debilitating.

Despite this, the advancement of cancer treatments in recent years has improved their effectiveness and doubled the rates of survivorship (Cancer Research UK, 2013). But of those who do survive, a significant proportion is known to suffer a range of persistent and complex psychological difficulties which affects their day-to-day functioning and quality of life. Systematic reviews on the topic show that long-term survivorship is often accompanied by co-morbid depression (Palmer et al., 2004), anxiety (Golden-Kreutz & Andersen, 2004), and in a minority of cases, symptoms of PTSD (Gurevich, Devins, & Rodin, 2002; Kangas, Henry, & Bryant, 2002; Koutrouoli, Anagnostopoulos, & Potamianos, 2012; Smith, Redd, Peyser, & Vogl, 1999) which can manifest post-diagnosis either during or after treatment (Bruce, 2006; Kangas et al., 2002; Nir, 1995; Smith et al., 1999), and sometimes long after treatment is completed (Amir & Ramati, 2002; Cordova et al., 1995).

Theoretically, cancer has the potential to be traumatic on the grounds that a cancer diagnosis and treatment is truly a threat to one's life and physical integrity, and can generate peri-traumatic feelings of helplessness in the face of the disease (Vachon, 2006). This was confirmed in the DSM-IV PTSD Field Trials (Alter et al., 1996), and has since generated a large body of research in cancer populations into the prevalence of cancer-related PTSD (CR-PTSD), and factors contributing to its presentation (Thompson, Eccleston, & Hickish, 2011) to improve the psycho-social wellbeing of cancer patients and survivors (Foster, Wright, Hill, Hopkinson, & Roffe, 2009).

However, the applicability of the PTSD syndrome to the cancer population has been questioned because the prevalence statistics fluctuate widely, revealing that only minority of patients meet the threshold for PTSD caseness, and it is unclear what factors contribute to these rates (Kangas et al., 2002; Thompson et al., 2011). There are also additional problems in reconciling specific elements of the experience of cancer to Criterion A (Kangas et al., 2002); There are issues with detecting CR-PTSD due to Criterion A2's lack of sensitivity, which equally predicts major depression (Palmer, et al., 2004); and also differentially diagnosing CR-PTSD due to PTSD symptoms (Criteria B-D) being confounded by artefacts of the disease, such as a reasonable fear of recurrence over-endorsing intrusion symptoms (FOR; Hodges & Humphris, 2009; Mehnert et al., 2009), or treatment side-effects intrinsic to chemotherapy mimicking symptoms of hyper-arousal due to trauma (Kangas et al., 2002; Shelby, Golden-Kreutz, & Andersen, 2005; Thompson et al., 2011).

The aim of this chapter is to review and evaluate the PTSD epidemiology and risk-factor research in the cancer population, in relation to what is known about PTSD across other trauma populations. This chapter will focus mainly on female breast cancer populations as this is where most of the literature has focused.

1.5.1. Prevalence of PTSD Symptoms in Cancer Patients and Survivors

Alter et al., (1996), in the first major study of cancer-related trauma, assessed 27 cancer survivors from the DSM-IV Field Trials who were three-years post-diagnosis and compared them to a community control sample of people who suffered from other cancers. A statistically non-significant 4% (n = 1) of the cancer sample met criteria for current PTSD *v* 0% of the community sample, possibly due to the sample size for this initial study being extremely low and conducted on a very minor sub-population of an already large DSM-IV Field Trial for PTSD (Kilpatrick et al., 1997). Nevertheless, though the prevalence of cancer-related PTSD was low in this sample, their presenting syndrome was regarded as similar to those who experienced other traumatic events. Of special interest was that a statistically significant 22% of the cancer sample had lifetime PTSD which was specifically attributed to cancer *v* 0% of the community sample, and therefore suggested that at the very least, cancer was sufficiently disruptive as to predispose survivors to future PTSD over and above what would be expected for the normal population. This necessitated further investigation into the syndrome.

Similar prevalence trends were found by Andrykowski & Cordova (1998). They studied a cohort of female stage 0-III breast cancer survivors (n = 82) that were at least 30 months post-treatment. Using the PTSD Symptom Checklist – Civilian Version (PCL-C) and the cut-off scoring method, they yielded a PTSD prevalence of 6%, although interestingly, 13% of the sample has partial/subsyndromal symptoms.

This early study revealed that even though full-PTSD prevalence is low, a sub threshold form of the disorder is very common. Andrykowski et al., (2000) conducted a similar study on another sample of female breast cancer survivors (stages 0-III; *n* = 42) and found that the PTSD prevalence, even after 30 months post-treatment at time 1, and one year later at time 2, was 6.5%, revealing yet another similar prevalence rate. Of interest though is that despite the low conditional risk of PTSD in this sample, those who did have PTSD at time 1 were chronic at time 2.

Using a similar sample, Cordova et al (1995) conducted a cross-sectional study on a cohort of female stage I-III breast cancer survivors (*n* = 55) who were a mean of 30 months post-treatment. Using psychometric screening tools, they estimated the PTSD prevalence in the sample to be between 5-10%, consistent with previous studies. Cordova et al., (2000) conducted a similar study on the same population using a different sample (*n* = 99) who were a mean of 19 months post-treatment, and were diagnosed as having stage 0-IV breast cancer. Using similar measures, they found a markedly higher PTSD prevalence of 8.5% using the cut-off method, and 12.7% using the symptom method. However, this could be because their sample included a broader range of disease stages including a small proportion of patients who had stage IV metastatic cancer. In a study characterised by patients diagnosed with much higher disease stages (III-IV), Jacobsen (1998) assessed PTSD symptoms in breast cancer patients who were a mean of 19 months post treatment (bone marrow transplant [BMT]). Using the PCL-C, Jacobsen (1998) found that 12-19% of the women in the study screened positive for PTSD.

In another study, Green et al., (1998) studied 160 female breast cancer survivors who were between 4-12 months post treatment after being diagnosed with stages I-II breast cancer. Using the Structured Clinical Interview (SCID), they found a PTSD prevalence of 3%. However, this could be because of low disease stages and the use of a clinical interview, which has been argued to underestimate PTSD prevalence (Thompson et al., 2011). Conflicting results were found by Pitman et al., (2001). They studied of 87 female post-treatment stage I-III breast cancer survivors. Using a combination of psychiatric the PCL-C and Clinician-Administered PTSD Scale (CAPS), they found a current breast cancer-related PTSD prevalence of 9%, and a lifetime breast cancer-related PTSD prevalence of 15%. This is a markedly high statistic for this population, especially given the use of psychiatric interviews conducted by trained professionals to diagnose PTSD, rather than the administering of self-report screening tools. In a later study of 181 female stage I-IV breast cancer patients who were a mean of 18 months post-diagnosis, Levine et al., (2005), using the PCL-C, found a general PTSD prevalence of 14.4% (the proportion of PTSD sufferers who met both cut-off [24%] and cluster-related [26%] criteria).

Mundy et al., (2000), using the SCID, found no current PTSD in their sample of 17 stage 0-IV breast cancer survivors who were more than 100 days post-treatment; however, 35% of their sample was diagnosed with lifetime PTSD. This finding has been observed elsewhere: Mehnert & Koch (2007) studied 98 female breast cancer survivors just after surgery, and six months later. Using the SCID, they found a lifetime PTSD prevalence of 8.4% (18.5% with Impact of Events Scale & PCL-C), and a much lower cancer-related PTSD prevalence of 2.4%. This trend of low prevalence and a higher association between cancer and lifetime PTSD replicates findings found initially by Alter et al (1996). Unfortunately, Mehnert & Koch provided no information on the range and distribution of disease stages, providing no data on the level of traumatic exposure for their sample, so the generalizability of their prevalence figures is limited.

Shelby et al., (2005) studied a sample of 148 female stage II-III breast cancer survivors who were a mean of 18 months post-diagnosis. Using the PCL-C, they found a low PTSD prevalence of 2% for the cut-off method and 6.8% for the cluster method. In a future study, Shelby et al., (2008) studied 74 female stage II-III breast cancer survivors who were 30-40 months post-diagnosis. Using the PCL-C and the SCID, they found an overall prevalence of 15.6% cancer-related PTSD, but of particular interest was that those who were at stage III, 25% suffered from PTSD, and those with additional radical surgery, 85%. The fact that the PTSD prevalence was higher for those who underwent radical surgery was also found in a later study by Gandubert et al., (2009). They studied a sample of 144 female stage I-III breast cancer survivors who were a mean of 1.75 years post-surgery, and found that the lifetime PTSD prevalence related to the cancer index event was 18.2%. This is significantly high, and might be explained by the fact that this sample was characterised by a first episode of primary breast cancer that was treated by both radical mastectomy and chemotherapy in later stages, both very invasive and painful, and physically scarring treatments. Jim et al., (2007) found that greater physical symptoms and side-effects from cancer treatment predicted much greater cancer-related intrusions and distress. Epidemiological studies have revealed that additional physical injury is associated with higher PTSD prevalence in other trauma populations (Kilpatrick et al., 1989).

Though CR-PTSD can, and does occur, after the experience of cancer and its treatment, the prevalence rates are heterogeneous and partially dependent on sampling, patient characteristics, assessment methods, and general methodology. Nevertheless, the literature reveals that the prevalence of current cancer-related PTSD is generally low, whereas the prevalence of cancer-related lifetime PTSD is higher. There is some evidence that disease stage is implicated in increasing prevalence statistics, with higher stages being associated with higher PTSD prevalence in a similar manner to the dose-response relationships found in the general epidemiological literature. However, the heterogeneity of results, with lifetime cancer-related PTSD prevalence statistics ranging from very low (2%) to markedly high (22%) necessitates a deeper study of factors that may contribute to the risk of PTSD development in the breast cancer population.

1.5.2. Predictors of PTSD in Cancer Patients and Survivors

Thus far, the cancer literature has revealed a range of socio-demographic and clinical predictors of CR-PTSD symptoms. However, the results from some systematic reviews (Bruce, 2006; Kangas et al., 2002; Smith et al., 1999) imply that evidence for their predictive value is variable. This is markedly similar to the vulnerability factor trends discussed in chapter 1.2. Previously reviewed meta-analyses evaluated an equally large cluster of socio-demographic and clinical variables (Brewin et al., 2000; Ozer et al., 2003), from which Klein & Alexander (2009) compiled a small set of variables that are relatively consistent in their predictive value. This section shall focus on those variables, and their potential proxies, or disaggregated versions (c.f. Kraemer et al., 2001). While the focus is on breast cancer survivors, the literature on risk factor research for this population is relatively sparse; therefore several cancers have been included in this section. The variables have been discussed in order of temporal precedence (pre-trauma, peri-trauma, and post-trauma variables).

1.5.2.1. Prior Trauma

The experience of prior trauma has long been accepted as a predictor of post-traumatic symptom severity. This has also been revealed in several studies on cancer samples. For example, Andrykowski & Cordova's (1998) study of post-treatment breast cancer patients (n = 82) revealed that a greater frequency of pre-cancer stressor events was a significant predictor of greater PTSD symptom severity (r = .46). Green et al., (2000) yielded similar findings with their sample of post-treatment breast cancer patients (n =161). They found that prior physical (r = .27) and sexual (r = .23) trauma were moderate predictors of PTSD symptom severity. Other studies further demonstrate that prior trauma, while having some utility in predicting baseline distress, may also provide predictive power for symptom reduction over time: Andrykowski et al., (2000) discovered that in their sample of stage 0-III breast cancer survivors (n = 46), the degree of reduction in reported PTSD avoidance/numbing symptoms over time was significantly associated with fewer prior traumas (r = -.42), but not for any other symptom cluster. However, Tjemsland, Søreide, & Malt's (1996) study of stage I-II breast cancer patients (n =106) revealed no significant relationship between trauma history and presenting traumatic stress. This surprising find might be explained by the sample: The patients included were older (median age, 50), and being treated for early stage breast cancer (stages I-II).

1.5.2.2. Psychiatric History:

The presence of previously diagnosed mental health issues also holds some predictive power in this population. Green et al., (2000) found that a prior psychiatric diagnosis was a powerful predictor of adjustment after breast cancer (r = .35). Similarly, Palmer et al., (2004) reported that of their sample of breast cancer patients which met DSM-IV criteria for PTSD, 100% of them also met criteria for a previous major depressive disorder (MDD). Mundy et al., (2000) found that a lifetime diagnosis of PTSD prior to cancer predicted PTSD after cancer. However, Andrykowski et al., (2000) found no significant relationship between a pre-cancer depression history and subsequent PTSD, although there is a possibility that the small sample size precluded the detection of the modest relationship observed between these two variables.

1.5.2.3. Disease Stage:

The literature reveals varying trends on the role of disease stage in PTSD. One initial study by Cordova et al., (1995), studied breast cancer survivors who were diagnosed at stages I-III, but were post-treatment. They found no significant relationship between PTSD symptom severity and disease stage. Epping-Jordan et al., (1999) interviewed newly diagnosed breast cancer patients (stages I-IV), and then again at three and six months, taking measures of thought intrusions, coping strategies, and emotional distress. They found that disease stage was not a significant predictor of distress. Similarly, Cordova et al., (2007) did not find a significant relationship between disease stage and PTSD symptoms, but were surprised to find that of their sample; only half endorsed their cancer as being traumatic. Yet again, Andrykowski et al., (2000) found that there was no significant relationship between disease staging and change in PTSD symptoms. Nevertheless, Andrykowski & Cordova (1998) found that disease stage was a small but significant predictor of PTSD symptoms (r = .19, p = .05). Butler, Koopman, Classen, & Spiegel, (1999) studied 125 stage IV breast cancer patients who were still undergoing treatment, and found that 50% of the sample experienced clinically significant levels of traumatic stress symptoms. This prevalence is markedly higher than what has been reported in other studies (e.g. Cordova et al., 1995). Posluszny et al., (2011) report similar findings in their longitudinal study of stage I-IV gynecological

cancer patients. They compared these disease stage groups versus a benign tumour group, and took measures of perceived threat, impact of events, and PTSD symptoms at three time points: one week pre-surgery, seven weeks post-surgery, and 16 weeks post-surgery. They found an increased perception of threat for stage III-IV at later treatment stages, which also predicted high PTSD symptoms, with 34% of advanced stage cancer patients, 16% of early stage, and 15% of benign tumour groups reaching PTSD criteria. Similarly, Butler et al., (1999) studied a sample of women with metastatic breast cancer (n = 125) and poor functioning, and found that 52% of the sample had very high avoidance and intrusions symptoms, and were well above the total symptom cut-off. Butler et al., agree that this markedly higher result differs from Cordova et al., (1995) because the Butler et al., (1999) sample is all stage IV and therefore has a very poor prognosis. The studies that do not demonstrate a positive relationship between PTSD symptom severity and disease stage are mainly characterised by samples with lower disease stages (I-II), or samples, which while having a range of stages I-IV, include a majority of patients who are stages I-II. In contrast, Posluszny et al., (2011) sample contained an even distribution of disease stages, and Butler et als., (1999) sample were all stage IV. It has been argued that the more advanced the disease at diagnosis, the greater perceived threat to the patient's life (Cordova, et al., 1995; Koopman et al., 2002), and therefore arguably a positive relationship to PTSD symptom severity in keeping with dose-response trends found in the PTSD epidemiological literature.

1.5.2.4. Younger Age:

Green et al., (2000) showed from their sample that younger age at diagnosis was a powerful demographic predictor of more severe PTSD and depression symptoms. Koopman et al., (2002) reported similar findings, showing that women at greatest risk of PTSD symptoms at six months after diagnosis are those who are younger. It also appears that younger age also plays a role in the course of the disorder and symptom profile: Andrykowski et al., (2000) found that older age at diagnosis was associated with greater symptom amelioration (r = .31). In Palmer et al's (2004) study, age was found to be a significant predictor of perceived helplessness or horror, with those who are younger perceiving greater trauma. This may be because breast cancer diagnoses are generally associated with older women, so those who receive a diagnosis at a younger age may find it more unexpected, more life threatening, and therefore more traumatic (Green et al., 1997). There are also more far-reaching implications for the younger demographic, as this population is more likely to have children at home, and be in the middle of a career (Green et al., 2000). Receiving a diagnosis of cancer will therefore cause major disruptions to their ability to work, and to provide for themselves and their family, generating extreme anxiety, and impairing socio-occupational functioning. This may suggest that younger age in this population is a proxy variable for perceived unexpectedness of illness, or an aggregate of both perceived unexpectedness of illness, and greater perceived loss of life and resources.

1.5.2.5. Perception of Threat:

Posluszny et al., (2011) found in their gynaecological cancer sample that perceived threat to life was significantly positively correlated with intrusions at each time point. Furthermore, they found that increased perception of threat for stage III-IV cancer at later treatment stages for the disease groups, which also predicted higher PTSD symptoms. They also found increased intrusion scores for disease groups at follow-up.

1.5.2.6. Social Support and Constraint:

Another key predictor of PTSD lies on a continuum of social support, and social constraint. Social support is generally viewed as a support group of individuals who facilitate emotional openness and expression of feelings and reactions to the illness and its treatment. The typically comes from a spouse or family members, but can equally come from medical staff (or on a larger scale, the service), but also from support groups attended by other patients (see Levine et al., 2005). Levine et al., (2005) conducted an RCT on breast cancer patients which were entered into either a 12 week a) support group intervention or b) a psychosocial intervention. Both groups achieved reductions in PTSD symptoms, although highly significant reductions in re-experiencing and avoidance were found only in the support group. This suggests that social support groups are very effective in managing PTSD symptoms after diagnosis. Social constraint, on the other hand, relates to restrictions (either perceived or real) on the cancer patient talking about the illness or seeking other forms of support. This variable may be systemic in nature, involving the interaction between patient, support system/recovery environment, and traumatic event (Lepore, 2001), and research to date has identified a significant effect of the degree of social support on PTSD symptom severity across trauma populations.

Researchers such as Jacobsen et al., (2002) reported that decreased social support during cancer treatment was a predictor of future PTSD severity. Kornblith et al., (2003) also demonstrated that cancer patients with poorer social support had worse PTSD compared to the other survivors. In a key study, Green et al., (2000) found that a lack of social support was moderately associated with PTSD symptom severity (r = -.28). An earlier study by Andrykowski & Cordova (1998) found similar results, having identified social support as a key predictor of PTSD symptom severity, with higher levels being associated with less severe symptoms (r = -.39). In a later study, Andrykowski et al., (2000) found that social support, while not being associated with total PTSD symptom severity, was actually strongly positively associated with the avoidance/numbing criterion (r = .52), and similar findings have been reported elsewhere (Butler et al., 1999). This suggests that lack of social support is associated with increased avoidance and numbing strategies. It can be argued that as there is little social support, avoidance may be the chosen strategy to deal with emotions that are perceived to be overwhelming in an unsupportive environment. However, the correlational approach could ultimately predict the opposite: that avoidance/numbing actually predicts the withdrawal of support, as significant others may perceive the cancer survivor to either be coping, or unwilling to engage. In a more recent study, Cordova et al., (2007) studied a sample of 65 breast cancer patients (all disease stages, but with a majority stages I-II) and asked them to complete questionnaires on PTSD diagnostic Criterion A, another on social constraints, measures of PTSD symptoms, and post-traumatic growth. They found that greater social constraints on talking about breast cancer and the perception of cancer as a traumatic stressor were significantly associated with greater PTSD symptoms, but was not significantly associated with post-traumatic growth. Schroevers, Helgeson, Sanderman, & Ranchor (2010) investigated the predictive value of social support (perceived availability, received support, and dissatisfaction with support) on posttraumatic growth. They used a sample of 206 cancer survivors and asked them to fill in questionnaires and attend home interviews at three months post-diagnosis and eight years post-diagnosis. They found that higher levels of actual received emotional support at three months after diagnosis significantly predicted increased post-traumatic growth at eight years post (even when controlling for support at eight years). Therefore, there is

evidence to show that social support (or lack thereof) is a significant correlate of PTSD symptom severity.

1.5.2.7. Emotion Regulation and Cognitive Coping Styles:

It has long been documented that cancer patients often respond to their condition and prognosis with denial. It is only with recent advancements in treatment that clinicians began to see the psychological sequelae of the entire cancer experience and the role of coping strategies in subsequent psychopathology. It has been established that breast cancer patients who are lower in emotional self-efficacy are at greater risk of PTSD symptoms at diagnosis (Koopman et al., 2002). This is in keeping with King et al's (1998) study of military veterans, who after having been repatriated from protracted combat events, found that 'hardiness' (or resilience) was strongly related to PTSD diagnosis, and predictive of their ability to mobilise resources and emotional support. Epping-Jordan, et al., (1999) studied the processes of emotional adjustment in a sample of 80 newly diagnosed female breast cancer patients' stages I-IV (majority sample stages I-II). They used a prospective/longitudinal design, with interviews and questionnaires post-diagnosis, and then again at three and six month follow-up. Measures of optimism, intrusions, coping strategies, and emotional distress were taken at all time-points. They found a highly significant relationship between emotion-focused disengagement and anxiety/depression symptoms which increased with time from diagnosis (r = .45 to .75). There was also a highly significant relationship between problem-focused engagement and anxiety/depression symptoms that also increased with time from diagnosis (r = .40 to .48). Also, the use of problem-focused engagement was a predictor of less depression/anxiety symptoms. Conversely, the use of avoidant coping has been found to predict PTSD symptoms (Hampton & Frombach, 2000). Halstead & Fernsler (1994) used a snowballing convenience sample of cancer survivors of 5+ years to rate the efficacy of their coping strategies. Avoidance, fatalistic attitudes, and also relatively unregulated emotional responsiveness to cancer diagnosis and treatment decreased effective coping and quality of life, whereas facing the situation, seeking available support, and having an optimistic outlook, were shown to be effective and adaptive coping skills. More specific studies have been conducted on the cancer population that examine other well-known cognitive and emotional coping strategies.

1.5.2.7.1. Rumination: Chan, Ho, Tedeschi, & Leung (2011) explored the effects of an attentional bias on PTSD symptoms and post-traumatic growth as manifested through a ruminative coping style. They used a sample of 120 Chinese women with breast cancer (stages I-III) of which most received chemotherapy or surgery. Self-report psychometrics (validated into Chinese) were administered to assess PTG, PTSD, rumination and positive/negative attentional biases. They found that negative attention biases manifested in negative rumination and were a significant predictor of future PTSD symptoms, but bore no relationship to post-traumatic growth. Negative rumination acted as a partial mediator of PTSD symptoms. Whereas positive attention biases manifested in positive rumination predicted post-traumatic growth, but bore no relationship to PTSD symptoms. It is possible that as the cancer experience is rather protracted, patients will have longer to evaluate their situation, facilitating persistent negative ruminations (Brewin & Holmes, 2003).

1.5.2.7.2. Suppression: Amir & Ramati (2002) conducted a cross-sectional study which assessed the long-term effects of cancer and its treatment on breast cancer survivors in a sample of 39 breast cancer survivors (> 3 years symptom free and > 5 years post treatment), and 39 disease free controls.

Self-report psychometrics indexing emotional coping styles, PTSD symptoms, emotional distress and quality of life were administered to both groups. Result revealed that the breast cancer survivors demonstrated significantly more suppression over the non-disease group, and that this coping style was significantly correlated with hyper arousal in PTSD.

1.5.2.7.3. Dissociation and Acute Stress Disorder: Kangas, Henry, & Bryant (2005) investigated the relationship between ASD and PTSD in a sample of 63 out-patients with head, neck, and lung tumours (at disease stages I-IV). The patients were assessed by a clinical psychologist using the acute stress disorder interview and were asked to respond with reference to their cancer being the stressor. Interviews were conducted one month post-diagnosis; six months post diagnosis, and three months post-treatment. They found that the absence of ASD symptoms provided a much greater mean predictive value of who would not get PTSD (r = .85) than the presence of ASD symptoms predicting who would (r = .50). Also, some patients who went on to develop PTSD did not meet ASD criteria post cancer-diagnosis. However, the ASD symptoms were entered into a regression, and emotional numbing, a sense of reliving, and motor restlessness were significant predictor variables of PTSD. This suggests that the ASD syndrome has some negative predictive power in that those people who do not appear to apply avoidant and dissociative coping strategies immediately post-diagnosis are very likely to not develop full-PTSD. This in itself is entirely consistent with the idea that PTSD is the result of failed, ineffective, or avoidant processing (chapter 1.3.). However, when evaluating the power of ASD to predict PTSD, the construct is equivocal in its value. It is arguable that this may be due to the fact that ASD diagnosis is dependent on the time of assessment and does not take into account the buffering effects of the recovery environment that might reduce the need for these emotional regulation strategies. Kangas et al., (2005) state that high dissociation and emotional distress soon after diagnosis significantly predicts likelihood of future PTSD development. Peri-traumatic dissociation prevents the retrieval of trauma memories, and affects capacity to process emotional experience (van der Kolk & van der Hart, 1989). Kangas et al., (2005) suggest that this introduces a style that precludes them processing, and thus integrating their cancer experiences. This ultimately suggests that dissociative styles presented after diagnosis are poor coping strategies that link directly to future PTSD (see Harvey & Bryant, 2002). Conversely, they may also be a manifestation of maladaptive coping strategies that predate the cancer diagnosis (McNally, Bryant, & Ehler's 2003).

1.5.3. Discussion

It is established from these reviews that cancer patients and survivors do experience traumatic stress symptoms, independently of whether or not they qualify for a full PTSD diagnosis. Second, of those that do qualify for a full PTSD diagnosis, the prevalence of full PTSD is generally low in this population, and as some have argued, lower than that of other trauma populations (Green et al., 1998). One thing that is immediately apparent is that the heterogeneity of prevalence and vulnerability factor statistics found in cancer-trauma research is similar to that of previously reviewed epidemiological and risk factor findings. The same level of heterogeneity and methodological limitations has been found in systematic reviews devoted to non-cancer medical trauma. Tedstone & Tarrier (2003) found that in their review of studies on myocardial infarction, miscarriage, abortion, childbirth, cardiac surgery, and stroke, that there was significant heterogeneity in methodology and results. Among the most universal findings was that PTSD

symptoms were more common than full PTSD, similar to the findings of sub-syndromal symptoms prevalent in the breast cancer population (Gandubert et al., 2009; Shelby et al., 2008).

The literature suggests that background variables such pre-cancer trauma act as vulnerability factors that increase the risk of PTSD symptoms in this population (Andrykowski et al., 1998; Green et al., 2000). There is also some evidence that the disease stage at which one is diagnosed appears to be related to the perception of threat to life (Posluszny et al., 2011), and also the severity of PTSD symptoms (Andrykowski et al., 1998). This is concurrent with epidemiological findings and risk factor research in populations other than that of cancer survivors and is consonant with accepted clinical theories of PTSD (Brewin & Holmes, 2003). Younger age at diagnosis, while increasing the statistical risk of a traumatic stress response, may be a secondary variable that has a strong relationship with the perceived unexpectedness of illness and resource loss (Green et al., 2000). Also a lack of social or emotional support may also interact with prior trauma (c.f. King et al., 1998) and current cancer-related distress to completely overwhelm a cancer patient's coping capacities (Butler et al., 1999) and predicting more severe PTSD symptoms in cancer survivors (Kornblith et al., 2003). With regards to how cancer patients and survivors cope with their emotional responses, the literature demonstrates that having a strong personal efficacy is a buffer against future symptoms, whereas using learned avoidant strategies such as negative rumination, suppression, and dissociation exacerbate PTSD symptoms or predict future PTSD symptomatology. However, when comparing the cancer-related vulnerability factor research to that of those in other populations, it is apparent that while vulnerability factors have been researched extensively both cross-sectionally and prospectively, less work has been done to assess each factors relative contribution, and to the best of our knowledge, none has been done to assess the interactive effects of these variables. The work of King et al., (1998) has already demonstrated the interactive moderating and mediating effects of pre peri and post trauma characteristics in protracted traumatic situations. This is arguably of superlative importance with cancer, being a long and arduous experience, introduces many systemic variables that influence the course of trauma and adaptation, and the processes responsible may not be captured in the research.

1.5.3.1. Methodological Limitations:

Andrykowski et al., (1998) suggested that elucidating the true prevalence of cancer-related PTSD across studies is complex due to the differences in study procedures and sampled populations. The studies in this review are heterogeneous in terms of the samples they used, the type of treatments experienced, the stages of breast cancer experienced; country of study; and measures used to assess PTSD, and as such, the literature examining the epidemiology of PTSD in cancer patients and survivors is riddled with the same methodological limitations as that of those examined in chapter 1.1. Furthermore, the use of cross-sectional methods to assess PTSD in this population is dependent on the retrospective reporting of peri-traumatic experiences that may be confounded by mood at the time of assessment (Cordova et al., 2007), and more importantly for this population, cognitive-evaluative factors related to the time since the beginning of the cancer experience, which as seen from the prevalence studies place time of initial assessment between four months (Green et al., 1998) and 37-40 months (Andrykowski & Cordova, 1998; Shelby, Golden-Kreutz, & Andersen, 2008) post-treatment.

1.5.3.2. Sampling:

Studies which take an epidemiological approach use large sample sizes to increase sensitivity to factors that are over-represented in traumatised populations (Norris & Sloane, 2007). At a population level this necessitates samples in the thousands. This approach is not as apparent in the cancer literature. In addition to having small sample sizes, most of the studies data distributions are skewed towards low disease stages; the combination of both small samples sizes and low disease stage may limit these studies' sensitivity to disease stage as a factor in traumatic exposure intensity. This in itself is very revealing. Low disease stages are more likely to be included in samples as they may be less avoidant (c.f. Andrykowski et al., 2000), and less burdened with the increasingly aggressive treatment regimens required for metastatic cancers. But also, as medical screenings improve, cancers may be caught earlier, skewing the distribution of disease stages within the cancer population rather than just being an artefact of a self-selecting sample. There are also qualitatively different aspects to disease stages. Stage zero is relatively benign, and it is not until late stage three that tumours reach beyond the original site and then become metastatic, are seriously life-threatening, requiring the most aggressive of treatments. Therefore it could be that high disease stage may be more traumatogenic because a) the threat to life is severe and more imminent, and b), that this will require more aggressive, painful, and scarring, treatment. Additionally the protracted nature of the cancer experience in comparison to other traumas where death is imminent may facilitate more complex evaluations of the self in relation to the trauma (see Brewin & Holmes, 2003).

1.5.3.3. Measurement:

Another issue related to epidemiological studies is that the majority of those reviewed in chapter 1.1 used a structured clinical interview to assess the presence of PTSD in addition to using psychometric questionnaires to quantify symptom severity. The body of literature in this chapter is arguably more heterogeneous in its approach to assessment and measurement. For example, one established trend in the cancer literature is the use of self-report tools that a) may not be validated for use in cancer populations, and b) can be scored via two methods: the *cut off* or *symptom cluster* method. Of the studies reviewed by Thompson et al., (2011), the system cluster method appears to overestimate the prevalence of PTSD found in the women with breast cancer, whereas there appears to be some discrepancy in the score that should be used to warrant a likely diagnosis of PTSD for the cut-off method, or whether the cluster method is a more appropriate diagnostic gatekeeper. Many of the studies have used the PTSD Checklist Civilian Version (PCL-C) to assess the prevalence of PTSD in breast cancer survivors. The PCL-C appears to be the most frequently used self-report measure of PTSD symptoms and as a clinical screening test for PTSD. The PCL-C yields a total score and sub-scale scores for intrusive, avoidant cognitions, numbing and arousal. Participants are likely to merit a formal diagnosis of PTSD if they obtain a score of 50 or above (*cut-off method*) or if they meet at least one intrusion, three avoidance and two arousal symptoms (*symptom cluster method*; Jacobsen, et al., 1998). Most of the research uses a cut-off score of 50 or above. This value of cut-off is recommended for combat veterans, whereas a cut-off score of 44 is recommended for civilians (McDonald, & Calhoun, 2010). Other measures such as the Davidson Trauma Scale (Davidson, 1997) have taken a similar approach, using different cut-offs for different trauma populations depending on PTSD prevalence data. However, it is unclear what cut-off should be used in a cancer population when the traumatogenic nature of the cancer experience *per se* is in question, and the prevalence figures range from very low to surprisingly high. In addition, the PCL-C was developed to assess PTSD in non-combat veterans (Weathers, Huska, & Keane, 1991), and like many psychological

measures, has not been validated for use with survivors of chronic, life-threatening illnesses (Herschbach et al., 2005). Therefore, the reporting of symptoms specific to PTSD may be confounded by the experiencing of cancer-specific symptoms (Shelby et al., 2005).

Other studies use the Structured Clinical Interview for DSM-IV (SCID) or its combination with psychometric screening tools. For studies that used the SCID, the prevalence of cancer-related PTSD ranged from 0% (Mundy, et al., 2000) to 2.4% (Mehnert & Koch, 2007), to 15.6% (Shelby et al., 2008). Compared to the PCL-C, PTSD prevalence is lower when using the SCID, particularly compared to the symptom cluster method for the PCL-C. The discrepancy between self-report and clinician-administered ratings in relation to prevalence estimates have been reported where the PCL-C tends to overestimate, and the SCID underestimate, the prevalence of PTSD (Mehnert & Koch, 2007). The SCID may lack sensitivity, especially in physically ill patients and as a result underestimate the prevalence of PTSD. However, it can be argued that the SCID's apparent underestimation of PTSD prevalence is merely the result of its ability to assess functionality, distress, and differential diagnosis, and thus yield a diagnosis that is more specific than self-report screening tools. Self-report tools such as the Impact of Events Scale (Weis, 2007) and the PCL-C (McDonald & Calhoun, 2010) are very sensitive to PTSD, but there is a danger that relying on symptom measures alone can give false positives (Kangas et al., 2002) as greater scores on measures of intrusion, avoidance, and hyper-arousal, while being increasingly sensitive to PTSD, may not have sufficient specificity to diagnose the syndrome. Some psychometric measures are also equally predictive (and indicative) of current psychological distress, adjustment responses, and other co-morbid disorders such as generalized anxiety or major depression (Alter et al., 1996; Palmer et al., 2004). For example, the use of self-report measures subtly implies that the patient understands the difference between intrusive flashbacks of prior events (that are PTSD-specific) and intrusive worry about the future (or fear of recurrence), which is more indicative of generalized anxiety or major depression (APA, 2000). One example is provided by Green et al., (1998), who found that allowing any repetitive intrusive thoughts to qualify for Criterion B resulted in higher prevalence rates for lifetime (5%) and current (2.5%) PTSD, than when only re-experiencing intrusions were endorsed (lifetime, 3%; current, 1.9%). Systematic reviews have identified this issue, highlighting that many reports of intrusions are future-orientated and focused on fluctuating prognostics status, rather than just being sensory flashbacks (Bruce, 2006; Kangas, Henry, & Bryant, 2002). Furthermore, the DSM Axis I only endorses a diagnosis if the symptoms impair functioning and/or generate clinically significant distress. Self-report measures do not have the sensitivity to screen for these subtleties (APA, 2000), whereas the SCID is designed to assess the resulting functioning and distress, but, unlike self-report questionnaires, is not sensitive to symptom frequency or intensity (First et al., 2002).

1.5.3.4. Diagnostic issues:

The final issue is one of diagnostic criteria. O'Connor et al., (2007) argued that the fluctuation in PTSD prevalence is the result of using less specific DSM-IV criteria or different diagnostic criteria altogether (e.g. DSM III-R. Gandubert et al., 2009). However, some of the studies diagnose PTSD based on meeting all the six specific diagnostic criteria (A-F) to qualify for the diagnosis of PTSD (the SCID uses a similar approach). Other studies, having identified potential confounds to diagnosis, eliminate diagnostic criteria that may be over-endorsed due to artefacts of the cancer experience rather than PTSD symptomatology (Green et al., 1998; Shelby et al., 2005). This reduces the PTSD prevalence in the

sample, but also arguably sacrificed sensitivity for specificity, raising the chance of false negatives. Similar issues have been found when trying to identify hyper-arousal symptoms due to PTSD, while trying to discriminate them from the physiological side effects of treatment (Kangas et al., 2002; Shelby et al., 2005). Palmer et al (2004) among others (Green et al., 1998), have disagreed with the view that PTSD can fit the cancer experience (Meeske et al., 2001) and that given the nature of the cancer experience and the symptom profile, that PTSD may not be the best syndrome within which to conceptualise the distress of cancer patients and survivors.

This position is also reflected in the new DSM-5 (2013), where the new trauma criterion states "…a life-threatening illness or debilitating medical condition [such as cancer] is not necessarily considered a traumatic event. Medical incidents that qualify as traumatic events involve sudden, catastrophic events [e.g., waking during surgery, anaphylactic shock]" (p.274). A recent article by Kangas (2013) evaluated the change in the PTSD diagnostic criteria, and its implications for the psychological aftercare of individuals who have completed cancer treatment. As a consequence of the new trauma criterion, a PTSD diagnosis is less likely to be given to cancer survivors who present with PTSD symptoms, in favour of DSM-5's adjustment disorder (AD). AD, like PTSD, is a trauma/stress-related disorder, and is a diagnosis given to individuals who either a) present with subsyndromal PTSD, or b), experience the full PTSD syndrome in response to an event that is not considered traumatic. Kangas (2013) argued correctly that relatively few DSM-IV studies investigated the rates of AD and PTSD in cancer samples, and thus could not determine if PTSD was the primary disorder. This casts doubt on the appropriateness of PTSD as a diagnosis for cancer survivors who present with the PTSD syndrome.

1.5.4. Conclusions

While this emphasises the importance of clinicians correctly diagnosing a cancer survivor, it does not address the importance of clinicians and oncology services having an empirical estimate of the proportion of their patients who are likely to present with PTSD or AD, or of the relative risk factors contributing to their presentation. However, as is evident from systematic reviews to-date (Gurevich et al., 2002; Kangas et al., 2002; Smith et al., 1999; Thompson et al., 2011) efforts to achieve a general prevalence estimate have been impeded due to the substantial variability in rates, which is often attributed to extreme between-study heterogeneity in assessment methods (e.g. questionnaire v clinical interview), assessment points (post-diagnosis or post-treatment), and sample characteristics such as severity of disease, or mean age (Andrykowski et al., 1998). In order to counter this heterogeneity, it has been argued that future studies of PTSD in cancer populations would benefit from much larger and broader samples (Palmer et al., 2004). It is for this reason that a meta-analysis of studies reporting CR-PTSD prevalence statistics may be required. This will establish a mean PTSD prevalence estimate of the proportion of full CR-PTSD (as diagnosed via structured clinical interviews), or caseness-level PTSD symptoms (via screening questionnaires) that may present in survivors of adult cancer. A meta-analysis will also establish the degree of variance in prevalence estimates that can be explained by between-study heterogeneity, with a focus on disease stage sample characteristics, mean sample age, and time since diagnosis or treatment, and will provide evidence for patient variables that may be risk factors for the development of a PTSD.

1.6. Overview

Thus far, this literature review has revealed a range of findings and related issues that need to be addressed. Epidemiological research shows that lifetime trauma exposure is common, and that type and duration of event, and the proximity to perceived danger, all appear to be implicated in the toxicity of the experience. As such, the level of traumatic exposure is consistently implicated in the likelihood of developing subsequent PTSD. However, PTSD is not the normal course for those who endure these events. Even the most toxic of events (e.g. combat exposure, interpersonal violence) do not typically lead to PTSD, suggesting that additional factors contribute to its development.

Epidemiology has consistently revealed that experiencing trauma or mental disorder prior to the current event increases the risk of subsequent PTSD. Nevertheless, careful study reveals that the predictive value of these variables is not reflected in surprisingly weak variance contributions yielded in meta-analytic studies. This may be because the cross-sectional nature of the research cannot provide an accurate measure of their contribution, and that these variables, rather than being true vulnerability factors, may be aggregates or proxies of underlying psychosocial interactions that may interact with the trauma and the recovery environment to affect the risk of chronic PTSD. However, post-trauma variables such as a lack social support were revealed to be moderately predictive of PTSD across populations, but in veteran samples, were also shown to interact with personal resilience at the time of traumatic exposure.

Personal resilience, in that context, was said to reflect a person's ability to mobilise resources, both internally, and externally (that is, effective support-seeking). The ability to engage with one's own emotions and memories, and communicate them to seek effective support, is pivotal to the processing of trauma. Though the role of this emotional engagement is widely established, clinical theories of PTSD share a common weakness in that they struggle to cognitively represent, and predict, the relative contributions of emotions *other* than fear, emotional regulation strategies *other* than avoidance, and the role of emotion schemas in the implementation of such covert avoidance. This suggests that factors relating to the beliefs individuals have about emotions, and the strategies used to control them, though widely implicated across a broad range of disorders, is less widely researched and accounted for in PTSD.

Though trauma-focused treatments demonstrate considerable and reliable effectiveness in practice, a substantial minority of those with PTSD appear to receive little benefit from therapy. Problems are said to occur where beliefs about the experience of anxiety, or the experience of emotional numbing, may prevent full engagement with trauma-focused approaches. As such, co-morbid presentations of depression or anxiety disorders have been implicated in treatment resistance, and this may be because generalized anxiety (worry), panic, or depressive features (rumination and emotional numbing), may cause patients to over or under engage, and thus hamper the effectiveness of therapy. Further examination reveals that underlying many of these disorders is a covert avoidance of emotion that manifests itself in cognitive distraction techniques such as worry, rumination, and suppression, which have been widely implicated in the development and chronicity of many psychological disorders (including PTSD), and have demonstrated considerable predictive power similar to that of dissociation and social support. Several studies have shown that the severity of PTSD is positively correlated with problems labelling emotions

and regulating them, and schemas surrounding the negative consequences of experiencing and expressing emotion. Consequently, it has been advised that there should be an additional focus on these factors to facilitate engagement in treatment non-responders.

The nature of emotion avoidance suggests that it is a vulnerability which prevents engagement with distress, impairs the ability to adapt and adjust to distress, and stalls emotional processing - which in the case of PTSD, defines the disorder. These conclusions may of course have theoretical, clinical, and service-based, implications for cancer survivor populations who appraise their experience of cancer as traumatic. However, the literature is replete with debate about whether elements of cancer are indeed sufficiently stressful to induce PTSD. Presupposing that CR-PTSD can be reliably diagnosed via clinical interviews in this population, its prevalence is generally low, but fluctuates remarkably along with a range of sample characteristics and study methods. The generally low rates of CR-PTSD in cancer populations suggest a low conditional risk of PTSD for this event, and given the themes from the literature review, a diathesis-stress model of vulnerability to PTSD in this population would suggest that a range of ecological diatheses (such as social support and emotional resources) are strongly implicated in PTSD development. The strongest relationships with PTSD symptom severity in this population appeared to be the absence of social support and a small subset of studies into emotion regulation. This was in stark contrast to medical variables, which serve as proxies for peri-trauma factors, which though implicated, are not consistently reliable.

This thesis is focused primarily on breast cancer survivors, who are widely known to present with a range of psychological difficulties during and after the treatment of cancer. Problems with post-cancer adjustment are common, whereas PTSD due to the experience of cancer may be an uncommon, but clinically important, course of maladjustment. However, as cancer may not be widely considered a typical criterion A event (unlike combat exposure and interpersonal violence), CR-PTSD might go undetected in those requiring trauma-focused intervention. For this reason, oncology specialists may need an empirical estimate of how many cancer survivors will present with CR-PTSD following treatment completion, and what medical factors are implicated in risk of PTSD. Furthermore, it may be prudent to investigate how beliefs about emotion, and the strategies used to control them, differentiate cancer survivors with PTSD from those who suffer general problems with adjustment. This may aid screening and assessment for these vulnerable individuals. Therefore, this thesis presents a range of studies designed to elucidate these issues.

Chapter 2. Methodology

2.1. Research Aims

The overarching research aim of this thesis is fourfold: to establish 1) the commonality of CR-PTSD; 2) variables that may differentiate CR-PTSD groups from non-PTSD groups; 3) the aspects of cancer that are experienced as traumatic; and 4) variables that influence psychological adjustment and possible emergence of CR-PTSD over the course of the disease.

Study 1 addressed Aims 1 and 2. Aim 1 was achieved by implementing random effects meta-analysis to establish a mean PTSD prevalence estimate for CR-PTSD in adult cancer survivors, using diagnostic interview methods and screening questionnaires. This provided an estimate of the presentation of full-PTSD, and caseness-level PTSD symptoms indicating the need for a full assessment, respectively. Aim 2 was achieved through the analysis of moderator effects which established the percentage of variance in prevalence estimates explained by between-study heterogeneity. Focus on disease stage sample characteristics, mean sample age, and time since diagnosis or treatment provided evidence for patient variables that may be risk factors for the development of PTSD.

Study 2 addressed Aims 2 and 3. Aim 2 was further achieved by using clinical interviews to allocate cancer survivors to diagnostic groups (independent variable) such as CR-PTSD and Adjustment Disorder, and evaluating their scores on validated symptom measures of PTSD, depression, anxiety, and emotional processing variables (dependent variable). The objective was to examine whether CR-PTSD, subsyndromal PTSD, and adjustment disorder (AD) groups differed in their endorsement of emotion regulation strategies (rumination, suppression, avoidance, dissociation). In addition to group differences, Aim 2 was further investigated by examining how much variance in PTSD symptom severity would be explained by emotion regulation variables. Aim 3 was achieved by collating qualitative data of each patient's index trauma on their PTSD symptom measures.

Study 3 was implemented as a preliminary investigation of Aim 4. This was achieved by using a prospective design which followed a group of students at two time points: Time 1, when they begin their semester; and time 2, when they start their exams (the stressor). Measures of emotional processing and were used to ascertain whether the emotional processing variables in study 2 serve not only as useful concomitants of CR-PTSD but also as predictive risk factors of adaptation to stress. Similarly, measures of beliefs regarding the experience and expression of emotion were completed at both time points to explore how these attitudes may be implicated in adapting to stress.

Study 4 addressed Aim 4. This investigation used a case series approach with clinical assessment and formulations for PTSD, to explore each patient's experience of cancer, diagnosis, and treatment. The objective was to explore factors implicated in the processes of traumatisation and adjustment. The clinical focus was on the impact of beliefs about the experience and expression of emotion and emergent emotional processing styles based on findings from the first three aims.

2.2. Rationale

To answer these questions, the investigator used mixed-methods research (MMR). According to Holloway & Wheeler (2010), MMR is where a research program uses qualitative and quantitative approaches to capture multiple perspectives to develop new knowledge. The rationale for synthesising

both types of data is that these data-types are neither incompatible, nor mutually exclusive, but can rather work synergistically to enrich current theory and practice. MMR has been chosen for these reasons: 1) the CR-PTSD literature provided inconsistent data on the contributors to CR-PTSD in adult cancer survivors, with only social support being a consistent predictor; 2) the broad PTSD literature reveals social support and dissociation (an emotion-regulation variable) to be the strongest PTSD predictors; and 3), PTSD theory, while robust, is less able to predict the role of emotion schemas in predisposing individuals to chronic PTSD and these factors are now beginning to be investigated trans-diagnostically as well as in PTSD. However, the role of emotion schemas and regulation variables in CR-PTSD has been barely investigated. The discussion points in the literature review suggested the hypothesis that systemic interactions between emotion schemas, regulation strategies, and social support (c.f. King et al., 1998) may be implicated in the course of adjustment and cognitive processing during and after the cancer. The discussion argues that quantitative methods (cross-sectional / prospective designs) may not adequately capture this dynamic interaction over the course of events. Therefore, additional qualitative approaches were needed to investigate the cancer survivor's experience of adjusting to the cancer in the context of their coping strategies and close relationships.

2.3. Design

2.3.1. Mixed Methods Sequential Explanatory Design:

Given that the literature review highlights a need for richer explanations behind the role of emotion regulation and social support variables, this study used a sequential explanatory design (SED). According to Creswell (2006), the purpose of SED is that qualitative data is used to enrich, explain, or elaborate upon, results gained from quantitative approaches. This method has two phases: Phase one involves the collection and analysis of quantitative data. The second phase employs qualitative methods to elaborate on the results from the quantitative phase. There are two variations of SED: these variations are to do with how the use of qualitative methods is linked to the preceding quantitative results. The *participant selection model* is used when researchers are interested in using quantitative information to screen in participants to a more detailed qualitative study. The *follow-up explanations model* is used to elaborate on and explain group differences or statistical relationships found at the quantitative phase. This can be done by identifying study participants that fit into respective categories, and use qualitative methods to further explain these differences. The primary emphasis is on the quantitative data, and it is this phase qualitative that is of greater emphasis in this thesis.

2*.3.2. Follow-Up Explanations Model:*

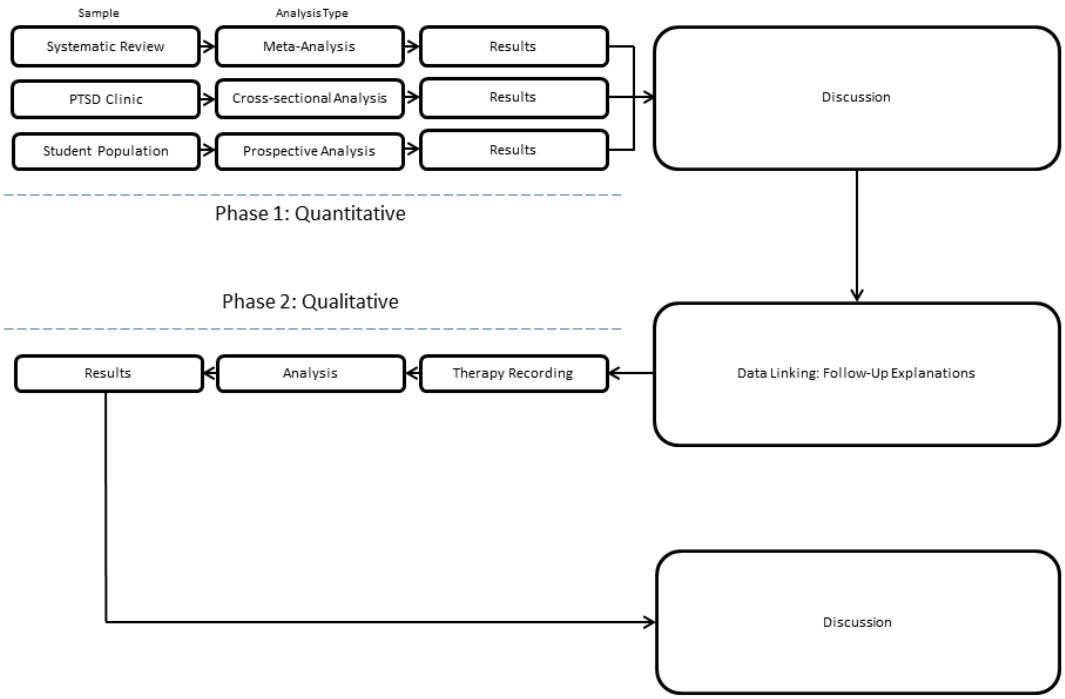
In this thesis, the follow-up explanations model is used. The quantitative data from Phase 1 (Studies 1-3) was collected and analysed prior to the collection and analysis of Phase 2 (Study 4). The data for Study 3 was collected at the same time as Study 4, but was analysed prior to the analysis of Study 4. Phases 1 & 2 were connected by using the statistical data from Phase 1 on emotion variables to determine which variables needed follow-up explanations for Phase 2.

2.3.3. Timeline:

The data from Phase 1 were focused on establishing the mean prevalence of CR-PTSD in adult cancer survivors (Study 1), demographic/medical (Studies 1 & 2), and clinical (Study 2) moderators and

correlates of CR-PTSD. The data from Study 2 was used to inform which variables were to be investigated as temporally-precedent predictors of psychiatric distress (Study 3). The data from Studies 1 & 2 was used to inform a) participant selection for Phase 2 (no PTSD *v* CR-PTSD cases), and b), with the support of data from Study 3, provide variables that require follow-up explanations in clinical case studies. The data from Phase 2 was used explain the role of key variables from Phase 1 in the course of PTSD symptoms in two adult cancer survivors. Therefore, the data gathered and analysed from Phase 1 (quantitative) provides a general overview of the clinical problem in diagnosing PTSD and identifying stable factors contributing to its emergence in adult cancer survivors, whereas the data from Phase 2 (qualitative) explores the systemic interaction of these variables in a clinical context. Though the aim of this study was to explain the relationship between quantitative variables and vulnerability to PTSD in the adult cancer survivors, the priority was given to the quantitative approach. This was due to the abundance of quantitative data in this thesis versus two clinical case studies, which cannot carry substantial weight, but rather facilitate the generation of new hypotheses for future investigation. The process is depicted in Figure 1.

Figure 1: The procedure for a mixed methods sequential explanatory design (SEM): follow-up explanations model.



2.4. Phase 1 - Quantitative

2.4.1. Study 1 Rationale:

Study 1 is a meta-analysis of prevalence rates and moderating factors for cancer-related post-traumatic stress disorder. Chapter 1.5 is replete with examples literature generating conflicting results, theoretical controversy, and hindering replicability (Coolican, 2004). The resulting lack of clarity is often due to different research designs, work in different settings, different populations, or even within the same population, heterogeneous sampling, inconsistent measures, and different times of measurement.

Systematic reviews of this literature can (and have been) been conducted to highlight problem areas, and to reach some informed general conclusions (Kangas et al., 2002; Smith et al., 1999). But, despite being methodical, the systematic review method has the potential mto be biased by the researcher's preferred psychological paradigms or research methods, and systematically (and unscientifically) affect the way studies are appraised by influencing their relative weights in the hierarchy of evidence. As a result, different systematic reviews can reach informed, but divergent, conclusions. A meta-analysis, like a systematic review, focuses on selecting studies with similar research questions. But unlike systematic reviews, meta-analyses collate the effect sizes from each of these studies, and using statistical procedures, weights the contribution of each study's effect by sample size and confidence interval, leading to an empirically and scientifically estimated average effect across studies, reducing (but not eliminating) bias.

In addition to decreasing researcher bias, a meta-analysis aims to vastly increase statistical power. According to Everett & Howell (2005), one of the key problems with psychological science is that measurement error is introduced when studies are based on samples from a population rather than a population itself. However, carefully constructed experimental designs can measure an effect within their sample that is representative of its population. In order to estimate the population effect, confidence intervals either side of the main effect estimate where this true effect lies within a range of values. The larger the sample size, the more representative of the population, and the narrower the confidence interval becomes. Meta-analyses therefore increase the statistical power by combining the results of similar studies, averaging out the error from each individual study, yielding a more accurate estimate of the mean effect. One of the superlative aims of meta-analyses is to distinguish between random error and actual between-study heterogeneity, of which the latter is tested by statistically analysing the contribution of study-specific moderators (e.g. sample size, participant age, measures). In the context of this thesis, cancer-trauma research is characterised by small sample sizes, equivocal results between heterogeneous samples, and also between direct replications of studies. Systematic reviews (Kangas et al., 2002; Smith et al., 1999) are therefore unable to reach definitive conclusions about the traumatic nature of cancer treatment, and the specific/relative power of variables to increase or decrease risk. A meta-analysis of these studies is a logical option to answer these questions.

2.4.2. Study 2 Rationale

Study 2 is a cross-sectional analysis of a sample of breast and colorectal cancer survivors with cancer-related post-traumatic stress disorder attending a trauma-focused PTSD Clinic. Chapter 1.5 suggested that cancer patients are a population of people who are often traumatised by diagnosis and treatment, but yet only a small percentage meets PTSD diagnostic criteria. Of those that do meet diagnostic criteria, it is unclear what generated it, and it has remained relatively so after decades of research. Additionally, though Study 1 aims to provide an empirical estimate of CR-PTSD prevalence, and the study-level moderators of these rates, it does not provide insight into the role of emotion variables in those who do have CR-PTSD. However, the work of Prof. Baker and Lin Purandare at Royal Bournemouth's PTSD Clinic for cancer survivors presented an opportunity to investigate the nature of the trauma, and how the chronic PTSD experience might be maintained in this population – but from the context of the individual patient. Since 2008, they have found consistently that the way patients relate to their emotions is implicated in their risk of PTSD, as well as their chances of recovery (Baker & Purandare, personal communication). Similarly, how they deal with their emotions also appears related to the experience of

trauma during the cancer and its treatment. The Clinic provided trauma-focused therapy for PTSD, with a special therapeutic focus on training their patients to learn new ways of relating to, and experiencing, their own emotions, in order to facilitate recovery, and increase future emotional resiliency. The goal of trauma-focused NICE-recommended therapy (NICE, 2005), is to consciously experience the traumatic memories that occur, in order to process it and not have them re-triggered in future.

2.4.3. Study 3 Rationale:

Study 3 was a prospective study of a sample of undergraduate students who were about to undergo a significant stressful event. This study was designed to assess whether emotional processing styles can predict vulnerability to future stress. A prospective cohort study is a research design that follows a sample of individuals from a population of interest, but who differ in several key respects that are thought to be risk factors in the outcome of a disease (Howell, 2013), or in this case, a psychological disorder such as PTSD. Prospective studies are essential components of risk-factor research as it is extremely unethical to expose individuals to diseases or psychological conditions that are considered pathological, and therefore cause suffering and require extensive professional care. In chapter 1.5., risk factors for PTSD in cancer populations were often investigated using retrospective and cross-sectional approaches. But prospective studies provide arguably better evidence of true risk factors due to their initial measurements preceding the onset of disorder. They do, however, require large samples to account for those who do not develop the condition of interest, and are therefore very expensive. In the case of this thesis, PTSD is relatively uncommon across cancer trauma populations, and would therefore require cohorts in the thousands to assess the role of emotional processing styles as a risk factor. This is expensive and unrealistic for a PhD thesis. However, as argued in chapter 1.4., emotion schemas and emotional processing styles are trans-diagnostic and generally implicated in how individuals cope with stress. Therefore, this study shall draw a sample from a student population, capturing a range of psychological differences, in order to represent, as far as possible, potential predictive factors in the population as students all experience the same stressor – final examinations.

2.5. Phase 2 - Qualitative

2.5.1. Rationale:

Study 4 was a series of clinical case studies of two breast cancer survivors with adjustment disorder and chronic cancer-related PTSD who were receiving an experimental trauma-focused therapy. This setting was used because appropriately-applied trauma-focused therapy will ensure that all facets of the experience are discussed, and as such will also provide a great deal of information about the interplay between the cancer experience, the patient's support network, and their learned strategies for coping with painful emotions. According to Holloway (2008), case studies are focused on individual patients and are researched using natural settings and multiple data sources. They often have a small number of participants, are observational (not experimental), and are primarily concerned with the collection of rich data from clinical documentation (e.g. patient histories, family accounts), or psychological treatment episodes (e.g. transcripts of therapy). Holloway (2008) states that there is no specific method of analysis for a case study; clinicians typically observe the data, form categories, develops themes, and uses inductive reasoning to inform theories. One weakness of a case study is that it lacks generalizability

compared to the quantitative methods, so one must be aware of making unwarranted conclusions about generalizability. However, though they lack generalizability, the advantage of case studies is that they can be used to gather rich qualitative data from individual cases to inform research questions, or they can be used to enrich and inform the advancement of research where quantitative approaches are limited in their sensitivity. For this reason case studies have often been used as pilot studies for experimental interventions prior to clinical trials, inform the initial development stages of psychometric tools, and can also be used in MMR as part of a sequential explanatory design (SED).

3.1. Study 1: A meta-analysis of prevalence rates and moderating factors for cancer-related post-traumatic stress disorder (CR-PTSD).

3.1.1. Introduction

3.1.1.1. Rationale

Chapter 1.5. revealed that CR-PTSD prevalence rates, though generally low, fluctuate widely, and it is largely unclear why this is the case as no factors have provided consistent power to account for this variability. Similarly, those variables that have showing predictive power have been regressed on the severity of PTSD symptoms, not PTSD as diagnosed by a structured clinical interview. As mentioned in chapter 2.6., oncology specialists may need an empirical estimate of how many cancer survivors will present with CR-PTSD following treatment completion, and what medical factors are implicated in risk of CR-PTSD.

3.1.1.2. Research Aims

Consequently, the research aims of Study 1 were as follows: 1) to establish a mean PTSD prevalence estimate for CR-PTSD in adult cancer survivors, with regards to the use of diagnostic interview methods and screening questionnaires. This will provide an estimate of the presentation of full-PTSD, and caseness-level PTSD symptoms indicating the need for a full assessment, respectively. This aim will also provide a prevalence estimate of current CR-PTSD that can be used to set a clinical cut-off on the Davidson's Trauma Scale for Study 2. And 2), to establish the percentage of variance in prevalence estimates that can be explained by between-study heterogeneity, with a focus on disease stage sample characteristics, mean sample age, and time since diagnosis or treatment. This will provide evidence for patient variables that may be risk factors for the development of a PTSD.

3.1.1.3. Hypotheses

1) The prevalence of CR-PTSD in the cancer population will be low (<10%)
2) Disease staging, mean age, and time since treatment will be significant moderators of CR-PTSD prevalence rates.

3.1.2. Methods

3.1.2.1. Search Strategy

Seven online databases (MEDLINE, PsycARTICLES, PsycINFO, CINAHL Complete, CINAHL Plus, Academic Search, and E-Journals) were searched systematically. Studies from 1994 (the publication of the DSM-IV) until 11 June 2013 were included. The search terms (cancer) AND (PTSD) AND (prevalence) were used for every database. It was decided to adopt such broad terms to capture as many studies as possible that are focused on the number of cancer-related PTSD cases in their sample. Dissertations were included in the search, but case studies, and studies that were not available in English, were excluded. In addition, we examined seminal systematic reviews from 1999 to 2011 concerning CR-PTSD in survivors of adult cancers, and abstracted any relevant references from these reviews that were not returned in online databases. The following inclusion/exclusion criteria for this analysis were compiled by the primary and secondary authors following an investigation of issues identified in the above systematic reviews, and were agreed on by full consensus of all the authors.

3.1.2.2. Inclusion & Exclusion Criteria

Articles were included in the meta-analysis if they met the following criteria: 1) they were conducted with, and specify, a sample of cancer patients (those who are in treatment) or survivors (those who have completed treatment). Note that the search strategy does not specify any particular cancer due to the systematic reviews arguing the scarcity of evidence for the effect of disease variables on PTSD prevalence rates; 2) they provide an estimate of PTSD prevalence (that is, the number of people in the sample who meet caseness); 3) they report prevalence statistics that are in reference to cancer diagnosis and treatment as the traumatic stressor; 4) they provide the following summary statistics for their sample: a) disease stages in the sample, b) gender, c) mean age of sample, and d) mean time post-diagnosis or post-treatment; 5) they use both/either a structured clinical interview such as the Clinician-Administered PTSD Scale (CAPS; Blake et al., 1995**)**, Structured Clinical Interview (SCID; First et al., 2002), Watson's PTSD Inventory **(**Watson et al, 1991**)**, or a validated screening questionnaire that is based on DSM-IV PTSD criteria such as the PTSD Checklist – Civilian Version (PCL-C; Blanchard et al., 1996), PTSD Reaction Index **(**Steinberg et al., 2013); and 6) use cross-sectional, or longitudinal/prospective, methods. Articles were excluded from the meta-analysis if they met the following criteria: 1) they used the Impact of Events Scale as a standalone measure of probable PTSD (Joseph, 2000); 2) they specifically used samples of adult survivors of childhood cancers, 3) they were case studies; 4) they were not available in English, or 5) they used the same sample as another included study.

3.1.2.3. Study Selection

After the removal of duplicates entries from the literature search, the full texts of all the remaining records were read by the primary author. Studies that clearly met exclusion criteria (e.g. did not use cancer samples, or used the IES as a standalone PTSD measure) were excluded by the primary author. All authors read the remaining full-text articles, and selected from the remaining studies those that they believed met inclusion criteria, and discrepancy between authors was resolved by full consensus.

3.1.2.4. Data Abstraction

All prevalence figures, sample characteristics, and study methods, were abstracted onto a spread sheet for review by the primary author, re-checked four times to ensure accurate data abstraction, and a random sample of the included studies were independently checked by a clinical psychology postgraduate (Table 1). There was full agreement. Though most of the data was easily abstracted in its presented form (e.g. mean age, gender, event rates, assessment method), some statistics were calculated manually. For example, in order to assess the moderating effect of disease stage distribution on PTSD prevalence rates, a summary statistic on the skew of the sample distribution had to be used. Given that many studies are skewed towards low disease stages, the mode (0, 1, 2, 3, or 4) was used to depict the disease stage that was most frequent in each sample. There was k=4 studies that were bimodal; in these cases, all bimodal distributions were characterised by either I-II, II-III, or III-IV, so given the nature of cancer-stage progression, the mean was used (1.5, 2.5, and 3.5, respectively). In some cases, the sample size reported in abstracts did not reflect those in the final analysis, so our abstracted n's reflected only those participants who completed the study. Finally, some authors reported time since diagnosis or treatment using different units (days/weeks rather than months). All studies were converted to the number of months. In all cases where it was reported in days or weeks, a month was treated as 30 days, and weeks were multiplied by seven, and then divided by 30 to get the number of months.

3.1.2.5. A-priori Statistical Methods & Analysis

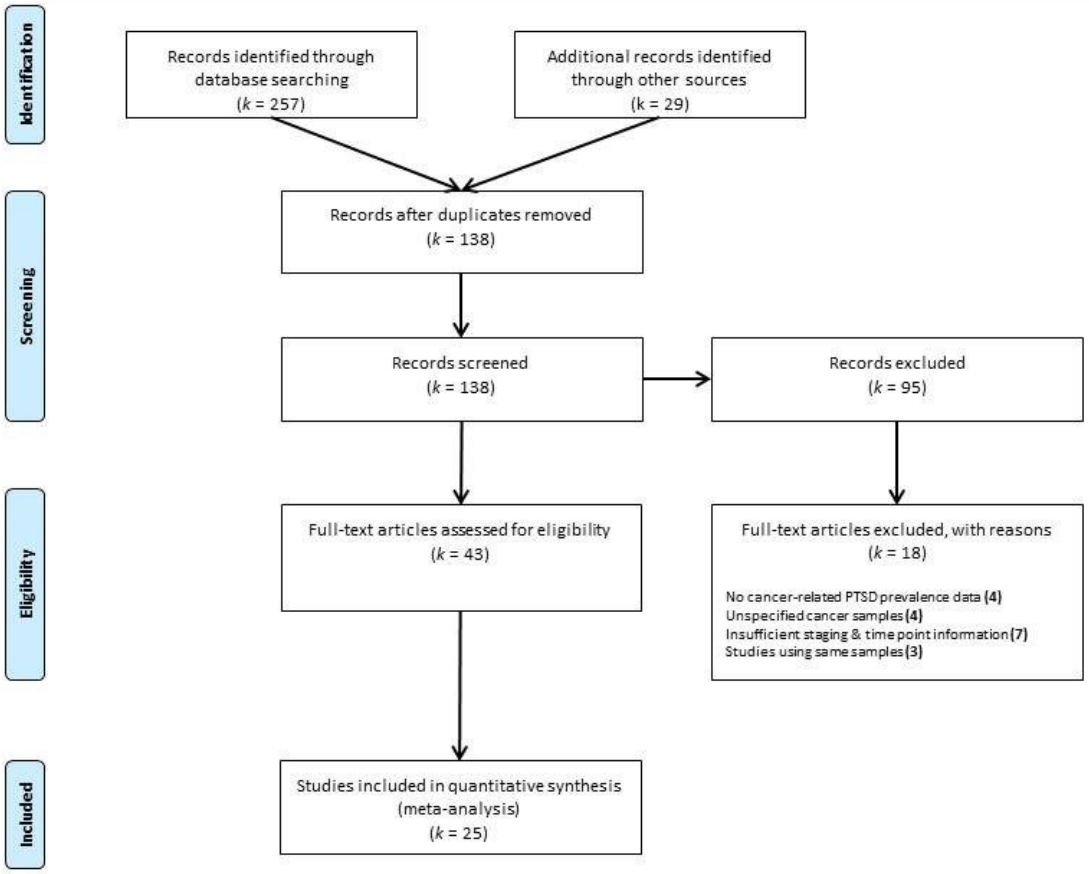
All statistical analyses and graphical presentations were conducted using Comprehensive Meta-analysis (CMA; Englewood, Biostat). Prevalence statistics were depicted using the event rate. 95% confidence intervals were calculated within the CMA software using the sample size (n) and standard error. When prospective studies were included in the analysis, the Time 1 measurements (being temporally associated with study-level moderators such as time since diagnosis and/or treatment) were used to calculate the event rates. In cases where the T1 measurements were not post-treatment, the T2 prevalence estimates (and the mean time post-treatment) were used. This was the case for one study only (Posluszny et al., 2011). All meta-analyses were conducted under a random-effects model due to the substantial methodological heterogeneity identified in systematic reviews. Mixed-effects meta-regression was used to examine the association between continuous variables such as time since diagnosis or treatment end and mean age of sample, on PTSD prevalence rates. Categorical analyses were conducted on studies where samples were characterised by either low (I-II) or high (III-IV) disease stages, and also on population studies (USA/Canada, UK/Europe, and Eastern Countries). Finally, publication bias was assessed using funnel plots. In order to calculate the variance contribution of statistically significant study-level moderators on PTSD event rates, we calculated the R^2 statistic using the following formula: $[R^2=1-(T^2_{unexplained} / T^2_{total})]$.

3.1.3. Results

3.1.3.1. Literature Search & Study Characteristics

We identified 257 articles. After duplicates were removed, 109 articles remained. A combined total of 138 studies were recovered from online databases and systematic reviews. Figure 2 depicts the PRISMA search strategy (Moher et al., 2009). Table 1 presents the abstracted prevalence data and study/sampling characteristics for the final dataset (k=25). The aggregated sample was n=4189, of which 88% were female, with Mystakidou et al., (2012) being the only study to represent both genders. In terms of methodology, k=19 studies were cross-sectional, and k=6 were longitudinal/prospective. The majority of studies were conducted in the United States (k=18), with one in Japan, five in Europe & the UK, and one in Israel. Though there were a range of cancers included in the final meta-analysis, 21 studies used exclusively female breast cancer samples, with one study using ovarian cancer (Goncalves et al., 2011), one gynaecological cancer (Posluszny et al., 2011) and two inclusive of breast cancer, but with the addition of others Alter et al., 1996; Mystakidou et al., 2012). Due to the low n for cancers other than breast, an analysis of the moderating effects of cancer type could not be conducted. Therefore a separate analysis was conducted on breast cancer alone, given that this is the most frequently studied cancer population. Also, the moderating effects of gender could not be analysed due to the substantial predominance of females over males in the final dataset.

Figure 2: PRISMA Flow Diagram for systematic review procedure.



3.1.3.2. CR-PTSD Prevalence Rates

The results of the meta-analysis of CR-PTSD prevalence rates are available in Table 2. The table depicts prevalence rates by assessment strategy, from clinical interview methods (SCID, CAPS, Watson's PTSD Inventory), to questionnaires (PCL-C, PDS, PTSD Reaction Index (RI), PTSD Scale). Separate statistics are also provided for the PCL-C and SCID given their use in the majority of studies. Table 2 also presents these statistics for the whole pool of cancer studies and for breast cancer alone.

All cancer types: Studies that used the PCL-C yielded lower event rates using the cut-off method [7.3%, 95% CI = 4.5-11.7, *k*=10] compared to the symptom cluster method [11.2%, 95% CI=8.7-14.4, *k*=9]. Studies using the SCID alone yielded higher lifetime diagnoses [15.3%, 95% CI=9.1-24.7, *k*=5] than current diagnoses [5.1 %, 95% CI=2.8-8.9, *k*=9]. Studies using a clinical interview method yielded a combined event rate of 6.4% [95% CI=4.1-9.9, *k*=12] for current PTSD, and 12.6% [95% CI=7.5-20.7, *k*=7] for lifetime PTSD. Studies that used a cut-off score screening method used the PCL-C exclusively (see above results). All other screening tools used a symptom cluster method, and yielded remarkable similar prevalence estimates to the PCL-C cluster method [13.8%, 95% CI=9.5-19.6, *k*=11]. No comparison could be done between interview methods, or between screening tools, because only the PCL-C and the SCID was used more than once. Though the point estimates for each assessment method are comparable, each is characterised by considerable between-study heterogeneity across cancer samples [Table 2; I^2=54-86%].

Breast cancer: Studies that used the PCL-C yielded lower event rates using the cut-off method [6.4%, 95% CI=4.2-9.7, *k*=9] compared to the symptom cluster method [11.2%, 95% CI=8.7-14.4, *k*=9]. These figures are similar to those found across cancers, although the cut-off event rate for breast cancer alone is 1% less. The rates for the symptom cluster method here are the same for breast cancer as for all cancers. Studies using the SCID alone yielded lower but nevertheless comparable event rates for current CR-PTSD [4.1%, 95% CI=2-8.5, *k*=7]. Much higher rates were found for lifetime PTSD [14.2%, 95% CI=7.7-24.9, *k*=4]. Studies that used a clinical interview method yielded a combined event rate of 5.8% [95% CI=3.3-10, *k*=10] for current PTSD, and 11.5% [95% CI=6.3-20.1, *k*=6] for lifetime PTSD. Studies using the cut-off scoring method used the PCL-C exclusively (see above results). All other screening tools used a symptom cluster method, and yielded remarkable similar prevalence estimates to the PCL-C cluster method [12.1%, 95% CI=9.3-15.7, *k*=10]. Notably similar between-study heterogeneity was observed for breast cancer samples compared to heterogeneity across cancers [I^2=54.4-81.5%].

Table 1. Study Characteristics

Investigator	**Design**	**Population**	***n***	**Mean Age** *(y)*	**Female** *%*	**Disease Stage** [*Mode*]	**Cancer**	**Assessment Point** *(months)* *Post Dx*	*Post Tx*	**Questionnaire**	**Clinical Interview**	**Cancer-related PTSD Prevalence** *(%)* *Cut-off*	*Cluster*	*Current*	*Lifetime*
Cordova et al., [1995] *[26]*	Cross-sectional	USA	55	55.5	100	I-IIIA *[1]*	Breast (100%)		30.5	PCL-C	No	5.5	10.9		
Alter et al., [1996] *[6]*	Cross-sectional	USA	27	54	100	I-II *[2]*	Breast (81%) Other (19%)	64.8	55.2	None	SCID			4	22
Andrykowski et al., [1998] *[17]*	Cross-sectional	USA	82	56.6	100	I-IIIA *[1]*	Breast (100%)		37.3	PCL-C	SCID	5	6		
Jacobsen et al., [1998] *[27]*	Cross-sectional	USA	43	44.4	100	II-IV *[3]*	Breast (100%)		19.4	PCL-C	No	12	19		
Green et al., [1998] *[8]*	Cross-sectional	USA	160	53.4	100	I-II *[1.5]*	Breast (100%)		6.5	IES	SCID			3	5
Andrykowski et al., [2000] *[16]*	Prospective	USA	46	56.4	100	0-IIIA *[1]*	Breast (100%)	T1 - 29.8 T2 - 40.9		PCL-C	No	4.4 6.5			
Cordova et al., [2000] *[18]*	Cross-sectional Groups (Conv *v* ABMT)	USA	142	56.4 (G1) 44.4 (G2)	100	0-IV *[1]*	Breast (100%)	G1 - 35.6 G2 - 19.4		PCL-C	No	8.5	12.7		
Mundy et al., [2000] *[9]*	Cross-sectional Groups (BMT *v* Conv)	USA	37	43.3 (G1) 50.2 (G2)	100	I-IV *[2]*	Breast (100%)	G1 - 10.4 G2 - 43.4		No	SCID			0	35
Pitman et al.,	Cross-sectional	USA	50	52.5	100	I-III	Breast	19.5		PCL-C	CAPS			14	15

[2001] *[25]*						*[2]*	(100%)							
Amir & Ramati [2002] *[49]*	Cross-sectional	Israel	39	50.4	100	I-III *[1.5]*	Breast (100%)	60	58	PTSD Scale	No			18
Boyer et al., [2002] *[33]*	Cross-sectional	USA	133	65	100	0-IV *[1]*	Breast (100%)	37.2		PTSD RI	No		21.1	
Kornblith et al., [2003] *[21]*	Cross-sectional	USA	153	65	100	I-II *[1.5]*	Breast (100%)		216	PCL-C	No		4.6	
Luecken et al., [2004] *[45]*	Cross-sectional	USA	71	53	100	0-III *[2]*	Breast (100%)	6		No	SCID			3
Palmer et al., [2004] *[35]*	Cross-sectional	USA	115	55.6	100	I-IV *[2]*	Breast (100%)		12-60	IES	SCID			4
Matsuoka et al., [2005] *[46]*	Cross-sectional	Japan	155	46.8	100	I-III *[1.5]*	Breast (100%)		1.56	No	SCID			0.6
Shelby et al., [2005] *[20]*	Cross-sectional	USA	148	50.5	100	II-III *[2]*	Breast (100%)		6	PCL-C	No	2	6.8	
Cordova et al., [2007] *[28]*	Cross-sectional	USA	65	52.3	100	I-III *[1]*	Breast (100%)	9.4		PCL-C	No	11	17	
Mehnert & Koch [2007] *[43]*	Longitudinal	Germany	(T2) 98	54.9	100	0-IV *[2]*	Breast (100%)	0.47	0.1	PCL-C	SCID	11.2	16.3	2.4
Morrill et al.,	Cross-sectional	USA	161	59	100	I-II	Breast	48		PCL-C	No	1.9		

[2008] *[24]*						*[1]*	(100%)							
Mehnert & Koch [2008] *[44]*	Cross-sectional	Germany	1083	61.8	100	I-IV *[1]*	Breast (100%)	46.5		PCL-C	No	12		
Shelby et al., [2008] *[47]*	Prospective	USA	74	51	100	II-III *[2]*	Breast (100%)		6	PCL-C	SCID		16.2	
Gandubert et al., [2009] *[48]*	Case-control	France	144	53	100	I-III *[2]*	Breast (100%)		21	No	Watson's PTSD Inventory		4.9	2.8
Posluszny et al., [2011] *[34]*	Longitudinal	USA	53 50		100	I-IV *[1]*	Gyne cologial	T1 - 0.0	T2 - 1.63	PCL-C	No	23.4 23.4		
Goncalves et al., [2011] *[42]*	Longitudinal	UK	(T1) 161 (T4) 69	61	100	I-IV *[3]*	Ovarian	T1 - 1.77 T3 - 5.27	3	PDS	No	36 45		
Mystakidou et al., [2012] *[41]*	Cross-sectional	Greece	989	64.4	49.2	III-IV *[4]*	Breast (18%) Gastro (23.6%) Lung (21%) Other (10.3%)		0	No	SCID		11.4	

Key: T1, 2, 3 Time point
G1, 2, 3 Group
ABMT Autologous Bone Marrow Transplant
Conv Conventional Treatment

Table 2. PTSD Event Rates a) across cancers, and b) for breast cancer samples, according to method of assessment

Cancer-related PTSD Measure		***k***	**Event**	**95% CI**		***p***	***Heterogeneity***				***Tau²***			
		%	**Rate**	*Lower*	*Upper*		*Q*	*df*	I^2	*p*	*Tau2*	*Tau*	*SE*	*Variance*
Breast Cancer														
By Assessment Method														
Interview	*Current*	10	5.8	3.3	10	< 0.01	33.4	9	73.05	< 0.01	0.61	0.78	0.42	0.18
	Lifetime	6	11.5	6.3	20.1	< 0.01	27.01	5	81.5	< 0.01	0.53	0.73	0.44	0.19
Questionnaire	*Cut-off*	9	6.4	4.2	9.7	<.001	17.56	8	54.44	< 0.01	0.24	0.49	0.23	0.06
	Cluster	10	12.1	9.3	15.7	< 0.01	28.14	9	68.02	< 0.01	0.14	0.37	0.12	0.01
By Specific Tool														
SCID	*Current*	7	4.1	2	8.5	<.001	21.89	6	72.59	<.001	0.7	0.83	0.61	0.37
	Lifetime	4	14.2	7.7	24.9	<.001	15.55	3	80.71	<.001	0.4	0.63	0.42	0.17
PCL-C	*Cut-off*	9	6.4	4.2	9.7	<.001	17.56	8	54.44	<.03	0.24	0.49	0.23	0.06
	Cluster	9	11.2	8.7	14.4	<.001	18.8	8	57.45	<.02	0.1	0.31	0.1	0.01
Across Cancers														
By Assessment Method														
Interview	*Current*	12	6.4	4.1	9.9	< 0.01	42.8	11	74.29	< 0.01	0.41	0.64	0.34	0.11
	Lifetime	7	12.6	7.4	20.7	< 0.01	28.73	6	79.2	< 0.01	0.49	0.7	0.38	0.15
Questionnaire	*Cut-off*	10	7.3	4.5	11.3	< 0.01	31.4	9	71.33	< 0.01	0.47	0.68	0.33	0.11
	Cluster	11	13.8	9.5	19.6	< 0.01	75.89	10	86.82	< 0.01	0.41	0.64	0.29	0.08
By Specific Tool														
SCID	*Current*	9	5.1	2.8	8.9	<.001	35.46	8	77.43	<.001	0.53	0.72	0.49	0.24
	Lifetime	5	15.3	9.1	24.7	<.001	16.64	4	75.96	<.001	0.34	0.59	0.33	0.11
PCL-C	*Cut-off*	10	7.3	4.5	11.7	<.001	31.4	9	71.33	<.001	0.47	0.68	0.33	0.11
	Cluster	9	11.2	8.7	14.4	<.001	18.8	8	57.45	<.001	0.09	0.31	0.01	0.01

Figure 3: Current CR-PTSD prevalence as assessed by clinical interviews.

Study name	Outcome	Time Point	Diagnostic Method	Statistics for each study				
				Event rate	Lower limit	Upper limit	Z-Value	p-Value
Mystakidou et al., [2012]	Current Cancer - Related [Interview]	T1	Interview	0.114	0.096	0.135	-20.494	0.000
Mehnert & Koch [2007]	Current Cancer - Related [Interview]	T1	Interview	0.024	0.007	0.082	-5.614	0.000
Gandubert et al., [2009]	Current Cancer - Related [Interview]	T1	Interview	0.049	0.024	0.099	-7.682	0.000
Amir & Ramati [2002]	Current Cancer - Related [Interview]	T1	Interview	0.180	0.088	0.332	-3.638	0.000
Matsuoka et al., [2005]	Current Cancer - Related [Interview]	T1	Interview	0.039	0.018	0.084	-7.723	0.000
Green et al., [2000]	Current Cancer - Related [Interview]	T1	Interview	0.025	0.009	0.065	-7.235	0.000
Alter et al., [1996]	Current Cancer - Related [Interview]	T1	Interview	0.040	0.006	0.222	-3.236	0.001
Mundy et al., [2000]	Current Cancer - Related [Interview]	T1	Interview	0.013	0.001	0.178	-3.033	0.002
Pitman et al., [2001]	Current Cancer - Related [Interview]	T1	Interview	0.140	0.060	0.292	-3.831	0.000
Palmer et al., [2004]	Current Cancer - Related [Interview]	T1	Interview	0.040	0.016	0.096	-6.678	0.000
Luecken et al., [2004]	Current Cancer - Related [Interview]	T1	Interview	0.030	0.008	0.108	-4.997	0.000
Shelby et al., [2008]	Current Cancer - Related [Interview]	T1	Interview	0.162	0.094	0.264	-5.209	0.000
				0.064	0.041	0.099	-11.220	0.000

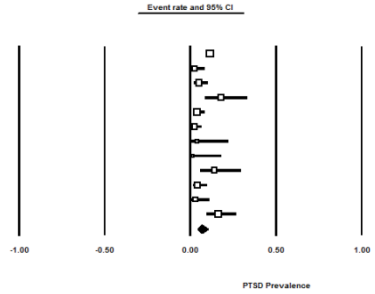


Figure 4: Lifetime CR-PTSD prevalence as assessed by clinical interviews.

Study name	Outcome	Time Point	Diagnostic Method	Statistics for each study				
				Event rate	Lower limit	Upper limit	Z-Value	p-Value
Gandubert et al., [2009]	Lifetime Cancer - Related [Interview]	T1	Interview	0.028	0.011	0.072	-7.022	0.000
Matsuoka et al., [2005]	Lifetime Cancer - Related [Interview]	T1	Interview	0.148	0.100	0.213	-7.738	0.000
Green et al., [2000]	Lifetime Cancer - Related [Interview]	T1	Interview	0.050	0.025	0.097	-8.117	0.000
Alter et al., [1996]	Lifetime Cancer - Related [Interview]	T1	Interview	0.220	0.102	0.412	-2.724	0.006
Mundy et al., [2000]	Lifetime Cancer - Related [Interview]	T1	Interview	0.270	0.152	0.433	-2.686	0.007
Pitman et al., [2001]	Lifetime Cancer - Related [Interview]	T1	Interview	0.150	0.067	0.303	-3.768	0.000
Luecken et al., [2004]	Lifetime Cancer - Related [Interview]	T1	Interview	0.180	0.107	0.287	-4.909	0.000
				0.126	0.074	0.207	-6.422	0.000

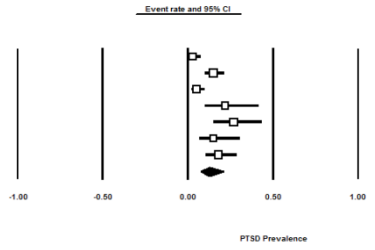


Figure 5: Prevalence of caseness-level CR-PTSD symptoms as assessed with screening questionnaires (cut-off method)

Study name	Outcome	Time Point	Diagnostic Method	Statistics for each study				
				Event rate	Lower limit	Upper limit	Z-Value	p-Value
Jacobsen et al., [1998]	Cancer - Related [Cut-off]	T1	Screening Questionnaire	0.120	0.052	0.255	-4.246	0.000
Mehnert & Koch [2007]	Cancer - Related [Cut-off]	T1	Screening Questionnaire	0.110	0.062	0.189	-6.476	0.000
Cordova et al., [1995]	Cancer - Related [Cut-off]	T1	Screening Questionnaire	0.055	0.018	0.156	-4.808	0.000
Andrykowski & Cordova [1998]	Cancer - Related [Cut-off]	T1	Screening Questionnaire	0.050	0.019	0.124	-5.811	0.000
Cordova et al., [2000]	Cancer - Related [Cut-off]	T1	Screening Questionnaire	0.085	0.049	0.144	-7.897	0.000
Andrykowski [2000]	Cancer - Related [Cut-off]	T1	Screening Questionnaire	0.044	0.011	0.158	-4.282	0.000
Cordova [2007]	Cancer - Related [Cut-off]	T1	Screening Questionnaire	0.110	0.054	0.212	-5.274	0.000
Morril et al., [2008]	Cancer - Related [Cut-off]	T1	Screening Questionnaire	0.019	0.006	0.057	-6.832	0.000
Posluszny et al., [2011]	Cancer - Related [Cut-off]	T1	Screening Questionnaire	0.234	0.137	0.370	-3.550	0.000
Shelby et al., [2005]	Cancer - Related [Cut-off]	T1	Screening Questionnaire	0.020	0.006	0.061	-6.628	0.000
				0.073	0.045	0.117	-9.633	0.000

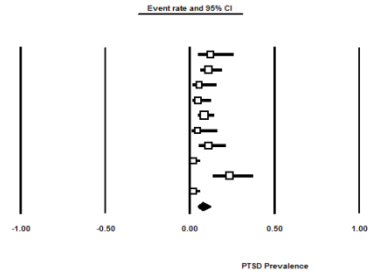


Figure 6: Prevalence of caseness-level CR-PTSD symptoms as assessed by screening questionnaires (symptom cluster method).

Study name	Outcome	Time Point	Diagnostic Method	Statistics for each study				
				Event rate	Lower limit	Upper limit	Z-Value	p-Value
Goncalves et al., [2011]	Cancer - Related [Symptom Cluster]	T1	Screening Questionnaire	0.450	0.337	0.568	-0.829	0.407
Jacobsen et al., [1998]	Cancer - Related [Symptom Cluster]	T1	Screening Questionnaire	0.190	0.099	0.334	-3.730	0.000
Mehnert & Koch [2007]	Cancer - Related [Symptom Cluster]	T1	Screening Questionnaire	0.163	0.102	0.250	-5.982	0.000
Mehnert & Koch [2008]	Cancer - Related [Symptom Cluster]	T1	Screening Questionnaire	0.120	0.102	0.141	-21.307	0.000
Cordova et al., [1995]	Cancer - Related [Symptom Cluster]	T1	Screening Questionnaire	0.109	0.050	0.222	-4.856	0.000
Andrykowski & Cordova [1998]	Cancer - Related [Symptom Cluster]	T1	Screening Questionnaire	0.060	0.025	0.137	-5.917	0.000
Cordova et al., [2000]	Cancer - Related [Symptom Cluster]	T1	Screening Questionnaire	0.127	0.082	0.193	-7.649	0.000
Boyer et al., [2002]	Cancer - Related [Symptom Cluster]	T1	Screening Questionnaire	0.210	0.149	0.287	-6.224	0.000
Cordova [2007]	Cancer - Related [Symptom Cluster]	T1	Screening Questionnaire	0.170	0.097	0.281	-4.802	0.000
Kornblith et al., [2003]	Cancer - Related [Symptom Cluster]	T1	Screening Questionnaire	0.046	0.022	0.093	-7.857	0.000
Shelby et al., [2005]	Cancer - Related [Symptom Cluster]	T1	Screening Questionnaire	0.068	0.037	0.122	-8.017	0.000
				0.138	0.095	0.196	-8.493	0.000

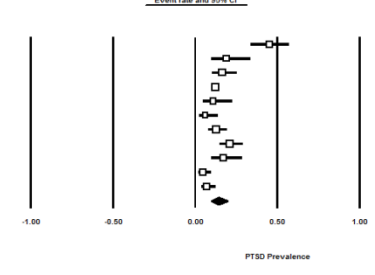
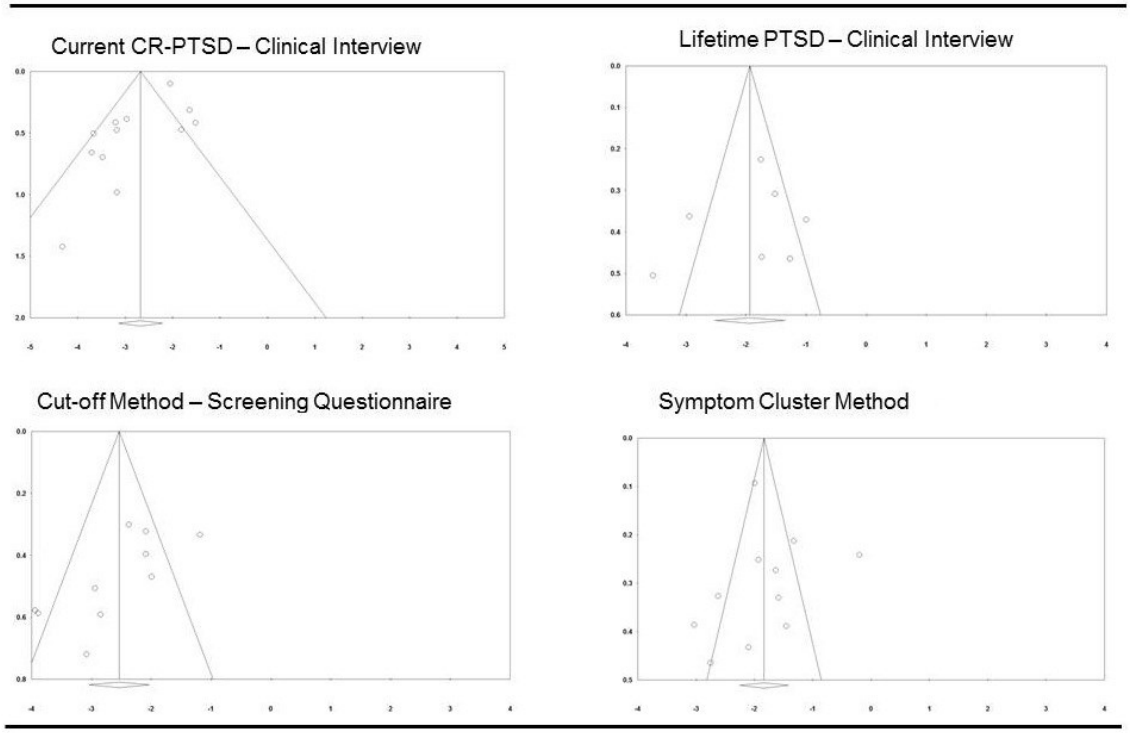


Figure 7: Funnel plots of PTSD prevalence by logit event (x axis) and standard error (y axis) across cancers by assessment method.



3.1.3.3. Sources of Heterogeneity

Mean sample age, mean time since diagnosis, and mean time since end of treatment were entered into a meta-regression for their individual variance contribution to CR-PTSD prevalence rates. Variables such as low or high modal disease staging, and population of origin (USA, UK & Europe, Japan, and Israel) were analysed categorically to assess group differences in prevalence. The time post-diagnosis was not significant for any analyses. Time post-treatment was not significant when using the SCID, PCL-C, or when using the questionnaire screening method, but was significant when including other clinical interviews to assess lifetime PTSD [$Q_{model}(1)=3.84$, $T^2_{unexplained}=0.22$ $k=3$, $p=.05$], with PTSD event rates decreasing when time since treatment increases, and a relative variance contribution of 56% [$R^2=.56$, $p<.05$]. The contribution of mean sample age to prevalence was not significant for breast cancer samples, or when using the cut-off and cluster screening methods, the clinical interview method, or when using the SCID to assess lifetime CR-PTSD in all cancers. However, it was significant when using the SCID to assess current CR-PTSD [$Q_{model}(1)=.43$, $p=.05$]. Disease stage was significant when using the SCID to assess current CR-PTSD, [$Q(1)=10.23$, $p=.05$]. Studies characterised by stage I-II samples yielding markedly lower prevalence rates (4.2%, 95% CI=2.1-8.1, $k=8$, $n=737$], than those with high stage (III-IV) disease [11.4%, 95% CI=9.6-13.5, $k=1$, $n=989$]. This was also true for the interview method [$Q(1)=6.07$, $p=.01$], with the only difference in effect coming from the additional interview methods other than the SCID in the low-stage group [5.7%, 95% CI=3.4-9.6, $k=11$, $n=970$]. Finally, the use of the questionnaire method with cluster scoring also yielded a significant difference [$Q(1)=3.71$, $p=.05$] with lower stages yielding significantly lower prevalence rates [11.6, 95% CI=8.8-13.5, $k=9$, $n=1894$], than higher stage samples [31.3 95% CI=11.8-61, $k=2$, $n=112$]. This moderator was not significant for pure breast cancer samples.

Population was not a significant moderator of event rates when using the questionnaire method or on the PCL-C, or the SCID, alone, either in the full cancer sample, or in exclusively breast cancer samples. But, significant differences were found when using the clinical interview method to diagnose current CR-PTSD [$Q(3)=9.2$, $p=.03$], with Israel having significantly higher rates [18%, 95% CI=8.8-33.2, $k=1$] than the US [5.6%, 95% CI=2.6-11.6, $k= 8$] Europe [6%, 95% CI=2.4-14, $k=3$], or Japan [3.9%, 95% CI=1.8-8.4, $k=1$]. Population was also significant for lifetime CR-PTSD [$Q(2)=11.28$, $p=.03$], with the USA [15.4%, 95% CI=8.4-26.7. $k=5$], and Japan [14.8%, 95% CI=10-21.3, $k=1$] having higher rates than Europe [2.8%, 95% CI=1.1-7.2, $k=1$]. Population was significant in exclusively breast cancer samples when diagnosing lifetime CR-PTSD with the interview method [$Q(2)=10.83$, $p<.01$], with the UK & Europe [2.8%, 95% CI=1.1-7.2] having a significantly lower prevalence than the USA [14.2%, 95% CI=6.8-27.5 $k=4$] and Japan [14.8, 95% CI=10-21.3, $k=1$]. Similar trends in breast cancer were found when diagnosing current PTSD [$Q(3)=11.43$, $p<.01$], with Israel yielding higher rates [18%, 95% CI=8.8-33.2, $k=1$] than Japan [3.9%, 95% CI=1.8-8.4, $k=1$], UK & Europe [4.1%, 95% CI=2.2-7.6, $k=2$], and USA [5.7%, 95% CI=2.5-12.6, $k=6$].

3.1.4. Discussion

3.1.4.1. Publication Bias

Before discussing and evaluating the results from this stage, one must consider the role of publication bias in generating these data. Though sources of bias are important when conducting a systematic review or meta-analysis, it may not be here as the conflicts-of–interest that underpin the file-drawer problem (FDP) are primarily concerned with the reported efficacy of interventions in controlled trials. This meta-analytic sample was extremely heterogeneous, and its effects widely variable, due to imprecision in such a way that increasing *n* will not greatly improve precision. Funnel plots for this meta-analysis revealed a substantial bias. However, this is not the bias one would expect. The FDP is concerned with a number of factors that lead meta-analyses to overestimate a fixed effect of an intervention on a population. Lower *n* studies have less statistical precision, so similar studies, or direct replications, will yield a larger range of main effects compared to larger (and therefore more precise) studies. As smaller studies are easier to conduct, there may be more of them, and only those which reveal a significant main effect may be submitted and published, and is identified by a skew of positive effect sizes at the bottom of the funnel plot. The presence of this type of bias is not visually depicted, but rather a negative skew.

3.1.4.2. CR-PTSD Prevalence

Given that an effect of publication bias was unlikely, the meta-analytic data exclusively for breast cancer survivors indicated that studies using clinical interview methods yielded a mean prevalence of 5.8% for current CR-PTSD, and 11.5% for lifetime CR-PTSD. When all cancer samples were included in the meta-analysis, the prevalence rates were comparable, with current CR-PTSD being found in 6.4% of cases, and lifetime CR-PTSD in 12.6% of cases. The rates of current CR-PTSD in this meta-analysis were remarkably similar to those found by Alter et al., (1996) in the DSM-IV Field Trials (4%), and with the works of Green et al., (1998) and Palmer et al, (2004) which diagnosed current CR-PTSD in 2.5-5% of

cases under standard DSM-IV PTSD criteria (APA, 1994). But, when compared to subsequent epidemiological surveys, the lifetime conditional risk of PTSD for women in the US general population was estimated to be between 10.4% [95% CI=8.8-11.7%] (Kessler et al., 1995) and 13% [95% CI=9.9-16.1%] (Breslau et al., 1998). But the lifetime CR-PTSD rate for the 100% female US sample in Phase IA was 15.4% [95% CI=8.4-26.7%]. Though this was higher than the conditional risk for the US population, it still falls within the confidence intervals for Breslau et al (1998). The prevalence rates from Phase IA are, however, notably higher than the lifetime conditional risk of PTSD due to life-threatening illness [1.1%], suggesting that breast cancer has a potential to be more traumatic than was originally thought. Nevertheless, cautious interpretation must be exercised here. The *a priori* assumption of a random-effects distribution increases the size of the confidence intervals in this phase. But, if only 6.4-12.6% of cancer survivors develop CR-PTSD post-treatment, then the cancer experience may not pose a significant risk of traumatisation. Like Green et al., (1998), Palmer et al., (2004), and now the DSM-5 (APA, 2013) suggest, a trauma framework may not adequately represent the level of stress experienced by most patients.

3.1.4.3. Sources of Heterogeneity

Though PTSD is uncommon in the cancer population, the variability in prevalence suggested that there were factors that contributed to PTSD in the cancer population. The moderator analysis revealed several factors implicated in the fluctuation of prevalence rates across the whole cancer sample. Neither time since diagnosis or treatment were statistically significant moderators of PTSD prevalence rates, with the exception of when lifetime PTSD was being assessed using the SCID, where rates decreased with time post-treatment. This finding should be interpreted with caution, for several reasons. First, this trend (though accounting for a large variance proportion), was only observed for lifetime CR-PTSD, and second, this factor is a proxy variable for time post-trauma. Since there is no agreement as to whether the diagnosis or the treatments are definitive traumas, it appears that many investigators adopted one (but not both) of the two indices; therefore, these moderators could not be combined, giving low statistical power to this analysis. However, a lifetime diagnosis is able to capture PTSD that has either continued or ameliorated since the end of treatment, and may better reflect the epidemiologic trend that non-cancer PTSD sufferers do recover naturally over time, but a minority remain symptomatic after many years (Kessler et al., 1995). The fact that a significant proportion of cancer survivors are at risk of PTSD months to years after their treatment has profound implications for both the patient's psychological and physical health, as potential cases may not be identified during routine follow-up.

Younger aged samples were associated with higher current CR-PTSD event rates on the SCID. However, this trend was just significant, so these results too must be interpreted with caution. Nevertheless, there may be good reason for this. The majority of research into relationship between younger age and PTSD symptom severity concentrates on age at diagnosis – this study-level moderator was the mean age of the sample, and as many of the samples used were many months, or sometimes years, post diagnosis, this likely reduced the sensitivity of the analysis. One strength of this analysis is that it was addressed the impact of younger age on the prevalence rates of lifetime cancer-related PTSD, not on symptom severity. Future research could focus on collating PTSD event rate data and with patient demographics from multiple oncology services both nationally and internationally.

The final aim of this meta-analysis was to synthesise disease-stage data to assess if it is a risk factor for PTSD after Gurevich et al's (2002) comments that much of the research used skewed samples. Post-hoc analyses of sampling distributions suggest that advanced disease is related to an greater CR-PTSD event rates on PCL-C and on the SCID. This is commensurate with studies that show a positive relationship between disease severity and PTSD (Boyer et al., 2002; Jacobsen et al., 1998; Mundy et al., 2000; Posluszny et al., 2011), but cautious interpretation is advised. Though differences in event rates were highly significant, additional factors may have contributed. First, additional factors such as the effect of culture (Europe vs USA), mean sample age, or the differences in care given to those with metastatic disease may have introduced systematic differences between groups. Second, the higher-stage sample in Mystakidou et al., (2012) study was set in palliative care, which introduces environmental and existential factors in the development of PTSD that are not characteristic of lower-staged breast cancer samples. Also, the degrees of freedom from the early-stage group were far larger than for the advanced stage group, but, as Gurevich et al (2002) state, this merely reflects the abundance of earlier-stage cancers in the majority of studies. The comparative lack of significance of disease stage in breast cancer survivors may be because breast cancer's comparative ease of detection, and as such may be diagnosed and treated sufficiently early as to enjoy a better prognosis. This does, however, have clinical implications: fluctuations in PTSD prevalence may be due to factors other than the disease in itself. Of important note was that none of the other moderators (mean sample age, time since diagnosis/treatment) were significant for breast cancer samples. Though much of this finding may be attributable to within-sample variance that is not testable using meta-analytic methods, the lack of significant relationship suggested that variables other than the ones included were implicated in the fluctuation of CR-PTSD prevalence.

3.1.4.4. Limitations and Strengths

It should be noted that there are additional limitations to this analysis. First, only study-level moderators were included. Many included studies did not assess psychiatric history or additional life stress at the time of assessment, so intrapersonal vulnerability factors were unaccounted for in this analysis. Second, the substantial variability in reporting standards, and limitations of using the comprehensive meta-analysis program (CMA), may have introduced variance into analyses that is not attributable to the moderators of interest, and prevented a multiple regression model of all the significant variables. Third, studies that have used questionnaires may over-inflate rates of CR-PTSD due to symptom endorsement being confounded by artefacts of cancer drugs, and medical conditions (PTSD Criterion G; APA, 1994), and realistic fears of cancer recurrence being endorsed as the acceptance of a foreshortened future (Green et al., 1998). Nevertheless, the sensitivity and specificity of the PCL-C against the SCID for PTSD is sufficient to screen for those survivors who are suffering from CR-PTSD. Out of the studies that have used interviews, very few assessed the relative rates of other co-morbid disorders, meaning PTSD still might not be the primary disorder (Kangas, 2013). This, however, does not discount the presence of PTSD. The vast majority of studies also did not provide specifics about the individual experiences throughout the diagnosis and treatment that were endorsed as traumatic, and could shed light on the likelihood of PTSD in this population. Also, all of the studies included in this meta-analysis used DSM-IV criteria, not the new DSM-5. Early epidemiological studies documented the

inflation of PTSD prevalence owing to the revised DSM-IV criteria, so PTSD prevalence may decrease owing to the new DSM-5 PTSD criterion A.

A strength of this investigation was that it included 25 studies and a substantial pool across several populations. This affords our analysis a measure of generalizability that the individual studies could not achieve. Accuracy of these prevalence estimates was enhanced by only including data from questionnaires and clinical interviews that were anchored to the experience of adult cancer. Though this does not take into account differential diagnoses, it does take into account significant PTSD symptoms due to the experience of a serious stressor. At a minimum, the prevalence statistics will at least reflect the proportion of survivors who will meet criteria for one of several presentations that can occur after the experience of extreme stress (adjustment disorder, subsyndromal PTSD and PTSD). This, in itself, is more clinically useful than focusing on PTSD alone. Finally, this investigator found no retrievable record of a published meta-analysis of this kind. Such an empirical synthesis had provided a foundation for understanding the impact of diagnosis and treatment on the psychological wellbeing of cancer survivors. Another strength of this meta-analysis is that it provided the expected prevalence rates for both diagnosable PTSD, and clinically significant PTSD symptoms via cut-off and cluster scoring. This meta-analysis revealed that screening questionnaires yielded much higher rates of caseness-level CR-PTSD symptoms, than did the use of clinical interviews for current CR-PTSD. This is commensurate with Thompson et al's review (2011). The reason for this is apparent: interviews diagnose disorder, questionnaires screen clinically significant symptoms. Screening questionnaires are not clinician-administered, and they do not account for disorders that better explain the symptoms. However, the PCL-C has demonstrated its reliable sensitivity and specificity in correctly identifying clinical cases (Andrykowski et al., 1998), but there is still debate on where to place the cut-off score (Thompson et al., 2011). The Davidson's Trauma Scale (Davidson et al., 1997) can be used as an alternative to screen for CR-PTSD, as it has a variable cut-off depending on the prevalence of PTSD in the population of interest. As this meta-analysis revealed a current PTSD prevalence of 6.4%, the Davidson's cut-off for the cancer survivor population would be 47.

3.1.4.5. Future Improvements

It should be noted that the quality of a meta-analysis is influenced primarily by the specificity of the research question and the criteria used to include studies into the dataset, which in turn are influenced by what is currently known and the quality of available studies. Future research might improve upon this meta-analysis by performing an updated literature search given that this meta-analysis was 14 months out of date at time of publication (August 2014). This may give different results. Similarly, several studies were omitted from this meta-analysis because some researchers did not present analyses on potential moderators of their prevalence rates. Researchers seeking to replicate this meta-analysis might be more persistent in contacting authors and asking for their datasets – especially with regards to unavailable or unclear data on disease stage, psychiatric history, or age. They could analyse the raw data to get standardized figures for each moderator which may strengthen the meta-analysis.

3.1.4.6. Conclusions

Overall, this random-effects meta-analysis revealed that prevalence rates from questionnaires show that a significant minority of cancer survivors with present with clinically significant symptoms as a result of cancer. Prevalence rates from clinical interviews show that a significant minority of cancer survivors meet (or have previously met) full DSM-IV criteria for CR-PTSD after the conclusion of their cancer treatment, and that those who are younger, are diagnosed with more advanced disease, and recently completed treatment, may be at greater risk of PTSD. This data can be used to provide an empirical baseline by which clinicians can determine the proportion of cancer survivors who may be traumatised. However, a critical evaluation of the studies included in the meta-analysis suggested that differential diagnosis was not accounted for and thus necessitated further investigation of breast cancer survivors with possible CR-PTSD. Also, the lack of significant moderators for breast cancer survivors with PTSD suggests that further research was needed to identify other probable factors that contributed to CR-PTSD.

3.2. Study 2: Cancer-related post-traumatic stress disorder (CR-PTSD) in breast and colorectal cancer survivors attending a trauma-focused PTSD Clinic.

3.2.1. Introduction

3.2.1.1. Rationale

The literature in section 2.5 suggested that cancer patients and survivors were a population who were sometimes traumatised by diagnosis and treatment, but yet only a small but widely fluctuating percentage ever met diagnostic criteria for PTSD. Of those that did meet the diagnostic criteria, it was often unclear what experiences generated it, and this has remained so after decades of research, with no useful estimate of CR-PTSD prevalence, or stable predictor variables, to enable oncology specialists to detect these uncommon cases.

Study 1 was designed to establish the prevalence of current CR-PTSD in a population of cancer survivors. The results showed that current CR-PTSD presented in only 6.4% of survivors. The second aim was to identify study-level moderators that significantly contributed to the variance in these rates across the whole cancer pool (disease-stage at diagnosis, time post-diagnosis/treatment, and mean age of sample). However, none significantly contributed to the variance in current PTSD apart for mean age, which was only significant for the complete pool of cancer survivors. Study 1 provided a substantiative estimate of how much CR-PTSD would likely present in an oncology service based on standardised clinical interviews which were anchored to the experience of cancer. Nevertheless, it was argued that the prevalence rates in the included studies were inflated because they either did not implement, or did not provide data for, differential diagnoses for adjustment disorder (Kangas, 2013). This is a valid concern, as the research into PTSD in cancer survivors is largely absent of data recording the specific traumatic aspects of cancer. This may rest on an assumption that aspects of the cancer experience were intrinsically traumatic. However, the absence of trauma-specific data also does not discount the occurrence on cancer-related trauma.

Though differential diagnosis via interview is a significant issue in the literature, questionnaires (such as the often used PCL-C) are necessary to screen in for interview those who are most likely to suffer from PTSD. However, questionnaires with cut-off scores may also misrepresent the rates of CR-PTSD in this population (Thompson et al., 2011), or they may have insufficient power to detect true cases in this population (Andrykowski & Cordova, 1998). It is for this reason that Study 1 used the establish CR-PTSD prevalence rate (6.4%) to set an evidence-based clinical cut-off on the Davidson's Trauma Scale (Davidson et al., 1997). However, it was unable to examine the role of several important factors implicated in the development of PTSD. Significant pre-trauma variables such as trauma/psychiatric history, though implicated in this population (Andrykowski et al., 1998; Green et al., 2000), were not accounted for. Also, the additional role of emotional and cognitive coping strategies such as dissociation (Kangas et al., 2005), suppression (Amir & Ramati, 2002), and rumination (Chan et al., 2011), though significantly implicated in PTSD symptom severity, were studied infrequently and could thus not be subjected to meta-analysis.

For the reasons cited above, there is a need to a) differentially diagnose CR-PTSD from adjustment disorder (AD) and subsyndromal PTSD symptoms (SS-PTSD); b) gather qualitative data on what specific experiences cancer survivors endorse as traumatic; and c), determine the contribution of emotion-regulation variables (suppression, rumination, and dissociation) to the development of CR-PTSD, and whether these variables provide clinic utility in screening for CR-PTSD. This will provide important clinical information that may contribute to the course of adjustment throughout the experience of cancer. This study employed a mental health sample of cancer survivors who were likely to present with clinically significant PTSD symptoms, ranging from the subsyndromal (but nevertheless impairing) adjustment-related reactions related to cancer, to the full diagnosis of cancer-related PTSD. This was needed to address the issues regarding differential diagnosis (Kangas, 2013) and study the distinctive features of CR-PTSD and adjustment disorder (AD).

3.2.1.2. Research Aims

The aims of this study were as follows: 1) to determine the clinical utility of the new cut-off for the Davidson's Trauma Scale (47) as suggested in Study 1; 2) to examine if CR-PTSD, subsyndromal PTSD, and adjustment disorder (AD) groups differ in their endorsement of emotion regulation strategies (rumination, suppression, avoidance, dissociation); 3) to examine how much variance in PTSD symptom severity is explained by emotion regulation variables; and 4) determine the cancer experiences identified as index traumas in this sample.

3.2.1.3. Hypotheses

1) The high cut-off on the Davidson Trauma Scale will increase sensitivity to CR-PTSD.
2) The CR-PTSD group will a) endorse a greater use of suppression and a greater experience of emotional numbing, compared to the subsyndromal and adjustment disorder groups; and b) that the CR-PTSD group will exhibit PTSD symptom scores above the Davidson's cut-off compared to the non-PTSD groups.

3) Suppression, rumination, avoidance, and emotional numbing will account for a statistically significant proportion of the variance, with each variable providing and independent contribution.

3.2.2. Methods

3.2.2.1. Participants

The participants (*n*=58, 7 male, 51 female) finished treatment for either breast or colorectal cancer, were disease-free, and survived for at least six months with the expectation of continued survivorship. They were all within the Dorset and Hampshire catchment area for Poole and Royal Bournemouth and Christchurch Hospital's oncology service. They were referred (between 2008 and 2014) to the PTSD clinic at Royal Bournemouth Hospital by an oncology specialist doctor or nurse if the patient reported unremitting psychological difficulties at follow-up appointments - namely cognitive intrusions, nightmares, or flashbacks, related to specific aspects of their cancer experience (DSM-IV PTSD Criteria A1/2, and B1/2; APA, 1994). All referred patients attended a full clinical interview with a consultant clinical psychologist. Patients 1) suffering from, or having a history of, psychotic illness, or those who 2) at the time of assessment and treatment were not stabilised, so were at risk from others, or a serious risk to self (e.g. actively suicidal), were referred to the relevant community mental health teams for their catchment area. All patients included in the clinic were invited to contribute their anonymous data to peer-reviewed research, and provided full written informed consent. Those patients who did not provide consent still received therapy from the service.

3.2.2.2. Ethics

All information regarding the staging and date of cancer diagnosis, type and sequence of treatments, including complications, number of recurrences and metastases, were abstracted from the clinic's files by the primary author (GA). Permission to access patient records such as referral letters, therapy notes, and questionnaires was granted by REC (26/09/12) and no sensitive information was accessed outside the care team (Q11 of REC 26/09/12). Access was confirmed by the Research Lead/Gatekeeper to the Service and Patient Records. To protect confidentiality, all data were made anonymous using a study-specific code, entered into an Excel file. Clinical information about patients (such as diagnosis of PTSD) outside of their questionnaire data were obtained, anonymously, from the consultant clinical psychologist (RB) and then attached to each specific code. All anonymous data was then transferred into the SPSS statistical package for analysis. The data was abstracted and analysed after approval was obtained from the Research and Development Lead at Royal Bournemouth Hospital and is also covered by the ethics application to the NRES Committee London – Dulwich (REC Reference 12/LO/0236) and Royal Bournemouth Hospital R&D.

3.2.2.3. The PTSD Clinic

The Royal Bournemouth Hospital PTSD Clinic was started by Consultant Clinical Psychologist Roger Baker (RB) and Oncology Nurse Specialist Lin Purandare (LP) in 2008 to respond to the psychological needs of cancer survivors who, when attending their follow-up clinics, complained of unremitting

psychological difficulties comprising of overwhelming cognitive intrusions, that in some cases presented as cancer-related post-traumatic stress disorder. The PTSD Clinic provided a trauma-focused prolonged-exposure therapy (as per recommended guidelines; NICE, 2005), that was reconceptualised into an emotional exposure-based framework which approaches therapy from the view that the way patients relate to their emotions is implicated in their risk of PTSD, as well as their chances of recovery. The therapeutic focus was on training their patients to learn new ways of relating to, and experiencing, their own emotions, in order to facilitate recovery, and increase future emotional resiliency prior to exposure to improve the therapeutic outcome of prolonged-exposure (Baker, Gale, Abbey, & Thomas, 2013).

Emotional Processing Therapy [EPT] was developed from Baker's emotional processing model (Baker, 2007), which draws on and synthesises concepts of emotional processing (Rachman, 1980; 2001), physiological habituation (Marks, 1979), emotional processing theory (Foa & Kozak, 1986), experiential focusing (Gendlin, 1981), and multiple code theory (Bucci, 1997; 2001). It's mechanisms of action are based on emotional processing theory (Foa & Kozak, 1986), and therefore also involve all the elements of prolonged exposure (Foa & Hembree, 2007), and is thus similar to other NICE-recommended trauma-focused therapies to date. However, EPT is different in that it approaches the practice of prolonged exposure from the context of pre-existing, and unhelpful, emotional processing and coping styles that according to Baker (2010) not only are a contributing vulnerability factor in the risk of developing PTSD, but also preclude the processing of distressing memories and the associated affect, maintaining the disorder. The aim of emotional processing therapy is therefore not only to enable the processing of current trauma, but also to modify pre-morbid emotional processing styles to facilitate full, effective, engagement in the exposure, and carry this new strategy into their lives after therapy. Several sessions are dedicated towards implementing ways to adapt and change these styles for the ease of processing current and future emotional distress. EPT operates on the assumption that prolonged exposure therapy is, at its core, exposure to emotional experience, rather than a purely behavioural exposure, because the act of confronting previously avoided distressing memories allows the patient to be exposed to powerful and distressing emotional experiences. At this point, memories can then be processed effectively to the point that the recall of memories no longer disrupts day-to-day functioning (Rachman, 2001).

EPT consists of 12, 50 minute sessions. The first three focus on processing styles. Session 1 involves emotional preparation of the client by exploring their current emotional processing style. Session 2 focuses on providing them with skills to practice a style that is more open and accepting. Finally, session 3 provides the patient with psycho-education on the nature of unprocessed memories, how prolonged exposure therapy works, and how to devise action plans of self-care when dealing with the impact of facing the distress. The session will also address the patients' expectation of the therapy and its outcome. The remaining nine sessions are conducted as prolonged exposure therapy, but within the context of this emotional framework. For a full explanation of EPT's protocol, see Baker (2010).

3.2.2.4. Materials

The Davidson Trauma Scale (DTS; Davidson et al., 1997): The DTS is a 17-item self-report questionnaire that assesses the frequency and severity of PTSD symptoms by total or by symptom cluster according to DSM-IV diagnostic criteria for PTSD. Higher scores index greater frequency and/or severity

of the PTSD symptom. There are three subscales - measuring intrusions (Criterion B), avoidance/numbing (Criterion C), and hyper arousal (Criterion D). The DTS is administered to assess the patient's response to individual traumatic events. The patient is orientated to this by a question asking the patient to identify their most disturbing trauma (DSM-IV PTSD Criterion A1). For this study, the PTSD syndrome was anchored to the experience of cancer diagnosis and treatment. The DTS cut-off score can be varied depending on the assumed prevalence of PTSD in the trauma population of interest. Recent meta-analyses revealed a current cancer-related PTSD prevalence of <10% (Study 1), so the DTS cut-off used for this study is 47. The DTS has good internal consistency, good concurrent and construct validity (Zlotnick et al., 1996), and good convergent and divergent validity (Davidson et al., 1997).

The *Delusions Symptoms States Inventory (DSSI; Bedford & Foulds, 1978)*: The DSSI is a seven subscale self-report questionnaire that measures the severity and frequency of mood states and psychiatric symptoms from the preceding two-three weeks. Whereas the DSSI contains a hierarchical array of subscales, this study uses the neurotic subscales (somatisation, obsession, rumination, phobia, and dissociation), and the mood subscales (depression, and anxiety), but exclude the last two levels (integrated, and disintegrated delusions). This leaves the DSSI with 49 remaining items. The DSSI has demonstrated validity and sensitivity to therapeutic change (Baker et al., 1998).

The Emotional Processing Scale (EPS-25; Baker et al., 2010): The EPS-25 is a 25-item self-report questionnaire that assesses emotional processing styles and deficits. Two questions ask the respondent to write their strongest most pleasant and most unpleasant emotional experience from the past week, and these responses are used to orientate the respondent to the 25-item questionnaire. This component uses a 10-point Likert-type ordinal scale, where higher scores measure higher degrees of emotional processing impairment on each subscale, and on the total score. Emotional processing styles are measured across five subscales collapsed into three facets of Baker's emotional processing model (Baker, 2007): *control* (suppression and avoidance), *experience* (impoverished and unprocessed), and *expression* of emotion (unregulated). The EPS-25 is able to discriminate between healthy controls and persons with mental health difficulties. The cut-off scores for each subscale are as follows: *total* [5], *suppression* [5.5], *avoidance* [4.5], *impoverished* [3.5], *unprocessed* [6.5], *and unregulated* [4.5] (Baker, Thomas, Thomas, Santonastaso, & Corrigan, 2013). The EPS-25 has demonstrated considerable internal consistency (Cronbach's ά = .92) and convergent validity with measures of symptomatology on the Delusions Symptoms States Inventory, and alexithymia on the Toronto Alexithymia Scale (Baker et al., 2007).

3.2.2.5. Data Abstraction & Analysis

The analysis was conducted in several steps: First, prevalence point estimates were calculated from clinical interviews for a) cancer-related PTSD, b) subsyndromal/adjustment disorders, c) primary disorders such as depression/anxiety disorders. Second, Multivariate ANOVAs were performed to check for significant differences between PTSD and no PTSD groups, on emotional processing deficits (EPS-25), anxiety/depression symptoms, and PTSD symptoms (DTS) at time of assessment. Third, multiple linear regression was used to assess the contribution of emotion regulation strategies to PTSD symptom severity. Fourth, this study assessed the ability of the DTS to screen for the presence of CR-PTSD with the new cut-off of 47 that was established from the CR-PTSD prevalence rate found in Study 1, and the ability of the EPS-25 to discriminate between PTSD and Adjustment Disorder. This was done in the

following ways. First, by calculating the probability of achieving a positive screen if the patient does have CR-PTSD, where *sensitivity* = [n_{true+} / (n_{true+} + n_{false-})], and probability of a negative screen if the patients does not have CR-PTSD, where *specificity* = [n_{true-} / (n_{true-} + n_{false+})]. And second, by calculating the percentage of patients with a positive test who actually have CR PTSD (the positive predictive value), where *PPV* = [n_{true+} / (n_{true+} + n_{false+})] and the percentage of patients with a negative test who do not have CR-PTSD (negative predictive value), where *NPV* = [n_{true-} / (n_{true-} + n_{false-})].

3.2.3. Results

3.2.3.1. Sample Characteristics

The sample consisted of *n*=58 cancer survivors (7 male, 51 female) who were diagnosed at a mean age of 47.33 years (*SD* = 10.52 years), whereas the mean age at clinic referral was 51.65 years (*SD* = 11.75 years). The distribution of cancer types in the sample was 65.5% breast cancer, 12% colorectal, 1.7% haematological, 1.7% lymphoma, and 19% not recorded. The distribution of disease stages at diagnosis was 24.1% stage II, 17.2% stage III, with 41.4% also not recorded. The types of treatment included chemotherapy, radiotherapy, and various surgical procedures (including mastectomy – both complete and breast conserving). In this clinical sample, *n*=43 received chemotherapy, *n*=28 surgery, and *n*=11 radiotherapy, of which *n*=18 had one treatment only (*n*=5, surgery, *n*=13, chemo), *n*=24 had two treatments (*n*=21 had chemotherapy and surgery, *n*=3 had radio and chemotherapy) and *n*=2 had all three treatments. The mean time between cancer diagnosis and assessment at PTSD Clinic was 50.7 months (*SD* = 44.23 months).

3.2.3.2. CR-PTSD Prevalence Statistics

67% of the clinical sample (*n*=39) met caseness for probable CR-PTSD when using the cut-off score of 47 as suggested in Study 1. *N*=12 of the sample were under the cut-off, whereas *n*=7 did not have any recorded DTS scores at assessment. In addition, the entire sample attended a full clinical assessment with the consultant clinical psychologist (RB) and oncology specialist (LP). Of this sample, *n*=24 (41%) received a primary diagnosis of CR-PTSD, *n*=15 a diagnosis of cancer-related subsyndromal CR-PTSD (SS-PTSD), and *n*=10 cancer-related adjustment disorder (AD). Of the remaining sample, *n*=3 were diagnosed with PTSD due to different index traumas (*n*=1, combat exposure flashbacks reactivated by cancer treatment, *n*=1, childhood sexual abuse, and *n*=1, traumatic injury with a medical device), and *n*=6 received other primary diagnoses (*n*=1 panic disorder, *n*=1 depression, *n*=1 phobia, *n*=1 chronic fatigue syndrome, *n*=1 grief, and *n*=1 no diagnosis). *N*=3 CR-PTSD patients suffered interpersonal traumas prior to the cancer. No other patients reported previous traumatic events other than the index trauma.

3.2.3.3. Sensitivity and Specificity at Assessment

As the results have shown, there is an observed difference between the CR-PTSD prevalence rates yielded by clinical interview (41%), and those suggested by meeting the clinical cut-off on the Davidson's Trauma Scale (67%). Table 5 shows the sensitivity-specificity and predictive value statistics

for the Davidson's Trauma Scale, and the EPS-25 subscales. The DTS in particular demonstrated excellent sensitivity to CR-PTSD, effectively screening in 90% of all true cases, which is an improvement on the sensitivity of the PCL-C in this population (Andrykowski et al., 1998). But, it did demonstrate a much weaker specificity to CR-PTSD (27%), having also screened in 72% of all adjustment and subsyndromal cases. Though the DTS served as a sensitive screening tool for CR-PTSD (90%), the lack of specificity (27%) introduced many false positives. The predictive value of these scales is also depicted in Table 5. The DTS screened in as many AD cases as CR-PTSD cases. It did however, have a much higher NPV (75%), suggesting it does correctly classify 75% of non CR-PTSD cases. The EPS-25 also demonstrated superior positive and negative predictive value to the DTS.

Table 5 **:** Ability of the DTS and EPS to detect CR-PTSD when diagnosed via clinical interview.

Diagnosis	DTS Caseness		EPS-25 Caseness											
	Total		Total		Suppression		Avoidance		Impoverished		Unprocessed		Unregulated	
	Yes	No	Yes	No	Yes	No	Yes	No	Yes	No	Yes	No	Yes	No
CR-PTSD	19	2	17	4	15	7	18	4	17	5	13	9	15	7
No (AD & SS-PTSD)	16	6	9	15	8	16	13	11	13	11	9	15	10	14
Sens*	90%		81%		68%		82%		77%		59%		68%	
Spec*	27%		63%		67%		46%		46%		63%		58%	
PPV*	54%		65%		65%		58%		57%		59%		60%	
NPV*	75%		79%		70%		73%		73%		63%		67%	

Key: *
SENS = the probability of a positive test, if the patient does have CR-PTSD
SPEC = the probablity of a negative test if the patient does not have CR-PTSD.
PPV = Positive Predictive Value: the percentage of patients with a positive test who actually have CR-PTSD
NPV = Negative Predictive Value: the percentage of patients with a negative test who do not have CR-PTSD

3.2.3.4. Group Differences

3.2.3.4.1. Disease Variables (Study 1):

A series of One-Way Independent ANOVAs were conducted to assess the contribution of disease variables to PTSD symptom severity and emotional processing problems. The results showed that there were no significant differences in subscale scores between a) disease stage groups (Stage II, III, and not recorded), or b) those who had a recorded diagnosis of cancer or those without. However, there were significant differences in PTSD symptom severity (DTS Total) between those diagnosed with breast or colorectal cancer [F (1, 39) = 7.50, p =.009], with the breast cancer group scoring higher [M = 72.5, SD = 31.57, n=36], than the colorectal group [M = 32.6, SD = 19.26, n=5]. Additional differences were found on emotional processing difficulties [F (1, 39) = 5.05, p =.03], with the breast cancer group reporting more severe difficulties [M = 5.32, SD = 1.85] than the colorectal group [M = 3.64, SD = 1.55] on the EPS-25 Total Score.

3.2.3.4.2. The Davidson's Trauma Scale:

The clinical sample was grouped by whether they were above or below the cut-off on the DTS, and entered into a series of statistical tests. First, a One-Way Independent ANOVA was used to ascertain group differences in mean age at diagnosis, age at referral to clinic, time since diagnosis, and clinical variables (DTS, EPS-25, and DSSI subscales). Table 6 shows that there was a significant difference in mean age at diagnosis [F (1, 37) = 9.38, p =.04], where those in the caseness group were diagnosed younger [M = 44.5, SD = 9.32] than those who did not meet caseness [M = 56.25, SD = 10.99]. Similarly, those who were in the caseness group presented at the clinic at a younger age [M = 49, SD = 10.5], than those who were below [M = 56.9, SD = 15], and this difference too was statistically significant [F (1, 48)

= 4.48, p =.04]. However, there was not a significant group difference in time post-diagnosis [F (1, 37) = 1.68, p >.05]. An analysis of the clinical variables in Table 6 revealed significant differences in signs of unprocessed emotion [F (1, 48) = 6.49, p =.01], where those above the cut-off [M = 6.65, SD = 1.68] experienced more distressing symptoms than those who were below [M = 5.17, SD = 1.99]. Similarly, depression scores were significantly different between groups [F (1, 48) = 6.96, p =.01], with those above caseness experiencing them more severely [M = 6.81, SD = 4.25] than the non-caseness group [M = 3.17, SD = 3.9]. All other variables were not significant, apart from the DSSI Phobia scale, but this scale did not meet homogeneity of variance assumptions.

3.2.3.4.3. By Diagnostic Category:

Table 7 presents the same data, but grouped according to primary diagnosis from the clinical interviews. The independent variable (primary diagnosis) had five conditions 1) CR-PTSD, 2) AD, 3) SS-PTSD, 4) Other PTSD, and 5) other primary diagnosis, such as depression. A One-Way independent ANOVA was conducted, with the DTS, EPS-25, and DSSI subscales as the dependent variables (DV). Bonferroni post-hoc tests revealed no significant differences between AD and SS-PTSD on any clinical scale [p>.05]. Though there were significant differences between CR-PTSD and a) AD [p = .03], and b) SS-PTSD [p = .03], the differences were only on the DTS Total subscale, and the same for both comparisons. Given the statistical and clinical similarity between SS-PTSD and AD diagnoses, they were collapsed into one group of equal size (n=23), for all subsequent analyses. Subsequently, a Multivariate Analysis of Variance (MANOVA) was conducted to detect group differences (CR-PTSD *v* AD/SS-PTSD) in PTSD symptoms (DTS), emotional processing difficulties (EPS-25), and anxiety/depression symptoms (DSSI). The analyses revealed that the CR-PTSD group reported greater distress and more emotional processing difficulties than the AD/SS-PTSD group [λ = .524, F (9, 32) = 3.225, p = .007].

According to the univariate analyses **(**Table 8**)**, those who were diagnosed with CR-PTSD were not significantly younger at diagnosis [F (1, 34) = 0.34, p =.57], or referral [F (1, 45) = 0.46, p =.50] than those who were not. There was a significant difference in DTS Total score [F (1, 41) = 14.42, p =.01], with the CR-PTSD group exhibiting much more severe PTSD symptoms [M = 87.38, SD = 29.18] than the AD/SS-PTSD group [M = 54.95, SD = 26.81], although both these means were above the 47 cut-off score established from the Study 1 CR-PTSD prevalence rate. These differences were also reflected on the remaining DTS subscales. There were also significant differences in generalized anxiety [F (1, 44) = 6.66, p =.01], where those with adjustment disorder or subsyndromal symptoms experienced less distress [M = 5.74, SD = 3.61] than those with CR-PTSD [M = 8.61, SD = 3.93]. The CR-PTSD group also endorsed significantly more frequent symptoms of depression than the AD/SS-PTSD group [F (1, 44) = 5.33, p =.03]. Though symptoms of depression and anxiety were more frequent and severe for the CR-PTSD group, there were also substantial between-group differences on the EPS-25. First, there was a significant between-groups difference in EPS Total Score [F (1, 44) = 5.18, p =.03], where the CR-PTSD group endorsed more emotional processing difficulties [M = 5.93, SD = 1.63, above the cut-off], than the AD/SS-PTSD group [M = 4.78, SD = 1.79, below the cut-off]. A more specific analysis of the EPS subscales revealed significant differences in suppression use [F (1, 44) = 5.48, p =.02], and problems experiencing emotions [F (1, 44) = 4.82, p =.03], with the CR-PTSD group using greater suppression [M = 6.51, SD = 2.08, above cut-off] and experiencing more impoverished emotions [M = 5.26, SD = 2.05],

than the AD/SS-PTSD group. The AD/SS-PTSD group were below the EPS-25 cut-offs for suppression [$M = 5.14$, $SD = 1.88$] and impoverished emotional experiences [$M = 3.92$, $SD = 2.11$].

Table 6: Descriptives (Mean, SD) and One-Way Independent ANOVA for DTS Caseness and No Caseness Groups

		Descriptives				Inferential Statistics		
		Caseness (n=38)		Below Cut--Off (n=12)		*F*	*df*	*p*
		Mean	SD	Mean	SD			
	Age At Referral	48.95	10.05	56.92	14.98	4.48	1, 48	.04
	Age At Diagnosis	44.52	9.32	56.25	10.99	9.38	1, 37	.04
	Months Post Dx	56.03	47.78	33.5	18.85	1.68	1, 37	NS
EPS-25	Total	5.52	1.47	4.66	2.3	2.35	1, 48	NS
	Suppression	6.01	1.92	5.45	2.01	0.76	1, 48	NS
	Avoidance	5.44	1.9	5.63	1.96	0.09	1, 48	NS
	Impoverished	4.65	1.96	3.77	2.77	1.52	1, 48	NS
	Unprocessed	6.65	1.68	5.17	1.99	6.49	1, 48	.01
	Unregulated	4.82	1.88	4.15	2.32	1.02	1, 48	NS
DSSI	Anxiety	8.28	3.73	2.75	2.63	22.69	1, 48	.01
(Severity)	Somatic	1.71	1.52	0.83	0.94	3.53	1, 48	NS
	Obsessional	1.61	2.15	0.75	1.29	1.69	1, 48	NS
	Depressive	6.81	4.25	3.17	3.9	6.96	1, 48	.01
	Phobic	1.89	2.13	0.17	0.39	7.72	1, 48	.01
	Ruminative	2.87	2.56	1.58	2.02	7.72	1, 48	NS
	Dissociative	0.82	1.49	0.17	0.39	2.21	1, 48	NS
DSSI	Anxiety	5.18	1.54	2.58	1.93	23.02	1, 48	.01
(Frequency)	Somatic	1.21	1.09	0.67	0.65	2.64	1, 48	NS
	Obsessional	1.05	1.33	0.58	0.9	1.29	1, 48	NS
	Depressive	3.89	2.04	2.42	2.15	4.68	1, 48	.04
	Phobic	1.26	1.27	0.17	0.39	8.6	1, 48	.01
	Ruminative	1.79	1.36	1.17	1.27	1.98	1, 48	NS
	Dissociative	0.45	0.76	0.17	0.39	1.5	1, 48	NS

Table 7 : Descriptive statistics for the PTSD Clinic

		Other Disorder (*n* =6)		Other PTSD (*n* =3)		AD (*n* =9)		SS-PTSD (*n* =15)		CR-PTSD (*n* =24)	
		Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
	Age At Referral	65.83	8.18	65.33	8.6	50.11	14.15	49.87	11.03	48.1	8.84
	Months Post Dx	46	21.99	105	0	28.8	22.75	61.31	53.22	47.63	45.37
DTS	Total	41.4	40.2	60	2.83	52.11	27.16	56.36	26.49	87.38	29.18
	Intrusion	12.4	11.78	30	1.41	14.56	7.21	19.71	9.68	25.19	11.44
	Avoid	13.6	18.39	9.5	0.07	19.11	15.07	19.36	13.05	33.24	14.29
	Hyper	15.2	12.28	20.5	3.53	18.44	10.6	18.71	8.79	28.67	9.05
EPS-25	Total	4.24	1.68	2.88	1.92	4.81	2.29	4.73	1.37	5.93	1.63
	Suppression	4.93	1.2	3.9	2.97	5.34	2.02	5.17	1.89	6.51	2.08
	Avoidance	5.17	2.11	4.2	2.83	4.86	2.23	5.2	1.75	5.81	1.8
	Impoverished	3.27	2.18	1.8	2.55	4.38	2.62	3.56	1.61	5.26	2.05
	Unprocessed	4.4	2.7	2.5	3.54	6.22	1.5	5.64	2.21	6.78	1.82
	Unregulated	3.43	1.73	2	2.83	4.36	2.81	4.08	1.33	5.3	1.89
DSSI	Anxiety	3.67	2.73	3.67	5.51	5.7	3.71	6.29	4.02	8.61	3.93
(Severity)	Somatic	1.17	1.6	2	1.73	1.6	1.84	1.21	0.97	1.7	1.49
	Obsessional	1	1.55	0.67	1.15	1.3	1.77	1	1.84	1.7	2.27
	Depressive	2.67	2.34	3.3	3.21	7.7	6.11	3.71	3.69	7.3	3.99
	Phobic	1.17	2.04	2	1.73	1.3	1.95	0.57	0.93	2.3	2.48
	Ruminative	1.17	2.04	1.33	1.53	2	2.54	2.14	1.96	3.04	2.79
	Dissociative	0.67	1.21	0	0	0.2	0.42	0.43	0.94	0.91	1.7

Given that mean sample age was implicated as a moderating factor in Study 1, and age at referral significant in the univariate analysis, it was then entered as a covariate for MANOVA. Age was a significant covariate, and mildly reduced the multivariate effect, but not to non-significance [$\lambda = .533$, F

(9, 30) = 2.919, p = .013]. However, the univariate outcomes (when adjusted for age at referral) revealed that there was no longer a significant group difference in suppression, [F (1, 44) = 3.42, p =.07], or total emotional processing score [F (1, 44) = 3.89, p =.06]. The only remaining difference on the EPS-25 was in impoverished emotional experience, which was more severe in the CR-PTSD group, but a marginally weaker effect [F (1, 44) = 4.78, p =.04]. Though impoverished emotional experience differentiated CR-PTSD and SS-PTSD/AD groups, so did depression symptoms in an earlier analysis. Given that emotional numbness is an associated feature of depression, an Analysis of Covariance (ANCOVA) was conducted to test the hypothesis that depression symptoms could account for the problems experiencing, labelling, and linking emotions (the dependent) between the two diagnostic groups. When frequency of depression symptoms were added as a covariate, the group difference in the impoverished subscale was rendered non-significant [F (1, 44) = 2.03, p =.16].

Table 8: Descriptives (Mean, SD) and One-Way Independent ANOVA for CR-PTSD and AD/SS Groups

		Descriptives				Inferential Statistics		
		AD / SS-PTSD (n =23)		CR-PTSD (n =23)		F	df	p
		Mean	SD	Mean	SD			
DTS	Total	54.95	26.81	87.38	29.18	14.42	1, 41	.01
	Intrusion	18.27	8.76	25.19	11.44	4.98	1, 41	.03
	Avoid	19.31	13.85	33.24	14.29	10.52	1, 41	.01
	Hyper	18.27	9.37	28.67	9.05	13.65	1, 41	.01
EPS-25	Total	4.78	1.79	5.93	1.63	5.18	1, 44	.03
	Suppression	5.14	1.88	6.51	2.08	5.48	1, 44	.02
	Avoidance	5.16	1.9	5.81	1.8	1.45	1, 44	.24
	Impoverished	3.92	2.11	5.26	2.05	4.82	1, 44	.03
	Unprocessed	5.87	1.99	6.78	1.82	2.58	1, 44	.12
	Unregulated	4.28	2	5.3	1.89	3.13	1, 44	.08
DSSI	Anxiety	5.74	3.61	8.61	3.93	6.66	1, 44	.01
(Severity)	Somatic	1.43	1.38	1.7	1.49	0.38	1, 44	.54
	Obsessional	1	1.71	1.7	2.27	1.38	1, 44	.25
	Depressive	5.13	5.1	7.3	3.99	2.59	1, 44	.12
	Phobic	0.87	1.49	2.3	2.48	5.68	1, 44	.02
	Ruminative	1.96	2.12	3.04	2.79	2.22	1, 44	1.4
	Dissociative	0.35	0.78	0.91	1.7	2.01	1, 44	.16
DSSI	Anxiety	4.09	2.17	5.39	1.16	6.46	1, 44	.01
(Frequency)	Somatic	1.04	0.98	1.26	1.05	0.53	1, 44	.47
	Obsessional	0.74	1.29	1.09	1.2	0.9	1, 44	.35
	Depressive	2.87	2.36	4.3	1.81	5.33	1, 44	.03
	Phobic	0.57	0.84	1.3	1.26	5.47	1, 44	.02
	Ruminative	1.3	1.36	1.91	1.28	2.44	1, 44	.13
	Dissociative	0.26	0.54	0.43	0.79	0.76	1, 44	.39

3.2.3.5. Emotion Regulation Strategies and PTSD Symptom Severity

As the CR-PTSD and AD/SS-PTSD groups were initially differentiated by the clinical cut-offs for suppression and impoverished emotional experience, a multiple linear regression was conducted to examine the variance contributions of emotion regulation strategies (suppression, behavioural avoidance,

rumination, dissociation) and the role of impoverished emotional experience on PTSD symptom severity (DTS Total Score). Assumptions were met for multi co-linearity, with no tolerance data below .46, (Menard, 1995), no VIF data exceeding 2.2 (Myers, 1990), and no correlation between residuals (Durbin-Watson, $d = 2.00$). The model was able to explain 27% of the variance [*Adjusted* $R^2 = .27$], which was found to significantly predict PTSD symptom severity [$F\ (5, 43) = 4.56$, $p < .01$]. Table 9 shows the co-efficients, where of the five predictors; only two variables provided independent variance contributions. Impoverished emotional experience [$\beta = 5.66$, $t = 2.05$, $p < .05$], and rumination [$\beta = 4.49$, $t = 2.20$, $p < .03$], were related to more severe PTSD symptoms. Dissociation, suppression, and avoidance, did not significantly contribute to the regression model.

Table 9: Multiple regression of emotion regulation strategies on PTSD symptom severity (DTS Total).

Variable	Weight	Unstandardised Co-efficients		Standardised Co-efficients		
		B	*SE*	*B*	*t*	*p*
Impoverished	1	5.66	2.77	.37	2.05	.05
Rumination	2	4.49	2.04	.33	2.20	.03
Dissociation	3	5.58	3.85	.22	1.45	.15
Suppression	4	-3.12	2.79	-.18	-1.11	.27
Avoidance	5	-1.54	2.90	-.09	-.53	.60

3.2.3.6. Predictors of Outcome

It was also prudent to assess the ability of time 1 measurements to predict treatment outcome. After treatment completion and follow-up, n=9 individuals completed and returned the DTS, EPS-25 and DSSI, of which n=6 were initially diagnosed with AD or SS-PTSD, n=2 with CR-PTSD, and n=1 with PTSD due to war trauma. An independent One-Way ANOVA was run to ascertain any group differences (those who returned time 2 questionnaires *v* those who did not) in Time 1 symptoms (DSSI, EPS, DTS), and there was a significant difference in depression scores at time 1 [$F\ (1, 54) = 6.27$, $p = .015$], where those who did not return time 2 questionnaires were significantly more depressed at Time 1 [$M = 6.40$, $SD = 4.59$] than those who did return time 2 questionnaires [$M = 2.44$, $SD = 2.51$]. To see whether time 1 assessment scores were predictive of scores at time 2 follow-up, the remaining data were entered into a Pearson's correlation (two-tailed). The majority of subscales at T1 yielded no significant relationships with Time 2 PTSD symptoms. In particular depression frequency at time 1 did not predict PTSD symptoms at Time 2 [$r = .19$, $p = .63$]. The DTS Total score at Time 1was positively correlated with DTS score at Time 2 [$r = .74$, $p < .01$], suggesting that initial symptoms predict treatment outcome. Finally, impoverished emotional experience at time 1 was a highly significant predictor of PTSD symptom severity at time 2, for DTS Total [$r = .90$, $p < .01$], intrusions [$r = .69$, $p < .05$], avoidance [$r = .82$, $p < .01$], and hyper-arousal symptoms [$r = .93$, $p < .01$], suggesting that problems experiencing and labelling emotions were implicated in the treatment response.

3.2.3.7. Qualitative Findings

All cancer survivors who attended assessment at the PTSD Clinic were asked to complete the Davidson's trauma scale, stating the severity and frequency of the PTSD symptoms (Criteria B-D), but in the context of specific cancer events (Criterion A1). All clinic attendees wrote down on the DTS that the cancer was their traumatic stressor, but many patients provided additional information regarding their experience. For example, frequent comments were a) the pain of needles penetrating the body, b) the severe and persistent sickness during chemotherapy, and c) the loss of, or pervasive damage and scarring to, body parts. In addition, the therapy notes revealed more information on fear of cancer recurrence (FOR). Though FOR was frequently reported in therapy (clinical notes), it was never related to the fear of death in this sample, but rather to the fear of having to endure the treatment again due to traumatic pain and loss of physical attributes and/or abilities at time of first treatment. Though this information was preliminary and anecdotal, further discussion with RB & LP revealed that FOR often ameliorated when clinic attendees were told that cancer recurrences were treated with a view toward improving quality of life, rather than a curative outlook which required aggressive treatment.

3.2.4. Discussion

3.2.4.1. Findings

Though there have been several studies into the role of emotional regulation and cognitive coping strategies in maintaining or exacerbating CR-PTSD symptoms, these have not been widely replicated, and therefore this area is under researched. The central aim of Study 2 was to elucidate differential features of CR-PTSD; and to the best of the investigator's knowledge, this study is the first of its kind in the CR-PTSD in literature. The results showed that 67% of the clinical sample was above cut-off on the DTS, suggesting clinically-significant CR-PTSD symptoms, whereas clinical interviews showed that 41% were actually diagnosed with CR-PTSD. The differences in CR-PTSD prevalence rates are concurrent with the cancer literature which reflects the trend that questionnaires inflate the rates of PTSD (Thompson et al, 2011), although in this study it may be more indicative of subtle but nevertheless clinically significant differences between the two diagnostic groups. A subsequent analysis suggested that the sensitivity of the cut-off was sufficient to screen in 90% of all CR-PTSD cases - an improvement on the sensitivity of the PCL-C in this population (Andrykowski et al., 1998). But, the DTS cut-off also demonstrated a much weaker specificity to CR-PTSD (27%), having also screened in 72% of all subsyndromal and AD cases. However, the EPS-25 Total Score demonstrated a higher level of specificity, and slightly superior PPV and NPV, that the DTS. Much of the superior specificity and NPV for the EPS-25 is due to the cut-off being set to distinguish between those who are psychologically healthy from those who have a diagnosable disorder (Baker et al., 2013). As such, the EPS-25 may be useful in screening out those who may be suffering from a sub-clinical adjustment disorder, but the sample was not sufficiently large to examine the joint utility of both scales.

However, subsequent analyses revealed group differences in the variables investigated in Study 1 (mean age, time post treatment/diagnosis, disease staging). First, the DTS caseness group was

significantly younger, and reported more severe depression symptoms, than the below caseness group. It is interesting to note that these differences are also reflected in the CR-PTSD literature where age and depression symptoms are regressed on symptom severity (chapter 1.5.). Similarly, the caseness group includes both AD and CR-PTSD subgroups. However, when the clinical sample was grouped by diagnosis, all these differences disappeared. This suggests that while mean age is a consistent predictor of reported distress, it does not significantly differentiate PTSD and non-PTSD groups.

Variables such as disease stage and time since diagnosis and treatment also did not significantly contribute to PTSD symptom severity or the diagnosis of CR-PTSD. This confirms findings from the meta-analysis where all moderators of prevalence were non-significant for breast cancer samples. Similarly, this sample was comprised of mainly breast cancer survivors who were generally characterised by stage II-III disease. Though a significant proportion of this sample did not have the disease stage recorded, there was no difference in PTSD symptom scores or any other subscale between disease stage groups (including the non-recorded group). This does not necessarily mean that disease stage is not a proxy for trauma severity in PTSD in cancer survivors, as there is clear evidence that samples with higher disease stages are in fact subject to much higher prevalence rates (chapter 1.5). But as breast cancer is often detected much earlier, the prognoses associated with more severe disease may not be a significant factor. While there was no difference in disease stage, there was a significant difference between cancers where breast cancer survivors reported much more clinical distress then colorectal cancer survivors. However this difference was found with vastly different sample sizes. Breast cancer survivors were much more prevalent in the sample and colorectal cancer survivors' differences may not adequately reflect the degree of distress experienced by colorectal cancer survivors.

However, emotion variables on the EPS-25 yielded the most consistent results. When searching for predictors of outcome, problems experiencing emotion at assessment were highly correlated with PTSD symptoms at the end of treatment, and is commensurate with a significant body of literature demonstrating that the client's capacity to emotionally engage with therapy predicts their outcome (Klein et al., 1969; Orlinsky & Howard, 1989). Similar results emerged between the diagnostic groups. The CR-PTSD group was characterised by significantly more severe anxiety, depression, PTSD symptoms, and difficulties with emotional processing compared to the AD/SS-PTSD group. Though this is to be expected for those with PTSD compared to those with subsyndromal difficulties, the CR-PTSD group endorsed greater use of suppression and significantly more impoverished experience of emotion compared to the AD/SS-PTSD group. In addition, the means for the CR-PTSD group were above the cut-offs for suppression and impoverished, whereas they were below for AD/SS-PTSD.

It is tempting to conclude that those who present with a more impoverished experience of emotion are more likely to have CR-PTSD, but the results also showed that these differences were no longer apparent when depression symptoms at assessment were added as a covariate. Of the five items on the impoverished subscale (EPS-25), two might reflect experiences of depressed patients (*"My emotions felt blunt/dull"*, Item 5; *"There seemed to be a big blank in my feelings"*, Item 20), whereas the remaining three items reflect externalising aspects of alexithymia (e.g. "*sometimes I got strong feelings but I was not sure if they were emotions"*, Item 25). Consequently, systematic differences in the impoverished subscale may also reflect symptoms of depression (e.g. feelings of emptiness; Criterion A1; APA, 2000) and its functional impact on the individual. It is interesting to note that PTSD itself is associated with high rates

of co-morbid major depression (Brady et al., 2000) which may interact to increase distress and dysfunction (Shalev et al., 1998), and is strongly associated with far less social support and poorer treatment response (Campbell et al., 2007). Though it is easy to accept that depression is a common response to trauma, coping responses specific to depression (such as social withdrawal), may serve to impair the emotional processing of trauma, and for this reason it may be clinically prudent to identify how these co-morbidities impact on therapeutic efficacy, and include adjuvant interventions to optimize treatment (Brady et al., 2000; Monson et al., 2004).

3.2.4.2. Strengths and Limitations

The key strengths of this study were that it addressed the limitations of Study 1, which also raised questions regarding the differential diagnosis between CR-PTSD and AD, and variables that might distinguish both diagnostic groups. This necessitated a different sampling approach that bore similarities to Mystakidou et al's (2012) protocol. Their study involved screening in participants from a larger sample of over 1000 cancer patients who attended structured clinical interviews for anxiety disorders. Of that large sample, n = 195 were screened in, of which 83% had PTSD due to a range of stressors, and 63% had PTSD due to cancer. Similarly, the sample for Study 2 was gathered from an entire NHS catchment area and patients were screened in using PTSD symptom criteria that have a good specificity (Criterion B1 & B2; APA, 2000) thus identifying those cancer survivors who were at risk of co-morbid anxiety and depression, adjustment disorder, subsyndromal PTSD, and cancer-related PTSD symptoms. This study identified those who were most likely to meet criteria for CR-PTSD, AD, or SS-PTSD related to cancer, and created a mental health sample of cancer survivors that was relatively unhampered by the PTSD prevalence issues that plagued the literature. This allowed a more direct comparison of the diagnostic groups. They then attended a full clinical interview with a consultant clinical psychologist and oncology specialist which enabled a comprehensive assessment of the individual while taking into account the relevant diagnostic issues inherent to CR-PTSD (chapter 1.5). This study therefore had sufficient statistical power to detect differences between diagnostic groups.

However, this study does have its own limitations. First, some caution is warranted on statistical grounds as only nine patients were involved in the prospective analysis, and it is uncertain if this relationship were to remain significant if the remaining sample had returned their outcome measures. Further investigation would require much larger samples to improve statistical power and increase confidence in these findings. Second, as this study is primarily cross-sectional, the presented findings can only reflect concomitant relationships between variables. Though there is no evidence here for the power of these variables to *predict* the development of CR-PTSD compared to AD, the findings do demonstrate that those who meet caseness for CR-PTSD (DTS) and for problems with experiencing, labelling, and linking emotions (Impoverished subscale, EPS-25) may be more likely to have CR-PTSD compared to those who are also above the cut-off on the DTS but below on the impoverished subscale (EPS-25).

Though the impoverished subscale provided significant predictive power, the results showed that a significant proportion of variance in PTSD symptom severity remained unexplained. It could be because other variables may contribute to PTSD symptom severity, but the sample size was not sufficiently large as to regress more than three predictors on the dependent. It is possible that if the sample was larger a

greater degree of variance would have been explained by other subscales. Therefore, these observations could not be further supported by the data. Binary logistic regression could be used to examine the joint utility of the DTS and EPS-25 subscales to correctly categorise patients to CR-PTSD and non-PTSD groups, but that would require very large samples which were not feasible for a single-centre therapy pilot. An additional limitation of this study was that there was no data collected on the perception of social support in these cancer survivors. This is important as social support is often reported to be the strongest predictor of PTSD symptom severity across trauma populations (chapters 1.2), and the most consistently predictive of PTSD symptom severity in the cancer population (chapter 1.5). Finally, though the PTSD Clinic was able to successfully treat CR-PTSD in a range of patients, many therapy completers did not return the outcome measures (DTS, EPS-25) after the final session, and thus pre-post comparisons may not reflect the true effectiveness of EPT (Baker et al., 2013). However, EPT is first and foremost a trauma-focused therapy, which, like several other therapies, has been augmented with emotion regulation skills to improve therapeutic efficacy (Berking et al., 2008; Cloitre et al., 2002; Cloitre et al., 2010; Varra & Follette, 2004). If further research reveals that emotional under-engagement is indeed a consistent factor in the chronicity of CR-PTSD, then studies piloting this intervention could be conducted to test its effectiveness.

3.2.4.3. Future Improvements

However, researchers would first need to address the factors that undermine the validity and reliability of these results before conducting further study. If the aim of the EPS-25 screening tool is to identify those suffering from mental ill-health, then it must also be able to identify those that are psychologically healthy. The validity of these results is compromised as there is no way to ascertain whether cancer survivors with PTSD or AD present with EPS scores that are significantly different from the cancer survivor population in general, or from survivors who have no presenting mental health complaints. Researchers choosing to replicate this study may decide to employ a matched control group of breast cancer survivors without presenting complaints, which would allow new norms to be generated from careful repeated sampling of the population of interest. This would allow a test of the hypothesis that the EPS-25 can distinguish between mental health and psychologically normal samples in cancer survivors. This, of course, would then provide better quality evidence for the utility of the EPS-25 and DTS in screening for low prevalence PTSD and AD the cancer survivor population.

The low prevalence of these disorders also has implications for sampling. This study sample, though sufficient to test for significant differences, was statistically underpowered in its ability to detect variance contributions from more than a couple of predictors. Larger samples are required along with an *a priori* power calculation which was not conducted for this study. Though finding a large control sample is feasible, the PTSD and AD groups may have to be sampled from several different oncology services.

This sampling process must also record those who choose to consent rather than decline participation. This study used a sample of patients that upon presentation to their oncology specialist or referring professional, knew they had the option of being referred to a research clinic and gave their consent to the use of their data. But, this suggests that an unknown proportion of patients may have opted out of that referral in favour of standard care pathways. Thus, those who attended the clinical constitute the only

available data.. Until this information is made available, it will be unclear whether the PTSD and AD groups were truly representative of the sub-populations from which they were derived.

Assuming the collection of this sample, the assessment and detection of PTSD and AD could be improved by the use of gold-standard structured interviews such as the CAPS (Blake et al., 1995) or SCID (First et al., 2002), which would remove some variance due to the assessing clinician and improve differential diagnosis in line with recent clinical advice (Kangas, 2013). Similarly, the reliability and validity of diagnosis can be established by an inter-rater reliability assessment as implemented in clinical trials. A more structured and comprehensive clinical interview would also elicit valid information on psychiatric history and perception of social support during survivorship, but validated questionnaires on social support would be a useful adjunct to these interviews. Finally, a prospective / longitudinal, rather than cross-sectional approach to assessment may provide more reliable data on the adjustment of cancer survivors. Survivors could be assessed using these measures at three, six, and 12 months post treatment, with further follow-up and 18 and 24 months, allowing for temporal precedence and thus some preliminary study of causation during the course of adjustment.

3.2.5. Conclusions

The most consistent finding in this study was the contribution of the impoverished emotional experience subscale to both PTSD symptom severity and differences between adjustment and cancer-related PTSD diagnostic groups. However, it is necessary to find follow-up explanations for the following issues: Problems experiencing emotions are implicated in CR-PTSD above and beyond sub-threshold presentations, and this is consistent with researching revealing that problems accessing emotions is implicated in emotional processing failure (chapter 1.4). As such, the impoverished subscale is designed to measure to what degree respondents have difficulty with labelling and linking emotional reactions to events. But, it is unclear how these cancer survivors develop such difficulties with experiencing their emotions before, during, or after the cancer. Study 2 shows that the variance contribution of emotion variables is significant, but small. Therefore, factors other than intrapersonal emotion regulation strategies (and the resulting experiential avoidance) are clearly implicated in the development of PTSD in this population. One particular candidate is a lack of social support, which is implicated in vulnerability to PTSD in all trauma populations including cancer (chapter 1.5). There is some evidence in military samples that social support interacts with personal resilience and the trauma to predict vulnerability (chapter 1.2.). This may also be the case in cancer survivors who have experienced a trauma during the course of their disease.

3.3. Study 3: Do emotional processing styles predict vulnerability to future stress: a prospective analysis in a sample of undergraduate students.

3.3.1. Introduction

3.3.1.1. Rationale

Studying factors that predict risk of PTSD following trauma is integral for the psychological care of trauma populations. In cancer survivors, age at diagnosis was a powerful concomitant of PTSD symptom severity and is a potential proxy for the perceived unexpectedness of the illness (Green et al., 1998), which reflect the helplessness and shock intrinsic to PTSD Criterion A2 (APA, 2000). In Study 2, removing variance due to younger age, though eliminating the significant differences in emotion regulation use, left a difference where a difficulty experiencing, linking, and labelling emotions differentiated CR-PTSD and non-PTSD cancer survivor groups. However, though this finding is clinically useful, it is a concomitant variable that demonstrates those with CR-PTSD generally have alexithymic tendencies. Whether or not this is partially responsible for the development of PTSD is not known from that data. It is equally likely that extreme anxiety triggered numbing responses that are also indicative of PTSD.

Study 3 was implemented to address the limitations from the PTSD Clinic data. In order to determine vulnerability, these factors must a) exist prior to the onset of disorder, and b) affect the initial conditions that set the disorder in motion. For this reason, a prospective study is required to assess whether emotional processing styles endorsed prior to the onset of a stressor can actually predict how individuals adjust to the demands set during the experience of the stressor. As mentioned in Chapter 2, this is unfeasible and impossible to run a prospective study with cancer patients from diagnosis through to follow-up given the protracted treatment time, and low prevalence of CR-PTSD. However, these emotion regulation and schema variables have trans-diagnostically implications (chapter 1.4), and thus their power to predict adjustment can be tested in a non-clinical sample. For this reason, Study 3 was implemented to test whether the emotional processing strategies implicated in Study 2, and beliefs regarding the experience and expression of emotions, were good predictors of future adaptation to stress, but in a sample of undergraduate students who were all due to experience and significant stressor.

3.3.1.2. Research Aims

Having established the role of emotional variables in cancer samples, the aims of Study 3 were: First, examine if emotional processing styles (EPS-25; Baker et al., 2007) could be used to predict adaptation to stress. The second aim was to examine if emotion schemas (LESS; Leahy, 2002) could be used to predict adaptation to stress.

3.3.1.3. Hypotheses

1) EPS-25 scores at Time 1 could significantly predict anxiety symptoms during a subsequent stressor at Time 2.
2) LESS scores at Time 1 could significantly predict anxiety symptoms during a subsequent stressor at Time 2.

3.3.2. Methods

3.3.2.1. Participants

The participants were undergraduate and master's level psychology students from Bournemouth University (*n*=24, 2 male, 22 female), who were recruited through an online research participation system in order to receive course credits. The mean age of the sample was 21.1 years (*SD* = 4.4 years).

3.3.2.2 Ethics

There were a number of potential ethical issues with this study. The data to be collected was personal and sensitive, and upon disclosure, could trigger feelings of distress in the participant. To address this, all data were collected from questionnaires and demographic sheets and were made completely anonymous with a code. All anonymous paper questionnaires were stored in a secure filing cabinet to which only the investigator had access. Once the anonymised data were entered into an electronic database, all paper recorded were shredded and disposed of in accordance with Bournemouth University's procedure for disposing of confidential paperwork. In terms of distress, all participants were informed prior to the study of this risk and told that if they felt distressed they could a) withdraw at any time, and that they would be signposted to appropriate psychological support. This study was approved by the Bournemouth University Ethics Committee.

3.3.2.3. Design & Procedure

Participants were recruited via an online research participation system to attend two 20-minute sessions: Time 1, at the start of the new semester when they were back from holidays, and there was no coursework or revision, and again at Time 2, a week before their final exams. At Time 1, participants came to a testing room where they were given an information sheet explaining the study, and taken through informed consent before proceeding. Participants took one copy of the form and the principle investigator took the other. No deception was used. Participants were invited to give consent to receive feedback on their results. They were asked to fill out the forms in the following order: 1) demographics including date of birth, year of course, and whether or not they have been diagnosed by a GP/mental health professional with anxiety or depression, number of episodes and dates; 2) the THQ, 3) the HADS, 4) EPS-25 and 5) the LESS-50. At Time 2, participants returned and filled out the HADS, EPS-25, and LESS-50. This study received ethical approval from Bournemouth University's Research and Development committee.

3.3.2.4. Apparatus / Materials

The Trauma History Questionnaire (THQ; Green et al., 1996): The THQ is a self-report tool that consists of 24 items measuring the occurrence of DSM-IV (APA, 1994) traumatic events along a continuum of crime-related incidents, natural or man-made disasters, and unwanted sexual experiences. The participant must indicate for each item whether or not they have experienced it, the frequency of said experience, and the age(s) of occurrence. This is purely an information-gathering tool used to aid assessment purposes.

The Hospital Anxiety & Depression Inventory (HADS; Snaith & Zigmond, 1983): The HADS is a 14-item self-report questionnaire that assesses a hospital patient's experience of psychological distress, but

without including symptoms that are also features of medical illness. The scale uses two dimensions: depression (HADS-D) and anxiety (HADS-A). Each dimension comprises seven items, yields a score out of 21 and each item is rated in severity from 0-3. Subscale scores between 0-7 are considered normal, 8-10 mild symptoms of clinical interest, and scores of 11+ are clinical caseness (Snaith, 2003). A cut-off of 8/21 yields a sensitivity of .83 and specificity of .78 for depression (HADS-D), and for anxiety (HADS-A), a sensitivity of .90, and specificity of .78 (Bjelland et al., 2002).

The Emotional Processing Scale (EPS-25; Baker et al., 2010): The EPS-25 is a 25-item self-report questionnaire that assesses emotional processing styles and deficits. Two questions ask the respondent to write their strongest most pleasant and most unpleasant emotional experience from the past week, and these responses are used to orientate the respondent to the 25-item questionnaire. This component uses a 10-point Likert-type ordinal scale, where higher scores measure higher degrees of emotional processing impairment on each subscale. Emotional processing styles are measured across five subscales collapsed into three facets of Baker's emotional processing model (Baker, 2007): control (suppression and avoidance), experience (impoverished and unprocessed), and expression of emotion (unregulated). The EPS-25 is able to discriminate between healthy controls and persons with mental health difficulties (Baker, Thomas, Thomas, Santonastaso, & Corrigan, 2013). The cut-off scores for each subscale are as follows: *total* (5), *suppression* (5.5), *avoidance* (4.5), *impoverished* (3.5), *unprocessed* (6.5), *and unregulated* (4.5). The EPS-25 has demonstrated considerable internal consistency (Cronbach's ά = .92) and convergent validity with measures of symptomatology on the Delusions Symptoms States Inventory, and alexithymia on the Toronto Alexithymia Scale (Baker et al., 2007).

The Leahy Emotional Schema Scale (LESS-50; Leahy, 2002). The LESS is 50-item self-report questionnaire that measures emotional schemas. Participants are asked to rate each item, on a 1-6 point Likert scale (1 = very untrue of me, 6 = very true of me), how reflective each statement is on what they have believed about, and how they have coped with, their emotions over the past month. These 50 items are divided into 14 schema dimensions that are described in detail by Leahy (2002) and Silberstein et al., (2012): positive/adaptive views on emotions (validation, acceptance of feelings, comprehensibility, higher values, expression, and consensus), and negative/maladaptive views on emotions (simplistic view on emotions, rationality, guilt, blame, rumination, duration, control, and numbness). Higher scores reflect more maladaptive schemas on all subscales. This scale has been validated in outpatient samples (Leahy, 2002), has been used in student samples (Riskind & Kleiman, 2012), has an acceptable to sufficient Cronbach's Alpha (Tirch, Leahy, Silberstein, & Melwani, 2012) and has demonstrated its reliability and validity (Leahy, Tirch, & Melwani, 2012).

3.3.2.5. Data Analysis

The analysis was conducted in several steps: ANOVAs for continuous dependent variables (depression and anxiety symptoms, emotional processing styles, and emotion schemas) were performed to 1) check for significant differences between study completers, and participants who, due to attrition, did not complete time 2 assessments; and 2), check for the effect group differences in trauma and psychiatric histories on current anxiety and depression symptoms. Paired-samples *t*-tests were used to assess for changes in anxiety and depression symptom severity, emotional processing styles, and emotion schemas

at time 2. Pearson's correlations were used to check the concomitant relationship between all variables at each time point, and were also used to check the predictive power of each dependent variable at time 1 to predict time 2. Given the small sample size, our chosen *A-priori* significance level was a two-tailed p=.05. This increases the risk of a type II error, but decreases the chances of a type I. Finally, the sensitivity and specificity of the EPS-25 to screen for anxiety and depression in psychology students was assessed in this sample, where sensitivity = [n_{true+} / (n_{true+} + n $_{false-}$)] and specificity = [n_{true-} / (n_{true-} + n $_{false+}$)].

3.3.3. Results

3.3.3.1. Sample Characteristics

Of the initial sample, 7/24 participants reported a psychiatric history, of which, 6/24 participants reported a GP's diagnosis of depression, and 4/24 participants reported a clinicians' diagnosis of anxiety (1 OCD, 1 GAD, 1 PTSD, 1 unspecified). 1/24 had a history of both anxiety and depression. The majority of participants (21/24) reported a trauma history, with 6/24 reporting a history of 1 or more physical/sexual assaults, 17/24 reporting 1 or more accident-related traumas (unexpected death of a loved one being the most prevalent), and 5/24 reporting crime-related traumas (excluding physical/sexual assault).

A series of independent one-way ANOVAs were conducted to assess the contribution of independent variables a) psychiatry history, and b) trauma history, to the dependent variables, severity of anxiety and depression symptoms. There was no effect of anxiety history on T1 depression [$F(1, 22) = 0.57$, $p = .46$], T1 anxiety [$F(1, 22) = 1.53$, $p = .23$], or on T2 anxiety [$F(1, 13) = 0.34$, $p = .57$], and T2 depression [$F(1, 13) = 0.01$, $p = .94$]. Similarly, there was no effect of depression history on T1 anxiety symptoms [$F(1, 22) = 3.15$, $p = .09$], but the effect on T1 depression symptoms was significant [$F(1, 22) = 7.87$, $p = .03$]. Finally, there was no effect of depression history on T2 anxiety symptoms [$F(1, 13) = 1.29$, $p = .28$], but there was an effect on T2 depression symptoms that approached significance [$F(1, 13) = 4.01$, $p = .06$].

There was no effect of trauma history on T1 anxiety [$F(1, 22) = 0.99$, $p = .33$] or T1 depression [$F(1, 22) = 2.80$, $p = .11$], or on T2 anxiety [$F(1, 13) = 0.96$, $p = .35$], or T2 depression [$F(1, 13) = 2.89$, $p = .12$] symptoms. There were no significant correlations between trauma frequency and anxiety or depression symptoms at any time point. However, the attrition rate by time 2 was 37.5%. Participants were grouped by those who dropped out (n=9), and those who completed (n=14). Time 1 measurements for all variables were then entered into between-groups one way ANOVA. Significant differences were found on the LESS-50 duration subscale [$F(1, 20) = 4.43$, $p < .05$] where drop-outs showed significantly higher scores [$M = 4.27$, $SE = .40$] than those who completed time 2 [$M = 3.23$, $SE = .27$].

3.3.3.2. Change in symptoms between Time 1 & Time 2.

Paired-samples t-tests were conducted to investigate changes in depression and anxiety symptoms, and also changes in emotional processing styles and emotion schemas. The results are depicted in Table

10. All tests were non-significant apart from the HADS-A [t (14) = -2.54, p = .02], where anxiety symptoms at time 1 [M = 8.79, SD = 3.95] increased significantly by time 2 [M = 10.4, SD = 4.48].

3.3.3.3. Concomitant Relationships (Time 1)

A series of Pearson's bivariate correlations (Table 11) were conducted to assess the strength of relationship between anxiety and depression symptoms, emotional processing styles and emotional schemas at T1. Depression and anxiety symptoms were significantly correlated [r = .46 p <.05], as were rumination and depression [r = .65, p <.01]. Though the EPS-25 control subscales were moderately correlated with anxiety, the unprocessed and unregulated subscales showed no relationship. The LESS-50 subscales, however, had much stronger correlations with anxiety symptoms, with control and rumination [r = .55, p <.01], and greater problems comprehending one's own emotions at T1 [r = .72, p <.001] demonstrating the strongest relationships. Comprehension contributed the most variance to T1 anxiety symptoms [R^2 = .51, p <.01].

Table 10: Means and SD for Time 1 (n =24) and Time 2 (n =15).

		Time 1		Time 2		Difference		
		Mean	SD	Mean	SD	*t*	*df*	*p*
HADS	Anxiety	8.79	3.95	10.4	4.48	-2.54	14	.02
	Depression	3.42	2.39	3.8	2.68	-0.62	14	.55
EPS	Total	3.97	1.29	3.90	1.83	-0.48	14	.64
	Suppression	3.80	2.58	3.19	2.16	0.07	14	.94
	Avoidance	4.01	1.40	3.93	2.14	-0.23	14	.82
	Impoverished	2.69	1.82	3.01	2.67	-0.69	14	.50
	Unprocessed	5.65	2.20	5.44	2.48	-0.17	14	.87
	Unregulated	3.47	1.42	3.96	1.58	-1.91	14	.08
LESS	Validation	3.08	1.04	2.71	1.1	0.44	12	.67
	Comprehensibility	2.68	1.42	2.79	1.59	-0.45	12	.66
	Guilt	2.55	1.18	2.52	1.52	-0.06	12	.95
	Simplistic View	3.68	0.93	3.89	1.25	-1.22	12	.25
	Higher Values	2.02	0.75	2.13	0.78	-0.68	12	.51
	Control	2.89	1.36	2.85	1.56	-0.72	12	.49
	Numbness	2.82	0.92	2.88	0.98	-0.37	12	.72
	Rationality	3.85	0.92	3.41	0.81	1.19	12	.26
	Duration	3.59	1.14	3.23	1.44	-0.31	12	.76
	Consensus	3.47	0.93	3.27	1.04	1.63	12	.13
	Acceptance	3.09	0.98	2.98	0.95	-1.18	12	.26
	Rumination	3.22	0.87	3.32	1.24	0.74	12	.94
	Expression	2.95	1.49	2.27	1.07	0.68	12	.51
	Blame	2.75	0.97	3.46	1.22	-1.61	12	.13

3.3.3.4. Risk Factors for Scores at Time 2

A series of Pearson's bivariate correlations (Table 12) were conducted to assess the ability of T1 anxiety and depression symptoms, emotional processing styles and emotional schemas, to predict symptoms at T2. Anxiety symptoms at T1 predicted anxiety symptoms at T2 [r = .85, p <.01], providing a substantial variance contribution [R^2 = .72, p <.01]. Similarly, depression symptoms at T1 predicted depression symptoms at T2 [r = .79, p <.01], and contributed substantial variance [R^2 = .62, p <.01].

However, contrary to our hypothesis, the EPS-25 subscales at T1 did not significantly correlate with anxiety and depression symptoms at T2, though Time 1 HAD scores did predict EPS scores at T2 [r = .79, p = <.01]. The LESS-50, however, provided substantial predictive power of both future emotional processing styles, and the severity of anxiety at T2. The comprehensibility scale at T1 in particular positively predicted anxiety at T2 [r = .55, p = <.05], and made a moderate variance contribution [R^2 = .30, p <.03]. This same scale also predicted substantial increases in suppression [r = .66, p <.01], impoverished experience of emotion [r = .71, p <.01], and behavioural avoidance [r = .81, p <.05].

Table 11: Pearson's correlation (2-tailed) of Time 1 measurements (*n* =24) * = <.05, ** = <.01

		HADS		EPS						LESS													
		1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22
HADS	1 Anxiety		.46*	.52**	.42*	.46*	.46*	.29	.13	.44*	.72**	.48*	.32	.25	.55**	.53*	-.00	.45*	.35	.42	.55**	.03	.33
	2 Depression			.33	.18	.27	.22	.41*	.16	.36	.40	.36	.12	.29	.34	.34	-.14	.29	.02	.11	.65**	.11	.22
EPS	3 Total				.82**	.53**	.82**	.63**	.71**	.54**	.60**	.46*	.35	.04	.43*	.48*	.21	.34	.05	.51*	.10	.01	.07
	4 Suppression					.27	.78**	.29	.33	.44*	.46*	.26	.13	.06	.33	.48*	.44*	.36	-.22	.52*	-.03	-.06	-.20
	5 Avoidance						.48*	.07	.35	.29	.63**	.50*	.15	.22	.33	.40	-.39	.08	.15	.36	.14	-.21	.12
	6 Impoverished							.25	.35	.41	.56**	.37	.28	.13	.32	.51*	.33	.12	.22	.43*	-.04	.00	-.02
	7 Unprocessed								.65**	.39	.22	.27	.42	-.02	.17	-.02	.05	.38	.16	.20	.28	.24	-.04
	8 Unregulated									.34	.29	.36	.46*	-.15	.26	.21	-.07	.19	.02	.15	.14	.05	.08
LESS	9 Validation										.67**	.67**	.28	.34	.77*	.4	.18	.41	.26	.64**	.32	.28	.28
	10 Comprehensibility											.69**	.30	.18	.64**	.56**	.13	.41	.36	.76**	.47*	.25	.15
	11 Guilt												.66**	.38	.74**	.33	.10	.46*	.27	.67**	.38	.38	.36
	12 Simplistic View													.30	.30	.01	.10	.47*	.25	.24	.36	.32	.17
	13 Higher Values														.34	.05	-.22	.48*	-.06	.06	.37	.12	.10
	14 Control															.59**	.15	.40	.16	.68**	.26	.43*	.47*
	15 Numbness																.32	.10	.17	.39	.19	.15	.37
	16 Rationality																	.15	.11	.45*	-.22	.47*	-.09
	17 Duration																		.01	.49*	.55**	.52*	-.02
	18 Consensus																			.18	.12	.28	.10
	19 Acceptance																				.10	.52*	.04
	20 Rumination																					.15	.29
	21 Expression																						-.17
	22 Blame																						

Table 12: Pearson's correlation (2-tailed) of Time 1 with Time 2 measurements (n=15). Note that T2 LESS completed by n = 12 * p = <.05, ** p = <.01

		Time 2																					
		HADS		EPS						LESS													
		1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22
Time 1																							
HADS	1 Anxiety	.85**	.54*	.74**	.61*	.63*	.65**	.50	.71**	.89**	.81**	.80**	.39	.70**	.80**	.51	.52	.63**	.70**	.75**	.55**	.47	.12
	2 Depression	.38	.79**	.43	.21	.23	.26	.59*	.53*	.40	.29	.28	.27	.63*	.45	.02	.15	.40	.50	.07	.34	.43	.35
EPS	3 Total	.24	.32	.42	.35	.35	.37	.30	.37	.43	.42	.49	-.06	.50	.40	.15	.29	.01	.55	.51	-.00	.27	.04
	4 Suppression	-.08	.08	.20	.30	.12	.27	.01	.10	.22	.28	.16	-.37	.17	.10	.17	.43	-.12	.24	.40	-.27	-.03	-.12
	5 Avoidance	.33	.38	.49	.26	.62*	.62*	.35	.09	.52	.63*	.61*	.13	.47	.52	.12	.08	.34	.55	.62*	.17	.30	-.18
	6 Impoverished	.15	.34	.24	.19	.27	.32	.05	.12	.41	.41	.32	-.11	.30	.17	-.05	.49	-.09	.43	.56*	-.21	.18	-.37
	7 Unprocessed	.41	.41	.50	.43	.25	.18	.63*	.68**	.32	.21	.45	.37	.71**	.44	.08	-.02	.14	.54	.04	.47	.36	.46
	8 Unregulated	.05	.09	.11	-.08	.19	-.09	.19	.36	.17	.07	.40	.20	.31	.34	.10	-.32	-.05	.40	.23	.17	.32	.30
LESS	9 Validation	.40	.21	.57*	.60**	.57*	.35	.56*	.23	.58*	.50	.51	.13	.62*	.51	.29	.63*	.28	.49	.36	.11	.77**	.23
	10 Comprehensibility	.55*	.42	.74**	.66**	.81**	.71**	.49	.31	.83**	.89**	.75**	.26	.55	.75**	.50	.54	.64*	.59*	.84**	.30	.54	-.09
	11 Guilt	.36	.40	.60*	.50	.78**	.30	.59*	.32	.76**	.46	.70**	.44	.50	.70**	.15	.43	.50	.62*	.73**	.14	.87**	.20
	12 Simplistic View	.41	.46	.47	.33	.60*	.10	.47	.55*	.70**	.32	.65*	.77**	.55*	.60*	.10	.18	.43	.63*	.54	.48	.78**	.16
	13 Higher Values	.33	.31	.29	.20	.36	.17	.43	-.02	.27	.24	.08	.30	.34	.01	-.12	.45	.31	.06	-.01	.13	.45	-.01
	14 Control	.47	.30	.54*	.49	.56*	.31	.51	.36	.61*	.41	.56*	.10	.33	.66*	.35	.62*	.40	.41	.57*	-.04	.74**	.41
	15 Numbness	.12	.20	.08	.04	.11	.21	-.15	.11	.36	.23	.09	-.50	.08	.25	.30	.52	.29	.23	.50	-.29	.11	-.06
	16 Rationality	-.22	-.09	-.08	.18	-.08	-.09	-.26	-.06	.15	-.06	-.15	-.31	-.18	-.12	.09	.64*	.02	-.09	.29	-.40	-.02	-.17
	17 Duration	.39	.36	.61*	.61*	.66*	.42	.50	.32	.68*	.56*	.48	.71**	.47	.46	.37	.38	.79**	.31	.48	.64*	.50	.05
	18 Consensus	.54*	.40	.45	.43	.56*	.39	.19	.27	.74**	.42	.48	.39	.54	.42	-.05	.45	.29	.70**	.56*	.29	.56*	-.38
	19 Acceptance	.28	.16	.63*	.74**	.68**	.57*	.41	.12	.72**	.73**	.65*	.10	.31	.67*	.50	.69**	.55	.40	.84**	.03	.49	-.01
	20 Rumination	.50	.41	.44	.26	.46	.26	.46	.44	.49	.51	.43	.69**	.51	.50	.41	-.01	.77**	.29	.16	.81**	.44	.13
	21 Expression	.25	.26	.34	.39	.46	.07	.34	.16	.49	.23	.23	.44	.09	.32	.11	.46	.65*	.10	.43	.13	.47	.03
	22 Blame	.08	-.40	.07	.07	.05	-.05	.10	.17	.19	-.15	.21	-.17	.17	.30	.13	.29	.02	.22	.09	-.16	.45	.50

3.3.3.5. Sensitivity and Specificity

The sensitivity and specificity of the EPS-25 to detect anxiety and depression in an undergraduate sample is depicted in Table 13. For Time 1 anxiety, 11/24 met caseness for clinically significant symptoms (*n*=24). The EPS-25 Total and Avoidance subscales each demonstrated an acceptable level of sensitivity to anxiety (64%). All subscales demonstrated moderate to excellent specificity, with the Total Subscale yielding a specificity of 100%.

Table 13: Sensitivity and specificity of EPS against the HADS

		HADS-A Caseness		HADS-D Caseness	
Time 1 (*n* =24)		Sens	Spec	Sens	Spec
EPS Caseness	Total	0.64	1	1	0.77
	Suppression	0.47	0.77	0.5	0.68
	Avoidance	0.64	0.77	0.5	0.59
	Impoverished	0.55	0.77	1	0.68
	Unprocessed	0.55	0.77	1	0.68
	Unregulated	0.18	0.92	0.5	0.95

3.3.4. Discussion

The aim of this experiment was to establish the ability of emotional processing styles and schemas to predict future responses to stress. This was done by first assessing undergraduate students at a baseline early in their semester when they had returned for the summer break and had no assignments due, and then again a week before their final exams while they were doing their revision. Exam stress was used as an ethical, and executable, alternative to a larger, less predictable, and uncontrollable, stressor. The results of this study did not confirm the first hypothesis that a person's emotional processing styles predict how they will respond to future stress. In fact, the reverse was true. Though the EPS-25 did demonstrate significant positive concomitant relationships with anxiety and depression symptoms at both time points, it failed to provide any power to predict anxiety and depression symptom severity during the exposure to exam stress. What was also interesting is that it is often expected that repeated measures using the same tool will elicit positive correlations between them. This is not well reflected in the data either. The majority of EPS-25 subscales at T1 did not significantly correlate with any other subscales at T2, apart from the avoidance subscale, which is also a characteristic of anxiety and depressive disorders. Given the strong concomitant, but non-significant predictive, relationship between emotional processing styles and anxiety/depression symptoms, it is possible that emotional processing styles endorsed when at rest (T1) do not serve as predictors of how people respond under stress (T2), but rather better reflect strategies that individuals use only when experiencing stress, and as a result, styles may change with the onset of stress. In this case, the experiencing of stress symptoms may trigger dormant vulnerabilities (McKeever & Huff, 2003) used to regulate unpleasant emotions.

Cognitive-behavioural models would suggest that behaviours people perform in response to a stressor are the result of the person's beliefs regarding the nature of the stressor, the experience of stress, and their

capacity to cope with both. In our analysis, a participants' ability to understand and interpret their emotions (LESS-50 comprehensibility subscale) at T1 was a significant and moderately powerful predictor of anxiety at T2. In addition, comprehensibility at T1 was able to strongly predict emotional processing styles at T2, and these EP styles were very highly correlated with anxiety at T2. Therefore, their understanding of their own emotions, and their own current symptoms, were better predictors of future stress. The fact that emotion schemas were strongly correlated with emotional processing styles, but differentially associated with depression and anxiety, suggested that while the emotion schemas gave insight into the beliefs surrounding the perception and imagined consequences of experiencing and expressing emotion, the emotional processing styles may better reflect strategies used to control the experience of emotion, given the unhelpful beliefs they have about experiencing them. Emotional processing styles may not serve as a long-term predictor of how people cope, but might better predict the duration of disturbance. Previous research has revealed that the greatest predictor of how clients progress in therapy rests in their initial capacity to experience and comprehend their own emotions (Gendlin, 1982; Klein et al., 1969; Leahy, 2007). This was verified in this study, with comprehensibility serving as a concomitant and predictor of future anxiety, and the strategies used to control it.

It is also interesting to note that variable sensitivity and specificity statistics for the EPS-25's ability to effectively screen for depression and anxiety. For anxiety in particular, the EPS-25 was less effective at correctly identifying clinically significant symptoms, but was superior at correctly screening out those who did not endorse clinically significant symptoms. Though the sensitivity of the EPS was poor in this sample, this may merely reflect that the EPS-25 is a trans-diagnostic tool for detecting the difference between normal and mental health populations. As such, the high specificity of the EPS-25 in this sample may reflect its ability to identify emotionally healthy individuals. This reflects the data from study 2, where the EPS-25 provided superior specificity to PTSD to screen out non PTSD patients. If this is true, it may serve as a useful adjunctive measure to questionnaires for individual anxiety disorders, including PTSD. However, the statistics for depression at T1, though useful, may not accurately reflect the utility of the EPS-25 in this population, due to 2/24 meeting caseness for clinically-significant symptoms. This may greatly affect the sensitivity and specificity of the EPS-25 against HADS-D.

3.3.4.1. Strengths

One strength of this study was that potential risk factors that were theoretically-driven and measured before exposure, and exposure was measured before the onset of disorder. In this case, it allowed the observation and assessment of multiple outcomes (depression, anxiety) from the exposure to a single event, and the predictive value of temporally-precedent variables in their ability to predict future adaptation. Temporal precedence can demonstrate the direction of causality whereas cross-sectional studies such as Study 2 cannot.

3.3.4.2. Limitations

Given the nature of this study, it is wise to consider the limitations of prospective methods, and their application in this particular study that may constrain one's conclusions. According to Hennekens &

Buring, (1987), there are several factors to consider before using prospective methods. First, appropriate group selection is central, as all participants must first be able to be exposed to the event, and must be able to develop the outcome to be investigated. This study employed a sample of students, all of whom were eventually exposed to the same stressor (final exams), and were also all capable of developing the outcome (signs and symptoms of increased stress). Though these criteria were met, there were problems with using this population in the sense that it was a non-clinical that was used to provide clinical insight into the role of emotion regulation variables on the experience of stress. As this sample was not comparable to the cancer population which is the centre of this thesis, no direct inferences could be made between these findings and the emergence of PTSD in cancer survivors. However, despite this not being a clinical sample, the prevalence of criterion A traumas (APA, 1994) was exceedingly high, and the endorsement of a psychiatric diagnosis constituted approximately 35% of the sample. In this sense, the results may be clinically-relevant and thus moderately comparable to mental-health populations in general. It must also be noted that the fact that these findings were detected in a small sample, using a very mild stressor, at a high alpha level ($p<.01$), suggested that the relationship may well be more pronounced in a larger, more diverse clinical sample. Additional replications of this research will be necessary to establish the clinical utility of using beliefs about emotions as a predictor of subsequent adaptation to stress.

Second, once these groups have been exposed, investigators have to consider how exposure is measured by identifying whether it is categorical (e.g. presence or absence of injury/disease/vulnerability), ordinal (number of exposures), or interval (e.g. duration of exposure). Also, the investigator must consider whether the true exposure is captured in the measurements. A lack of control over exposure may lead to miscategorisation of participants, or a false reading of the severity of the stressor to which one is exposed, and if there were additional factors per exposure that contributed to outcome. In this case, there was a significant limitation in the conceptualisation and operationalization of the exam stressor. The means for T1 and T2 anxiety, while significantly different, only moved from the mild to moderate range on the HADS-A subscale. This may have been insufficiently stressful to activate maladaptive processing styles. Also, whatever differences in anxiety were due to the stressor may be compounded by stressful or traumatic situations that occurred within a week of either of the two time points. The EPS-25 was designed to document these stressors, but the relative contribution of uncontrolled experiences during each time-point may have reduced the sensitivity of the stressor. Nevertheless, the aim of this study was not to assess the degree to which the experience is distressing (as in Study 1), but rather to ascertain what emotion variables (schemas and strategies) best predict future adjustment to stress. The results obtained, though from a small and non-generalizable sample, were highly consistent with clinical observations and emerging interventions that rest on the premise that beliefs about the comprehensibility and uncontrollability of emotion, and the strategies used to control them, actually predict not only the duration of distress, but also the degree to which it is experienced when faced with a significant stressor (chapter 1.4).

Third, experimenters must consider how outcome is measured and use the same measures for each group. In this study, the outcome (severity of stress-related symptoms) was measured using a highly validated questionnaire that also provides reliable cut-offs for clinically significant distress at different

levels of severity. This measure was able to detect small but significant increases in depression in anxiety symptoms alongside a stressful event in this small sample.

Fourth, the biggest concern is one of attrition. Prospective studies in clinical populations can suffer from high attrition rates, which may be attributable to systematic differences between drop-outs and completers. For example, those who are at the greatest risk of developing the outcome (e.g. clinically significant anxiety) may be at the greatest risk of attrition. In this study there was a systematic difference between drop-outs and completers in that the attrition group had significantly greater beliefs about the indefinite duration of unpleasant emotion. Though T1 duration was not a significant predictor of anxiety and depression symptoms at T2, it did strongly predict avoidance behaviour at T2 in the remaining sample. It is possible that this fear of extended duration motivated the drop-out group to avoid a second assessment. This clearly has introduced some bias into the results, but also suggests that had the drop-out group actually completed the study, the observed relationship between negative emotional schemas and future anxiety symptoms would be better established. The attrition rate also has methodological implications. The power calculations used suggest that assuming a good statistical power (β=.80), significance level, (α=.05), and anticipated medium effect size, (F^2=1.22), then the minimum required sample size would be n=37. Given the attrition rate, the remaining sample was very small (n=15) and thus could have instigated a Type II error, meaning that some clinically-relevant relationships were not observed.

3.3.4.3. Future Improvements

This research question, and the methodology used, can be altered to improve the reliability and validity of the results. In line with the first limitation (group selection), there may be an underlying assumption that the student population is homogenous, but the data suggest a range of ages and life experience, coupled with a diverse endorsement of criterion A events. Similarly, this is not a clinical population where adverse reactions to life stressors would be expected. This leads to two action points: sub-populations need to be accounted for in the analysis, and study design needs to account for the detection of small effect sizes using clinical measures. Future replications might also address how 'exposure' is measured. In the case of this study, exam stressors were categorical, discrete, and known. That is, participants either had them or did not, and the sample all experienced them at the same time. However, the degree of exposure was not measured, neither were additional variables that could have contributed to their adjustment to this academic demand – such as intervening events (relationship problems), frequency and proximity of exams, social support, or the role of actual or perceived intellectual ability on the level of stress experienced. Subsequent researchers might consider collecting data on these variables using validated questionnaires or simple demographics data, and performing a regression analysis.

The adjustments suggested above will likely improve the quality of the study should it be replicated. However, this will require very large samples to detect smaller, but nevertheless significant, variance contributions of multiple predictors. This may be feasible with a student population if data were collected over several academic years. Assuming a large enough sample, future replications may include either a matched control group (or a third assessment in the current prospective approach) that may not experience a predictable stressor (exams) to ascertain if the symptom changes detected in the exam group were

indeed caused by the exam stress. This, along with a large sample, may permit a mediation analysis (Baron & Kenny, 1986) to ascertain the role and contribution of emotion schemas and emotional processing styles in the onset and maintenance to the psychological adjustment of stressful events.

3.3.5. Conclusions

Overall, there was a statistically significant increase in anxiety symptoms by T2. In additional to T1 anxiety symptoms, comprehensibility of emotions (LESS-50) was the best T1 predictor of T2 symptoms, and was significantly sensitive at such low levels of stress. This suggests that the beliefs that individuals generally hold regarding the experience and expression of emotions in a social context may be implicated in adjustment to a stressor but also in the chronicity of psychological disorder, should it develop.

3.4. Summary of Conclusions for Studies 1-3

Cancer-related PTSD is uncommon in cancer survivors. Nonetheless, it still develops in a select minority of the population. Though several factors appear to be implicated in the development of CR-PTSD, most of these variables have been regressed on the severity for PTSD symptoms, which though impairing, do not constitute a PTSD diagnosis. Study 1, however, revealed that only mean age and time post treatment were mildly implicated in the prevalence of diagnosable CR-PTSD. Chapter 1.4. suggested that emotion variables (e.g. emotion regulation strategies, and symptoms related to emotional numbing and alexithymia) might be more strongly implicated in the development and maintenance of PTSD, and also in cancer survivors (chapter 1.5). Study 2 revealed that once the significant variables from Study 1 (sample age) were controlled for, all emotion regulation strategies were rendered non-significant, with only impoverished emotional experience differentiating CR-PTSD and non-PTSD groups. One interesting finding was that controlling for concomitant depression symptoms at assessment rendered this finding non-significant. It is possible that both scales measured overlapping constructs, and that problems experiencing, linking, and labelling emotions might also accompany a severe depressive episode. If this is so, the very presence of depression may trigger withdrawal and affect emotional processing.

4.1. Study 4: Assessment and treatment of subsyndromal and chronic CR-PTSD: clinical case studies of two breast cancer survivors during an experimental trauma-focused therapy pilot.

4.1.1. Introduction

4.1.1.1. Follow-Up Explanations / Qualitative Research Questions

The results from the quantitative phase (Chapter 3) demonstrated that demographic and disease variables were not significant predictors of CR-PTSD (Studies 1 & 2), but that variables relating to the

processing of emotion were implicated in the severity of PTSD symptoms (Study 2). More specifically, patients with CR-PTSD suffered from clinically significant problems experiencing, linking, and labelling, emotion compared to the subsyndromal/adjustment disorder group. Similarly, regression analyses suggested that while emotion regulation variables such as suppression, rumination, and alexithymic symptoms positively predicted PTSD symptoms, a large degree of variance remained unexplained (Study 2). This suggests that though these variables are significantly implicated in CR-PTSD development, most of the variance is attributable elsewhere. As such, it is still unclear how these variables affect the process of adjustment in this sample of cancer survivors. Chapter 1.4. revealed that social support was significantly implicated in PTSD symptom severity, but Phase 1 was limited as it did not measure this variable. It is therefore unknown in this sample how social support during and after the disease is implicated in this process of adjustment, and if (or how) it interacts with emotional processing factors. However, chapter 1.4. suggested that emotion schemas are socially constructed, so what individuals believe about the experience and expression of emotions is imbedded in their social system, suggesting a link between emotion variables and social support. For this reason, two breast cancer survivors were selected for clinical case studies at the PTSD Clinic (one with CR-PTSD, and one without), to enable follow-up explanations for the role of 1) emotion schemas, 2) emotional processing styles, and 3) social support, in the course of psychological adjustment during and after the cancer, how these phenomena integrate into the clinical presentation of the individual (e.g. CR-PTSD v no PTSD). Furthermore, information on their experience of traumatisation was gathered.

4.1.2. Methods

4.1.2.1. Participants

The patients in this study were part of an on-going referral pathway to the PTSD clinic at Royal Bournemouth Hospital. Patients were referred by an oncology specialist doctor or nurse due to the patient reporting unremitting psychological difficulties, namely PTSD intrusion symptoms. The inclusion criteria for participants in this study were that they would be cancer survivors who have finished treatment for cancer, and that they will be disease-free, and will have survived for at least six months with the expectation of continued survivorship. Exclusion criteria included those patients suffering from, or a history of, psychotic illness, or those who at the time of assessment and treatment were not stabilised, so were at risk from others or a serious risk to self (e.g. actively suicidal), or if they refused written consent to participate.

4.1.2.2. Ethics

There were a range of ethical issues that were covered. First, as patients were opting into research as well as NICE-recommended trauma-focused treatment, there had to be no conflict of interest where patient believe that had to consent to research participation in order to be treated. This was resolved by creating two equal tracks that did not differ on wait time or priority: 1) research track, where, after consent, they had their first contact with the investigator; and 2) the normal therapy track, where the clinic

runs as normal with no researcher present. Second, this study required the collection of sensitive audio transcripts and qualitative interviews that are highly identifiable. All mp3 audio files and audio transcripts were stored on NHS-approved and highly encrypted solid-state drives. All assessment forms and symptom measured were made anonymous using a code and no identifiable information was recorded. This study was approved by the NRES Committee London – Dulwich (REC Reference 12/LO/0236) and Royal Bournemouth Hospital R&D.

4.1.2.3. Case Selection

A range of criteria were used to include patients into Phase 2. First, they had to meet the general inclusion criteria for the PTSD Clinic. Second, the initial screening and subsequent clinical interview had to suggest the presence of a psychological disturbance due to the experience of cancer. In particular, this phase was focused on exploring differences between a) patients with CR-PTSD, and b) patients with adjustment disorder or subsyndromal PTSD symptoms.

4.1.2.4. Clinicians and Investigators

Initial screening and assessment of eligibility was conducted by the experimenter (GA) using the Patient Overview and PTSD module of the Structured Clinical Interview (SCID; First et al., 2002). Final clinical decisions were made via a blind assessment by the consultant clinical psychologist Prof. Roger Baker (RB) and Consultant Nurse Oncologist Lin Purandare (LP), who also provided the Emotional Processing Therapy.

4.1.2.5. Procedure

Referral: Cancer survivors, who, having finished treatment for either breast or colorectal cancer, and were in remission, presented to their oncology specialist or GP, reporting unremitting psychological difficulties, namely PTSD intrusion symptoms such as nightmares and flashbacks. The oncology specialist doctor or nurse, or referring clinician, referred the patient to the PTSD clinic at Royal Bournemouth Hospital for assessment. The oncology specialist (LP) took the referrals. Upon referral, clinical history was checked for a history of psychotic illness.

Invitation by Letter, Study Education, and Informed Consent: Patients referred over by their clinician were sent a letter covering several points. First, explaining that the PTSD Clinic was undertaking research, provide a patient information sheet about the study, and invited the participant to give informed consent to an eligibility screening conducted by the investigator (GA) prior to entry into the study. If the patient gave consent, they were asked to sign and date three copies of the consent form. One form was given to the participant, one kept in medical notes, and the other in the research file. Upon the patient's admission into the study, the assessing clinician kept the consent forms in safe storage on the NHS site, and allocated the patient a confidential ID code which will then be passed to the investigator. An exclusion criterion for the study at this stage was if the patient refused written consent to participate. If the patient did not give informed consent to participate in the eligibility screening (as conducted by GA) and hence entry into the study, then they were still offered a clinical assessment with RB and LP

consistent with the PTSD Clinic's current practice, and any psychometric data collected by RB & LP was not included in the study. Additionally, the letter also contained the time, date, and place of the appointment, and also a questionnaire for the patient to fill in (THQ) just before the appointment. The patient was informed in the letter that the assessment will be audio-recorded for transcription and note-taking purposes.

Eligibility Screening: Given informed consent to the screening, the investigator (GA) administered the rest of the questionnaire package and the SCID – I / NP Overview and PTSD module. Study exclusion criteria at this stage were those patients suffering from psychotic illness, or those who are at the time of assessment and treatment are not stabilised, so are at risk from others, to others, or are a serious risk to self (e.g. actively suicidal). If these exclusion criteria were met, the assessing clinician made a referral to the correct Community Mental Health Team (if appropriate) and notified the patient's GP.

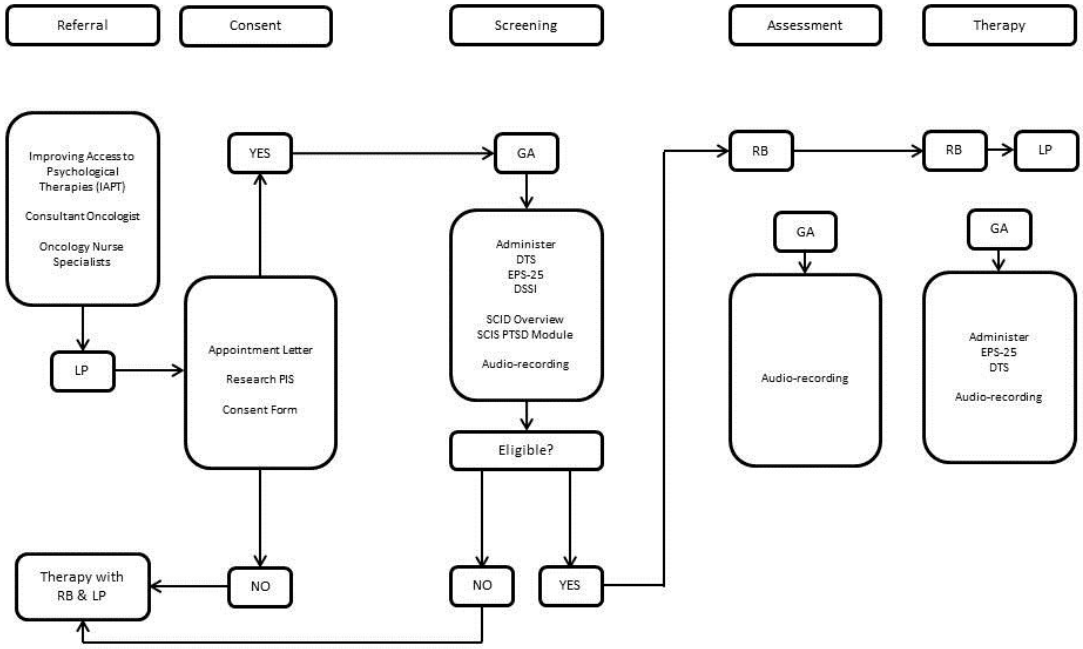
Assessment: When the eligibility screening was over, the patient was then seen straight after by the consultant clinical psychologist (RB). Consenting patients were assessed in a clinical interview by RB. When the assessment was over, the investigator (GA) then presented a written report of the screening and the questionnaire scores to the clinical psychologist (RB) and oncology specialist (LP), who made a final clinical decision on the following criteria: 1) Does the patient meet caseness on the DTS? 2) Does the patient meet full (or partial) PTSD criteria? And 3) Is cancer-related PTSD the primary disorder? If all three criteria were met, the patient was entered into the study. If these criteria were not met, they were moved into the non-research pathway.

Therapy: All patients in both the research and non-research pathways were treated using emotional processing therapy. Therapy was conducted by the clinicians (RB and LP) at the PTSD Clinic in Royal Bournemouth Hospital, in 50-minute sessions. GA administered outcome measures (see section 2.5) to the participant for 10 minutes prior to each therapy session. Screening, assessment, and therapy sessions were audio recorded and transcribed for analysis by the investigator (GA). The procedure pathway is presented in Figure 8.

4.1.2.6. Measures

The *Davidson Trauma Scale (DTS; Davidson et al., 1997):* The DTS is a 17-item self-report questionnaire that assesses the frequency and severity of PTSD symptoms by total or by symptom cluster according to DSM-IV diagnostic criteria for PTSD. Higher scores index greater frequency and/or severity of the PTSD symptom. There are three subscales - measuring intrusions (Criterion B), avoidance/numbing (Criterion C), and hyper arousal (Criterion D). The DTS is administered to assess the patient's response to individual traumatic events. The patient is orientated to this by a question asking the patient to identify their most disturbing trauma (DSM-IV PTSD Criterion A1). For this study, the PTSD syndrome was anchored to the experience of cancer diagnosis and treatment. The DTS cut-off score can be varied depending on the assumed prevalence of PTSD in the trauma population of interest. Recent meta-analyses revealed a current cancer-related PTSD prevalence of 4% (Abbey et al., 2014), so the DTS cut-off used for this study is 47. The DTS has good internal consistency, good concurrent and construct validity (Zlotnick et al., 1996), and good convergent and divergent validity (Davidson et al., 1997).

Figure 8: The procedure for SEM Phase 2



The *Emotional Processing Scale (EPS-25; Baker et al., 2010):* The EPS-25 is a 25-item self-report questionnaire that assesses emotional processing styles and deficits. Two questions ask the respondent to write their strongest most pleasant and most unpleasant emotional experience from the past week, and these responses are used to orientate the respondent to the 25-item questionnaire. This component uses a 10-point Likert-type ordinal scale, where higher scores measure higher degrees of emotional processing impairment on each subscale, and on the total score. Emotional processing styles are measured across five subscales collapsed into three facets of Baker's emotional processing model (Baker, 2007): *control* (suppression and avoidance), *experience* (impoverished and unprocessed), and *expression* of emotion (unregulated). The EPS-25 is able to discriminate between healthy controls and persons with mental health difficulties. The cut-off scores for each subscale are as follows: *total* [5], *suppression* [5.5], *avoidance* [4.5], *impoverished* [3.5], *unprocessed* [6.5], *and unregulated* [4.5] (Baker, Thomas, Thomas, Santonastaso, & Corrigan, 2013). The EPS-25 has demonstrated considerable internal consistency (Cronbach's ά = .92) and convergent validity with measures of symptomatology on the Delusions Symptoms States Inventory, and alexithymia on the Toronto Alexithymia Scale (Baker et al., 2007).

The *Delusions Symptoms States Inventory (DSSI; Bedford & Foulds, 1978)*: The DSSI is a seven subscale self-report questionnaire that measures the severity and frequency of mood states and psychiatric symptoms from the preceding two-three weeks. Whereas the DSSI contains a hierarchical array of subscales, this study uses the neurotic subscales (somatisation, obsession, rumination, phobia, and dissociation), and the mood subscales (depression, and anxiety), but exclude the last two levels (integrated, and disintegrated delusions). This leaves the DSSI with 49 remaining items. The DSSI has demonstrated validity and sensitivity to therapeutic change (Baker et al., 1998).

4.1.2.7. Diagnostic Assessment

The Trauma History Questionnaire (THQ; Green et al., 1996): The THQ is a self-report tool that consists of 24 items measuring the occurrence of DSM-IV (APA, 1994) traumatic events along a continuum of crime-related incidents, natural or man-made disasters, and unwanted sexual experiences. The participant must indicate for each item whether or not they have experienced it, the frequency of said experience, and the age(s) of occurrence. This is purely an information-gathering tool used to aid assessment purposes.

The *Structured Clinical Interview for DSM-IV Axis I Disorders Research Version, Non-Patient Edition (SCID-I/NP; First et al., 2002):* The SCID-I/NP is a clinician-administered diagnostic interview for the Axis I disorders of the DSM-IV. It has several modules ranging from general clinical history, psychotic episodes, mood episodes, bipolar screenings, and anxiety disorders. This study used the Patient Overview to collect a patient history, and the PTSD section from Module F: Anxiety Disorders, to screen for current, and lifetime, PTSD.

4.1.2.8. Data Collection & Abstraction

As is appropriate with clinical case studies, data was compiled from multiple sources (Holloway, 2008). In terms of investigating eligibility criteria for both the clinic and research, medical variables (e.g. disease stage, treatment regimens, age at diagnosis, time since diagnosis and treatment) were abstracted from both the patient during the initial screening (Part I – Patient Overview) and also corroborated from referral letters and medical history. Psychological history was also obtained from the Patient Overview. Data on the severity of PTSD symptoms was gathered by the investigator through the use of validated psychometrics for each session. All screenings, assessments, and therapy sessions were recorded. The screenings and assessment were then transcribed by the investigator. As the data was audio-recorded, direct quotes (in context) were taken from the sessions and incorporated directly into the case reports. All direct quotes are in italics. Clauses enclosed in brackets and italicised were added by the investigator to change tense or object e.g. *"I"*, to *"[she]"*. Clauses that are in brackets but not italicised were added by investigator to refer to the context in which the quote was given, but not stated by the patient.

4.1.2.9. Qualitative Analysis

As is consistent with this *sequential explanatory design* (SED), the case studies were conducted to search for follow-up explanations for the role of emotion schemas and regulation strategies in CR-PTSD that were established in Phase 1. The data to be abstracted was organised according to a) the PTSD framework established in theory, practice, assessment and therapy; and b) the data gathered in Phase 1. The previous phase identified that generally speaking, demographic and medical variables were not significant moderators of PTSD prevalence in breast cancer survivors (Study 1), but that in a mental health population of breast cancer survivors, cognitive avoidance strategies such as rumination, and alexithymic symptoms, were independent predictors of PTSD symptom severity (Study 2), and differentiated CR-PTSD and no PTSD groups. There was some *preliminary* evidence for the predictive power of beliefs about the experience and expression on future adaptation to stress (Study 3). For these

reasons, the investigator carefully searched the therapy data for patient's beliefs about the experience and expression of emotion, and the strategies used to control emotion. Quotes from the patient were then abstracted and categorised into the following groups: 1) the cancer experience as a trauma (PTSD Criterion A; APA, 1994); 2) coping strategies, which includes those that are behaviourally or emotionally avoidant (PTSD Criterion C; APA, 1994); 3) social support; and 4), clinical findings. All of these were embedded into a clinical narrative of how they emerged during the therapy.

4.1.3. Case Reports

This section presents the two selected clinical cases called Esther and Angela. Table 15 presents their diagnosis and related psychometric data at assessment (T1) and final follow-up (T2).

Table 15: Psychometric Scores at Assessment (T1) and Follow-Up (T2)

Measure		Angela (CR-PTSD)*		Esther (CR-AD)*	
		T1	*T2*	*T1*	*T2*
DTS	Total	131	98	84	44
	Intrusion	40	28	26	7
	Avoid	54	40	29	14
	Hyper	37	30	29	23
EPS-25	Total	8.2	5.8	6.5	4.2
	Suppression	8.8	6.4	6.8	4
	Avoidance	7.8	6	6.8	3.4
	Impoverished	8.2	6	3.8	3.2
	Unprocessed	7.4	6.2	8.4	5.6
	Unregulated	8.6	4.2	6.6	5
DSSI (Severity)	Anxiety	16		5	
	Somatic	2		2	
	Obsessional	5		0	
	Depressive	10		3	
	Phobic	2		1	
	Ruminative	10		2	
	Dissociative	2		3	

Key* CR-PTSD - Cancer-related PTSD
CR-AD - Cancer-related Adjustment Disorder

4.1.3.1. Case Study: Esther

4.1.3.1.1. Patient History (SCID Overview)

4.1.3.1.1.1. Background

Esther (name and all identifiable personal details changed) was a 54 year-old divorced woman who, at the time of her initial assessment, lived with her partner. She had no children. Esther completed A-Levels in Geography, a Diploma in Aromatherapy, and used to work as an artist before physical health problems necessitated her retirement. Much of Esther's time, during her attendance at the PTSD Clinic was spent looking after her severely ill stepfather with whom she had a painful relationship. Esther loved to spend her free time engaging in her favourite hobby, playing bowls with her good friends in a women's cancer survivor group. Esther gave informed written consent for her screening, assessment, and subsequent therapy to be recorded, and transcribed, for use in an unpublished case-report. However, according to CARE Guidelines (Gangnier et al., 2013), Esther has not given consent for these reports to be published as she has not read them and approved of their content.

4.1.3.1.1.2. Vulnerability Factors

Esther's grandfather (with whom she was very close) committed suicide when she was twelve. Esther's parents had a destructive relationship characterised by frequent rows, and ended in a protracted and painful divorce when she was 16. The terrible sense of loss emotionally devastated her father and herself ever since. Esther finally got married at age 19, but this ended in divorce in 1991. Esther started seeing a man for three years after she separated from her husband. This new partner demonstrated abusive personality traits, including extreme anger and physical abuse. She eventually left him, got her own flat, and threw herself into her work, and met her current partner.

4.1.3.1.1.3. Psychiatric / Medical History

Esther reported suffering from one severe clinical depressive episode following her marital breakdown in 1991, which was related to her feeling like *'a terrible failure'* for having a divorce. This episode lasted for two years, and was treated with antidepressants and sleeping pills for the associated insomnia. During this period she checked herself in as an inpatient for a month and received electro-convulsive therapy (ECT). This was the most upsetting period of her life. Esther reported having had two depressive episodes of much milder severity since then. In addition to this, she had a serious road traffic accident in 1993, which resulted in whiplash and fibromyalgia. Finally, she suffered an umbilical hernia in 2004 and a torn Achilles tendon in 2006. The resulting pain from the injuries meant she has not worked since. Finally, Esther was also diagnosed with Stage II breast cancer in August 2011, and had a mastectomy in September 2011, and is currently cancer-free. Many of her old injuries were exacerbated during this time.

4.1.3.1.1.4. Precipitating Events

One month prior to the initial screening, Esther reported poor sleep, irritability, and some anxiety following seeing her family at Christmas. Her brother-in-law (sister's husband) had chemotherapy at the same time. Watching his slow physical and psychological deterioration was a horrific experience, especially in the context of her survivorship. He died around the time of assessment, triggering several reminders and in particular some survivor guilt.

4.1.3.1.1.5. Presenting Symptoms

Esther's presenting complaints were of poor sleep, irritability, and trembling, which were indicative of chronic anxiety. It became apparent during the assessment that the source of this anxiety was constant rumination and worry about the effects of her cancer experience on her family. Though this was distressing for her, Esther refused to let her feelings overwhelm her and sought to *'get on with life'* by continuing to see friends, and engage in her favourite hobbies.

4.1.3.1.1.6. Risk / Substance Use

Given Esther's history of hospitalisation for severe major depression, it was prudent to enquire about the use of drugs or alcohol to regulate the anxiety she was feeling. There was no indication of substance abuse, or of any thoughts, or subsequent action, towards self-harm or suicide.

4.1.3.1.1.7. Medication

Esther was prescribed Omaprazole for gastric reflux and Amytriptaline to improve her sleep patterns.

4.1.3.1.2. PTSD Diagnostic Assessment (SCID PTSD Module)

4.1.3.1.2.1. Criterion A

Esther reports several traumatic events such as her grandfather's suicide when she was 12, her road traffic accident after which she suffered whiplash and fibromyalgia in 1993. All were reported as equally upsetting. Her experience of cancer in general was not reported as a traumatic event, although witnessing the deterioration and death of her brother-in-law due to his own metastatic cancer, was endorsed. Her chemo was not endorsed as traumatic, although the loss of her hair was endorsed as traumatic. It was unclear from the screening if Esther met this criterion at the time of any of these events, although there was evidence of helplessness and horror, but not necessarily anchored to any particular A1 event.

4.1.3.1.2.2. Criterion B

There was some evidence for persistent recollections of traumatic events (including the cancer experience), but these appeared to be equally reminiscent of ruminative thoughts rather than sensory flashbacks. No nightmares were reported. There was little additional evidence of severe physiological reactivity or feeling/acting like the trauma was re-occurring. Criterion B was not met; therefore PTSD was not diagnosed according to the SCID Diagnostic Tree, and Esther was screened out.

4.1.3.1.2.3. Criterion E

The course of the PTSD symptoms was unclear due to the endorsed symptoms being anchored in additional life stressors.

4.1.3.1.2.4. Criterion F, Psychometric Scores, and Final Diagnosis

Esther scored above the cut-off (47) on the Davidson's Trauma Scale, with a total of 84. This shows that she was rather distressed, and may possibly have PTSD. She also was above the cut-off on all the EPS-25 subscales, with a mean score of 6.5 (range, 5.6 – 7.4), which indicated significant difficulties in emotionally processing distress. Esther particularly endorsed strategies of suppression and avoidance, and problems experiencing emotions, but scored very low on symptom severity (DSSI) for anxiety, depression, and ruminative states. The clinical interview (as conducted by RB), also did not suggest PTSD, but rather a protracted adjustment response. The DTS Total score of 84 was above the suggested cut-off for this population, but the assessment suggested that much of the distress (as seen in Esther's endorsement of Criterion B symptoms) was related to social stressors such as her relationship with her father rather than the experience of cancer, or any other traumatic event. Additionally, the symptoms of sleeplessness were equally attributable to fibromyalgia and the intrusive recollections due to rumination over current circumstances, rather than sensory flashbacks. No diagnosis of PTSD was made, in favour of subsyndromal PTSD as the primary disorder.

4.1.3.1.3. Therapeutic Intervention

4.1.3.1.3.1. The Traumas

Though Esther did not appraise her cancer experience as traumatic (but rather an *'ordeal'*), there were several experiences throughout the diagnosis and treatment that were deeply distressing, and were worked through during the exposure sessions. This section documents these experiences.

Mammogram: Esther described having a routine check-up for her breasts (which had uncomfortable cysts) and how the nurse told her that she had found a mass. The nurse told her to feel for herself and said that *"'it felt like granulated sugar'...then I got it."* This was a surprise for Esther, who would *"never had found this"* if it wasn't for the nurse. Samples of breast tissue were then taken, and according to Esther, were really painful, which was quite unexpected. Though she was yet to be diagnosed, Esther picked up from the experienced nurses' expression that she has cancer. Esther was in shock as she drove home and was afraid she would crash the car. She told her partner that she thought she had breast cancer. She recalled that at that point she *"felt weird and different – I supressed all feeling"*.

The First Chemotherapy Cycle: Esther recounted how she first had to read and sign a large amount of paperwork. This involved first delineating the range of possible side-effects (such as damaging the heart and lungs), and second, consenting to the treatment. This was a deeply *"overwhelming"* for her. Esther *"...knew that chemo has horrendous side-effects"*, and that her close friend Jackie refused chemotherapy, and the cancer became metastatic, and she died. Esther was told that having extra chemotherapy would reduce the likelihood of recurrence from 50% to 8%. She stated that she was expecting that they would

say *"because you are quite young for this type of cancer, we do consider that you need to go and have chemo",* but instead, Esther recounts that she was *"angry they told me it was my choice …Why leave the decision to me? They are the experts!"* It appeared that Esther believed she had little choice but to sign the documents. If she refused to sign, her cancer would spread, but if she did sign, she would consent to treatments that could lead to equally serious medical problems. She was deeply angered by the *"insensitivity"* of the process, but knew she *"had to do this"*. In therapy, Esther stated *"that really frightened me* [starts sobbing]*...[but] I had to go through this paperwork and keep signing…if I wanted to continue [with treatment] – of course I didn't want to continue!"*

Having first been aware of the severe side-effects of chemotherapy, Esther described her first treatment as terrifying. She stated, *"[I felt] terrible fear* [starts crying/trembling]. *I had a heart scan. Had to have lung check. I saw my heart in my mind - my healthy heart - and saw that I could ruin that with this treatment. Is my heart gonna be weakened? Nobody told me chemo could affect lungs and breathing!"* By the time the drugs arrived, Esther *"absolutely quailed"*. They were *"...red - with labels saying '**cytotoxin'** in black bold letters on a red bag. I thought my legs were gonna give way. [I thought] 'oh dear God, what am I doing? Am I doing the right thing?'...*[begins trembling and crying]*….I felt real terror."* Finding a vein in which to administer the chemotherapy was, according to Esther *"a horrible experience"*. An inexperienced nurse made it very painful, and damaged a vein, which Esther described as *"blowing up"*. At the time, she was afraid she *"...might be bleeding inside….[that] something was going up to [the] heart…I was gripped by fear".* A more experienced nurse came in and found a viable vein. As the drugs were administered, Esther watched it (*"the red chemo…that makes your hair drop"*) as it went in. She recounted that *"It was just terrifying…putting poison into your body* [starts to tremble and cry]. *This could permanently damage parts of your body should you have a reaction."*

Hair Falling Out: Esther's main experience of these side-effects came when her hair began to fall out. After the first chemotherapy cycle, Esther described how she was in hospital and *"there was a man who had a reaction. They had curtains around him – oxygen tanks…I never knew that complications could be developed. The first one [chemo cycle] was bad – the feeling of being fragile, having to accept things that hurt but you don't understand. That fear. I felt like a child".* When Esther's hair finally started to come out, Esther *"shrieked with horror"*. She described how she felt at the time, *"I found the hair loss more traumatic than being told I have cancer. It was one of my most frightening moments…I put my hairbrush through it and half of it came away…I developed IBS, lost my taste, and had ulcers…[it was] torture."*

4.1.3.1.3.2. Coping Strategies

Esther's earliest descriptions of how she coped during and after the cancer treatment were of a *"blocking off"* of emotions. Though at first this appeared to be a suppression tactic to avoid experiencing unpleasant emotions altogether, later sessions revealed the opposite. Esther knew that her prognosis *"…was supposed to be good"*, but she also knew she would be going through a *"terrifying ordeal"* to enter into that survivorship. For this reason, Esther began to prepare herself to manage the aspects of her life (other than cancer) over which she still had some control, *"I began to feel that it will do [me] good to feel that [I] had some control – not over the cancer, but how [I was] dealing with it I suppose, to a certain degree. I recall feeling that".* In order to do that, Esther thought it imperative to gain a balanced

perspective on her experience in order to help her cope emotionally with the cancer and treatment, *"I was aware of the fact that I didn't feel it was worthwhile to indulge in my feelings. I had enough to contend with* [the treatment and horrible side-effects] *without letting myself wallow in self-pity or get depressed or thinking 'why is this happening to me'. I never thought 'why me?' – not once. I didn't want to let myself cry or feel helpless."* Esther often complained of rumination at night before sleeping, and if those feelings arose, Esther noted, *"I didn't want to let myself go down that road. 'its midnight - come on you need your sleep'. The cancer-treatment is exhausting. You get tired and emotional. There were times late at night when I thought this is not a good idea to start thinking about all of this stuff – you are going to be lying awake until four in the morning, and you have got to do this and got to do that".* So Esther's use of distraction was solution-focused - to conserve her energy for getting through the treatment. Similarly, there were times that Esther would allow herself to *"collapse"* and feel the emotions, but she acknowledged that for her, it wasn't always helpful, *"I did cry….but I didn't always feel better after a good cry. I felt worse. I didn't like feeling like that."* This was because Esther was very aware of her own temperament, and that allowing herself to cry would be less a cathartic exercise, but rather exacerbate her own distress *"it's difficult as I am very emotional – I am very likely to cry. I do cry… [but] when something is in my face I know it is going to upset me. Crying doesn't help to make it better, it just makes it raw."* Esther's preferred coping strategies appeared to be related to her history of severe major depression, *"I am afraid it might stir up all the old… depression. I had that very badly and it took a long time to get over. I am very afraid of sliding"*. Esther did not want to descend into the emotional suffering she experienced as an inpatient, and was motivated to manage herself in such a way that she would not be vulnerable to depression again. Esther believed her strategy to be successful, *"I was never depressed when I got cancer – I went through it with positivity of mind*". However, after the cancer treatment had finished, Esther had significant problems *"letting things go"*, and acknowledged that she ruminated considerably, *"when things upset me I go over them so much…literally just replaying"*. She had struggled to move on and appeared deeply frustrated because her rumination *"doesn't achieve anything"*.

4.1.3.1.3.3. Social System

Much of Esther's rumination was not about her cancer experience, but rather on the quality of her significant relationships and how they affected her before, during, and after the cancer.

Past history: Esther described a painful childhood where her parents were *"too busy arguing"* to tend to the needs of their children. During this period Esther was being severely bullied at school, but chose not to share this *"I didn't see any point….talking about this with my parents"* because *"they were oblivious to the idea I have needs and thoughts [of my own]"*. Later on, Esther's grandfather died, which Esther says *"destroyed the family"*. She was *"not allowed to go to the funeral"*, despite the fact that she was *"devastated when he died"*. Over the years, she began to realise that she was growing up in an environment where she couldn't share how she felt, and that this was re-enforced by her relationship with her sister and her father.

Sister: According to Esther, she was the younger of two sisters, and her older sister used to constantly condemn her, calling her a *"terrible nasty person for being so utterly selfish. It was said with the most scathing contempt, as if there was no hope for me".* This was bewildering for Esther at such a young age,

and she was unaware of the effect that it had until later in life *"Initially I was too young to know. I got a whole load of flak without knowing what I did wrong. She gave me this awareness of what it meant to be selfish – always putting yourself first – always thinking of you"*. This led her to constantly second-guess her motives and put others first in her life, *"she always called me a selfish little bitch/cow. No nice words, no sweetness. I grew up with this terrible…'am I being a stupid little cow?'"* Though Esther acknowledged as an adult that such condemnation was more reflective of her sister's behaviour, she realised that as a child, it made her *"feel like an unlovable and horrible person"*. Esther also described her sister's patronizing attitude while she was going through the cancer treatment, *"She said to me…'I am so impressed with how you have dealt with this* [the cancer].' *Cheeky cow! She is so arrogant"*.

Father: Esther described her father as *"quite* [a] *violent"* man, who was very confrontational and argued with her mother a lot when she was a young girl. Esther learned that people *"had to be very brave to challenge him on any issues"*, and that he was *"totally oblivious…to be sensitive and aware of the growing needs of [his] girls"*. Sadly, he was never there to support her through her cancer treatment, and he was hospitalised indefinitely because of repeated falls following a stroke, and Esther had looked after him ever since. This was extremely hard for her given that his failure to support her resulted in her *"hardening [her] heart"* towards him, and that seeing him in hospital kept her chemotherapy *"fresh in [her] memory"*.

Partner: Esther described her partner as *"surprisingly understanding"* through the cancer experience, especially since he was going through a very distressing constructive dismissal at the time that she was diagnosed. Esther was supporting him emotionally at the time, and wondered if the sheer stress actually triggered the cancer. After he got through this stress, Esther described how they *"became a lot closer"*, but after she was cured, they *"became distant"* and went back to having a *"non-relationship"*. Esther was very hurt and angry about this, wondering why she lived through the cancer only to be confronted with this rejection. Later on during the therapy some of his behaviour toward her began triggering old fear reactions related to childhood conflicts.

Brother-In-Law: Esther described her sister's husband as a very difficult and antagonistic person who was also a *"binge-drinker"* with a history of sexually harassing her. Though he was generally not well-liked, he eventually developed cancer at the same time as Esther and *"died in agony"*. Watching him deteriorate was an awful experience for her, and she was wracked with guilt for *"wishing him dead"*. This was the precipitating event that led Esther to seek support from the PTSD Clinic.

Jackie: Esther has a very close female friend (Jackie) who was part of a recreational club of cancer survivors. She was well loved and respected, and Esther saw her as an inspiration. Unfortunately Jackie refused chemotherapy, her cancer returned, and it was terminal. This was extremely hard for Esther, as with her brother-in-law, she was going to die, and Esther was going to live, while *"watching [her] slowly deteriorate"*. This linked in with her sense of survivor guilt (that is explored in the next section). Jackie eventually passed away in between the final two sessions of emotional processing therapy.

4.1.3.1.3.4. Clinical Findings

Emotional Preparation: Esther was initially feeling emotionally drained after the screening and assessment because she expected to be talking about the cancer only, and not to talk *"about childhood so*

much." She found that it *"stirred up"* a lot of emotions and memories that were left unresolved, which had since been *"cropping up"* through the emotional preparation. However, Esther was more than happy to engage with the therapy as she realised that much of her emotional distress was related to her relationships surrounding the cancer, and did not want to remain stuck where she was. Part of the emotional preparation sessions involved reading the book *Emotional Processing: Healing through Feeling* (Baker, 2007). Esther strongly identified with the chapter on rumination and how she tended to run things over in her mind without any closure. She began to realise that the memories *"do relate to unprocessed emotions from previous things [in her life]"*.

Exposure Homework: Part of the exposure homework was to keep a diary and write-down memories to be re-lived in therapy. Esther was actively engaged in keeping a diary about her cancer treatment, but was sometimes taken by surprise at the power of the exercise to trigger uncomfortable memories and emotions *"I was fine at first, and it just came late at night after a glass of wine. And this emotion just, you know…it wasn't nice".* At times, she experienced high anxiety, but realised that she might not remember what came up unless she recorded it *"I had a panic attack. I wrote it down just after, 'cause I knew I [was] not gonna remember this by the morning".* Esther described the experience of writing down her memories as they were coming up, *"I began to forget it. I am doing my 'blocking off'"*. This really surprised her, *"I was very shocked…this was just too big. I…really churned up some old muck* [from childhood]*"*. However, Esther fully engaged with the process and spoke in considerable detail about these experiences in session.

Exposure Sessions: Though Esther's cancer treatment was very distressing, it was never appraised as a traumatic event. Rather, the worst aspects of going through the treatment were related to the insensitivity of the treatment process, her family's reaction to her needs, her apparent unworthiness to survive, and her guilt surrounding having a good prognosis compared to her brother-in-law and her friend Jackie, both of whom she watch deteriorate slowly while her health improved. Esther recounted how her friend Jackie really wanted to live, in comparison to Esther herself, was *'not worthy of surviving'* because she tried to end her life previously, *"…I did that suicide attempt back in 1990. I am not deserving cause of what I did then…as if I had been flippant. I was desperately depressed and wanted to end what was happening to me. I could see no way out. I did this terrible thing and its terrible to face within yourself….I have had to sit and think about it over the years. I didn't want to die."*

4.1.3.1.3.5. Follow-Up

By follow-up, Esther had no more intrusive recollections, and decided to investigate the concept of guilt, including its definition, and began to realise that she wasn't actually guilty of anything. *"[Guilt] is all based on doing a crime – so that's not the word for what I felt…so what was the feeling that washed over me?"* For her, she acknowledged how she had compared herself to others with cancer – those that didn't make it, and believed them worthy of surviving *"because they had achieved so much more with their lives"*, and that this meant they deserved to survive more than her, and realised *"what a load of rubbish that [was]"*, when she noted that this came from the belief that she never *"measured up to [her] parent's or her own expectations".* Esther was very emotional about the ending of therapy but found it very helpful, *"[Originally], it was these feelings that suddenly emerged and engulfed me! I know look*

back on it and its fine". Her realisation that she wasn't guilty lead her to believe that *"the trauma …the guilt – that was the thing…has gone".* This left her with the resolve to move forward, *"I don't want to react this way anymore, its stopping my growth as a person. I didn't go through cancer for no reason at all. I must learn. I must grow from this, otherwise what do we exist for?"*

4.1.3.1.4. Summary

Since the initial screening and assessment, it was apparent that Esther had endured multiple distressing events during the cancer episode that she experienced as truly terrifying. For Esther, the diagnosis and mammogram was shocking, and the chemotherapy and its possible side-effects were overwhelming and frightening. But, the cancer diagnosis and treatment were not experienced as traumatic or life-threatening (Criterion A1) because Esther knew she had a very good prognosis and so PTSD was not diagnosed. This may be because Esther knew that she had a good prognosis, and was therefore committed to getting through the treatment. Knowing that she was of a sensitive temperament, Esther took informed steps to make sure that she managed her thoughts and emotions in such a way that she was not overwhelmed by the treatment process. She used distraction techniques and self-talk to warn herself of the impact of rumination and worry on her health, and also took it upon herself to think about the welfare of others who were suffering at the time of her treatment. Before, during, and after her cancer treatment, Esther lacked emotional support from her family members, and only experienced limited support from her partner who was also going through a terrible crisis at the time that she was diagnosed. However, Esther did have close friends (one in particular) who were also cancer survivors, and role models for her.

Esther's experience of emotional preparation was that she was surprised much of her remaining emotional distress was related to the quality of her relationships before, during, and after the cancer and that this distress manifested in frequent rumination. She was committed to engaging in the therapy. During exposure sessions, it was evident that the worst aspects of the cancer experience for her were the overwhelming treatment process, and her family's lack of response to her needs. When doing the subsequent exposure diaries, Esther often re-experienced deep anxiety rooted in relationships with attachment figures that was triggered when aspects of the cancer treatment were re-lived. It became apparent that she was suffering from survivor guilt and a belief that she was unworthy of survival. The conditions under which Esther experienced these events were such that the diagnosis of cancer, while shocking, was not unforeseen, and was not life-threatening. A review of Esther's history, and her level of adaptation through the illness into survivorship, revealed that even though she has a serious vulnerability to depression, and lacked a lot of support from her immediate family, she decided to manage what she could and concentrate on getting through the illness without succumbing to depression.

4.1.3.2. Case Study: Angela

4.1.3.2.1. Patient History (SCID Overview)

4.1.3.2.1.1. Background

Angela (name and identifiable personal details changed) was a 37 year-old, unemployed, devoted single mother. She was referred over to the specialist cancer clinic from the local NHS Primary Care Adult Mental Health Service due to probable cancer-related PTSD. She started higher education but could not complete it due to needing to find work, and because of becoming a single mother. At the time of assessment, she was not working and on employment benefit while recovering from cancer treatment. However, Angela had one hobby that kept her occupied – swimming, which was her favourite. Angela gave informed written consent for her screening, assessment, and subsequent therapy to be recorded, and transcribed, for use in an unpublished case-report. However, according to CARE Guidelines (Gangnier et al., 2013), Angela has not given consent for these reports to be published as she has not read them and approved of their content.

4.1.3.2.1.2. Vulnerability Factors

Angela has never had a healthy relationship with her mother, and much of this revolved around the grief of losing her grandparents. This has been responsible for a lot of hurt surrounding the periphery of her cancer treatment. However, her relationship with her father was much closer.

4.1.3.2.1.3. Psychiatric / Medical History

Angela was diagnosed with Stage II breast cancer in September 2010, which was the most upsetting, saddening, experience of her life. She had one round of chemotherapy, radiotherapy, a lumpectomy, and then a full mastectomy. She also suffered the full range of deeply unpleasant and painful side-effects of the treatment program. Angela finally had her ovaries removed in August 2012. Angela reported a constant decrease in mood that progressed since the start of treatment, including emotional numbness and guilt over the effect of her psychological condition on her two children.

Angela did not report any treatment for emotional difficulties, or histories of substance abuse/dependency, being a psychiatric inpatient. However, within the past six months prior to the assessment, Angela had seen four mental health professionals across the primary care pathways, receiving short-term interventions for worry and depression. The short treatment episodes, subsequent re-referrals, and waiting times, were a significant emotional strain on her ability to trust her therapists and to engage in therapy.

4.1.3.2.1.4. Precipitating Events

Up to the time of assessment, Angela's mood had been getting progressively lower. She had been feeling sad, angry, and scared, with the additional guilt surrounding the effect of her depressed mood on her two children, although she did not report any substantial relational difficulties with them. In addition to her depressed mood, Angela reported numerous but intermittent physical symptoms that were side-effects of the cancer treatment such as lethargy, insomnia, some pain, and shortness of breath. Though

Angela had reported coping through the cancer treatment, she did not anticipate the continued operations. Just when she thought the treatment would be over, she would need another operation. She endured an initial lumpectomy, only to move to a full mastectomy. It was not until she was informed that she needed to have her ovaries removed that she could no longer cope. Angela explained that she believed that *'there was nothing more that could be taken from [her]'*, and that up until the ovary operation, '*they had taken it all'*.

4.1.3.2.1.5. Presenting Symptoms

Angela most significant complaint was of serious insomnia. It took her hours to fall to sleep, and within this period she has frequent nightmares related to the chemotherapy and of her funeral and leaving her girls behind. When she would wake up, she would be sweating, and feeling exhausted. When Angela is awake, she is also easily startled, and experiences sweating, breathlessness, and additional panic symptoms when exposed to reminds of her cancer treatment. Consequently, Angela recounts (and tries to avoid) many situations that trigger her distress: She also avoided watching television programs even loosely related to surgery, going back to the oncology ward where she was treated, social situations because of people's tendency to want to discuss the cancer experience with her. A key trigger for her distress was seeing a lady in a supermarket with a scarf on her head reaching the top shelf.

4.1.3.2.1.6. Risk / Substance Use

No evidence of suicidal thoughts or self-harm. No reported history, or present use, of drugs and/or alcohol to regulate emotional distress.

4.1.3.2.1.7. Medication

Angela reported taking Tamoxifen (cancer treatment), and also Citalopram 40mg for six months, and Amitriptyline at the prescribed dosages. She was also receiving adjunctive cognitive therapy for major depression from the referring mental health service.

4.1.3.2.2. PTSD Diagnostic Assessment (SCID PTSD Module)

4.1.3.2.2.1. Criterion A

Angela reported two traumatic events in her lifetime. The first being in a road traffic accident with her grandfather when she was 11 years old, and the second being the cancer experience from 2010-2012. The cancer experience was endorsed as the most traumatic, and currently distressing, event. She reported feeling horrified when she received the cancer diagnosis, and further questioning revealed subsequent dissociative and numbing symptoms during, and after, the experience.

4.1.3.2.2.2. Criterion B

There was strong consistent evidence that Angela experienced recurrent flashbacks of memories from the cancer experience, including distressing dreams, and severe physiological reactivity to triggering stimuli. Flashbacks and nightmares include reliving the chemotherapy needle going in, and the associated excruciating pain. Physiological reactivity includes panic symptoms triggered by specific traumatic reminders. Four re-experiencing symptoms were clearly endorsed, three in excess of requirement.

4.1.3.2.2.3. Criterion C

Since the end of the cancer experience, Angela reported several avoidance symptoms. Though she did not report an inability to recall key aspects of the trauma, there was strong evidence for her avoidance of conversations about her experience, places and activities that trigger trauma recollections, and systematic avoidance of television programs that remind her of her treatment. Angela strongly endorses emotional numbing symptoms of detachment and a diminished ability to feel love for her children and her partner, and a strong sense that her future has been foreshortened. Five avoidance/numbing symptoms were clearly endorsed, two in excess of requirement.

4.1.3.2.2.4. Criterion D

Since the cancer experience, Angela had clearly been experiencing significant insomnia and irritability, and was easily startled. She also reported some trouble concentrating, and persistent hyper-vigilance. However, caution was warranted due to these symptoms also being associated with the side-effects of cancer treatment. Four hyperarousal symptoms were clearly endorsed, of which two were *not* initially reported by Angela as known side effects of the cancer treatment (exaggerated startle, irritability).

4.1.3.2.2.5. Criterion E

Angela did not begin to experience the PTSD syndrome until August 2012, when her ovaries were removed. The PTSD syndrome had been present for seven months from that event up until the time of assessment, with several symptoms in excess of the diagnostic requirement. The temporal distance between the end of cancer treatment and the emergence of the PTSD syndrome suggested a delayed onset.

4.1.3.2.2.6. Criterion F, Psychometric Scores, and Final Diagnosis

Angela scored far above the cut-off (47) on the Davidson's Trauma Scale, with a total of 131. This shows that she was extremely distressed, and very likely to present with PTSD. It was also noted that there was depression before, during, and after the cancer experience, which could impair engagement with the exposure approach due to emotional numbing. Nevertheless, this was being treated in Primary Care. Angela was also above the cut-off on all the EPS-25 subscales, with a mean score of 8.2 (range, 7.4 – 8.8), which indicated a serious problem in emotionally processing distress. Angela particularly endorsed

strategies of suppression and avoidance, and problems experiencing emotions, as well as problems regulating the expression of her emotions. In addition, Angela scored very highly on symptom severity (DSSI) for anxiety, depression, and ruminative states. The clinical interview (as conducted by RB), also suggested PTSD. Given the severity of her scoring on the Davidson's Trauma Scale (131), the duration of the PTSD syndrome (seven months), and the point of the syndrome's emergence (well over six months since the traumas indicated in flashback content), a diagnosis of severe, chronic PTSD with a possible delayed-expression, was justified.

4.1.3.2.3. Therapeutic Intervention

4.1.3.2.3.1. The Traumas

This section is a timeline of her experiences as they were re-lived in prolonged exposure therapy. They range from finding the lump, through to diagnosis, and the treatment phase. It includes the actual events plus her reactions.

Finding the lump: One night, Angela went to bed, but woke up startled with the urge to check her breasts. Both of her daughters were asleep at the time, and she was all on her own. She got up, and for 15 minutes stood naked in front of the mirror and massaged her breasts, where she found a lump, and took hold of it. She recalled experiencing disbelief, and was numb with shock. It was very hard for her to process, thinking *"It can't be me I'm too young…I'm only 35…." "I can't have it, I'm only 35…"*, and then recalling her mother also had breast cancer two years prior. The next day, she was *"feeling frightened and overwhelmed"*, but not wanting to worry her children, she *"woke up and acted normal"*. After taking them to the school, she went to her GP and had the lump examined. The GP examined it and was uncertain until Angela disclosed her mother's breast cancer diagnosis two years prior. An appointment was then arranged, but the letter took weeks to arrive. During that time Angela was extremely anxious, but told no one of these events. She decided to distract herself and look after her family.

Scan / Biopsy / Results: When Angela attended the biopsy she was already overwhelmed with anxiety and felt it was hard to process what was going on around her. She described sitting in a waiting from that was *"…very silent." She "didn't know how to feel"* while she was waiting for her series of scans, but because more unsettled when she began to realise that *"something isn't right"*. By the time she was going through these scans, Angela became very distressed by the diagnostic process. She experienced it as if the doctors were *"…rushing [her] around, and fast-tracking [her]"*, all the while worrying that she *"…can't be ill….don't have time…it can't be anything"*. Angela recalled her first biopsy where she was injected with local anaesthetic, and then had four samples of breast tissue taken from her. This was a horrible experience for her. Angela described it as *"a stapler…a horrible noise…they really pushed it in…it hurt."* She was *"shocked"*. It was *"overwhelming [for her], and it happened so fast"*. Angela described the thoughts she was having while the biopsy was being performed *"something's not right…something's not right"*. She then had to wait a week for her next appointment to discuss the results. But as Angela reported, *"they were inconclusive. I had to have it done again. I then realised it was serious*". She

described the second biopsy as *"hurting even more than the last one. I was very scared, [but] they got those results back [comparatively] quickly in a couple of days"*. By the time that the results appointment came, Angela was waiting to be called in, but was described being so overwhelmed that she did not hear her name being called. Angela described how she prepared for that moment – *"I just detached myself from me….I went into robust mode…and did everything the doctors wanted me to do."*

The needles (Chemo) / Hair falling out: Angela reported that she was advised a cycle of chemotherapy first to shrink the breast carcinoma before the mastectomy. By the time the first day of chemotherapy came, Angela has arrived on the ward with her close friend (affectionately dubbed *'stepmum'*). Angela was daunted by the prospect of chemotherapy and that she was *"the youngest one there [in the ward]"*. When the time came to receive the chemotherapy, she *"...got completely overwhelmed…and was very scared…shaking uncontrollably"*. Angela recalled thinking at the time that *"it was actually happening – it was real"*. At this point the nurses were having difficulty getting the needle into a viable vein, and this was causing Angela a lot of pain and distress. A second nurse finally succeeded. She then recounted when she first began to lose her hair, a week and a half after the first chemotherapy cycle. Angela was having a wash, and the hair started falling out in the in the bath. This was *"very distressing"* for her. Angela then bought head scarfs to cover her head, but refused to look in the mirror again until after the mastectomy.

Inpatient Stay / Waking up from Mastectomy: Angela was recalled into the hospital and had to spend one week in isolation because of *"...dangerously low white blood cells"*, and had to receive daily injections to improve her immune system. This was exceptionally distressing for Angela, who described it as *"a kick in the teeth"*. For her, the chemotherapy did not appear to be shrinking the tumour (as was intended), but was destroying her immune system… *"....yet another thing the chemo was doing to [her] body"*. Angela was angry that they didn't operate first, but given the lack chemo's lack of effect, was afraid that they *"would open [her] up and find something somewhere else, or bigger than what they thought, and would they be able to get it all"*. After waking up from the mastectomy, Angela stood in front of a mirror. She had not seen herself bald because she had refused to look into a mirror until this point. She recalled that *"the hair was a shock…I just remember thinking I look awful."* Angela also found it hard to take in *"what they had done to [her]"*. She recalled that she *"looked totally lop-sided"* and that *"it wasn't [her]"* in that mirror. She also stated that *"*[The breast]*…its just not me – its fake. I am now covered in scars…I had these two drains hanging out of me. Just horrible…but I knew I had to have it off….I didn't have an option. I was 35 years old and having a mastectomy – something else was taken away from me – out of my control.* Angela found it horrific to looking the mirror and see / experience the mutilation of her body, and since her initial diagnosis, described feeling that she had *"lost [her] self along the way"*.

4.1.3.2.3.2. Coping Strategies

For Angela, emotional displays were believed to be a *"sign of weakness"*, and that when she senses she is about to cry that she *"shakes it off"* and tells herself to *"stop being so stupid"*. Angela learned to suppress the experience of unpleasant emotion - especially tears. It became apparent that Angela had significant difficulty describing or experiencing her feelings, often using simple labels such as *'sad'*,

'annoyed', or *'hurt'*. When Angela did describe her feelings, she showed uncertainty about the truth of her evaluations, often saying *"I don't know…maybe"*. There was often a lack of emotional expression and long silences. For Angela, the expression of her emotions was associated with overwhelming the emotions of those in whom she chooses to confide *"I don't like putting on people".* During the cancer experience in particular, Angela recounted that friends and immediate family were immediately *"overwhelmed by what [she] said"* , and what was especially painful for her was that they were so overcome that they could not support her, and Angela started supporting them in their distress instead. Unsurprisingly, during the course of the disease, Angela kept *"…things bottled up and put a costume on in front of everyone. A false sense of…that everything was ok and nothing was wrong".* Though Angela acknowledged that this strategy contributed to her chronic low mood and intrusive memories, she was afraid that *"maybe [she had] bottled-up so much now that it's…what's the word…overtaken [her]."*

4.1.3.2.3.3. Social System

Past history: Angela's beliefs about the experience and expression of emotion had origins in family relationships that predated her traumatic experience of cancer. Angela told the story that she was strongly influenced by her grandmother when she was growing up, and one of the things she learned was that *"crying wasn't the done thing"*. She never saw her grandmother *"cry or raise her voice"*, and if Angela cried her grandmother would say *"don't be so silly!"*. Angela was really close with her grandparents, and was deeply bereaved when they died. But she was unable to process her own grief because her father, with whom she had a close relationship, was also devastated by the loss of his own parents. Angela took care of her father and put her own needs to the side. When asked about this, Angela stated that she didn't know why it happened this way, but that she *"…was always the strong one…who held everything together"*. A similar pattern emerged when her mother was diagnosed with breast cancer. Angela stated *"being the eldest daughter, I put my feelings aside to do the right thing."* This was hard at the time because she did not have a good relationship with her mother, and her intention to support her mother and siblings was *"thrown back in [her] face"*. This grieved her deeply, stating *"[I'm] sad that I haven't got a relationship with my mum. Every girl should, and I've never had that. I don't have nice memories with her."* A saddening pattern emerged in these relationships. For the majority of her life, "*whenever a relationship had broken down",* Angela *"would put it aside and just get on with it."* Eventually, she would just *"break away"* from the relationships that hurt her. As a result, Angela learned to *"block off"* her feelings, and *"never really deal with anything"*, and to *"worry about others"* rather than herself. Much of what Angela learned through her family history was played out during the cancer episode. Her key relationships were with her father, partner, her friend Jessica (affectionately dubbed '*stepmum'*), and her two young daughters.

Father: Her relationship with her father was very close and affectionate, with a lot of reciprocal love and concern for each other's welfare. However, much of the dialogue about this relationship centred around the theme of her protecting her father from her own illness, believing that he will be overwhelmed with worry if he knew how she was feeling. This appeared to stem from her father's history of suffering from *"manic-depressive episodes"*. Angela stated *"Before I was diagnosed it was always me who was looking after Dad."* But *"the role reversed and took its toll on him. If I was to tell him what was going on in my mind, I don't think he'd cope very well with it. So I protect him from it."* For this reason, Angela

thought it best to cope with the cancer herself because she could not bear to see him upset. When Angela received her diagnosis, though she was distressed, she was more concerned about her father, thinking *"I can't be ill, how am I gonna look after Dad?"*. She had Jessica give him the news because she could not bear to see his reaction. She said *"it broke him. He was beside himself with worry, crying. I think even then I was trying to hold it in".* During the exposure component of the therapy, Angela began to cry for the first time when she re-lived seeing her father's pain *"…he was very helpless. There was nothing he could do to help his little girl. I tried to comfort him".* However, by the time Angela was due her mastectomy and needed a hospital stay due to her low immune system, she recounted how her father said *"you gotta stay in and do it for me"*, and how there was a lot of resistance from her *"I don't have to do it for anyone"*. The treatment regime was horrific for Angela, yet it appeared preferable to seeing her father *"tearing up"*.

Partner: While her father appeared unable to emotionally support her through the cancer, her relationship with her partner was strained in physical and emotional intimacy. Angela describes her partner (whom she met during her radiotherapy) as loving and well-intentioned, *"He can be supportive…he does try. He says I can talk to him…",* but is unable to empathise with her because *"…everything is black and white with him – there is no in-between. I have got over the illness, and I should be ok now."* According to Angela, attempts to communicate with him have mixed results. *"I try to sit down with my partner and talk to him, but he doesn't listen"*. When trying to explain to him her fear of recurrence, she felt like if she told him she was *"afraid it would come back, he would be…like… 'no it won't!'"* In her mind, she was really annoyed because he cannot know that for sure, as *"not even the doctors know"*. It was evident that Angela was genuinely frightened by the uncertainty of her prognosis and he was unaware. As previously covered, Angela also has significant difficulty accepting her body after chemotherapy and mastectomy. This led her to refuse physical contact with her partner. Though he explained to her that her body *"doesn't bother him"*, she recounted that it bothered her.

Angela also reports that her partner tries to get her to talk about her feelings, but kept getting *"frustrated because [she] wouldn't open up to him"*. She believed that he *"just wanted a reaction"*, recounts that she has tried to explain that she *"doesn't know how [to talk]",* and that she feels like *"there is nothing there… [she's] just empty".* Angela was aware that her partner could see that she is really unhappy and withdrawing from him, and that it was affecting his resolve to be in a relationship with her. By the last session, Angela stated that the relationship had ended because *"[she] was not ready"*, but that she was really lucky to have had a partner who accepted her as she was through the cancer treatment.

Jessica ('surrogate-mum'): This friendship was perhaps the most significant one during cancer. Jessica was an older friend whom Angela saw as a *"mother-figure"*. Jessica was the only person in Angela's life that has the ability to be strong with her. During the scan, biopsy, and chemotherapy, she was always there to support Angela, and help handle her father, *"…she'd been through a lot with me. If ever there was a problem after the chemo – she'd come to the chemo's with me. She was there from day one."* However, one day Angela needed someone to drive her into hospital for her mastectomy, and described how Jessica just turned on her, and accused her of using her and her daughter. Angela reported feeling *"shell-shocked and numb"* and that she *"didn't need this"*, so she went in with another friend to have the mastectomy. During an exposure session, Angela re-lives waking up from the operation, and recounts what she was thinking at the time: *"When I awoke…I was mainly focusing on the op….didn't*

really think about Jessica, until I was more with it and able to get up. I was looking out the window thinking would she come today – she never did [voice trembling]. The surgery had disfigured me, and I couldn't stare at it long. After the nurses left, I sobbed. I couldn't phone Dad – or Jessica… I felt on my own" After the cancer treatment, Jessica attempted to repair the relationship, but Angela was numb towards her, explaining that the experience has made *"me wary to accept anybody's help. I'd rather muddle along by myself"*.

4.1.3.2.3.4. Clinical Findings

Emotional Preparation: Angela was initially very *"anxious and nervous"* about the prospect of allowing herself to feel emotions, thinking that *"being tearful would be strange for her",* especially since the consequences for her involved experiencing flashbacks, and for others, seeing them very upset. When asked how she felt about talking and crying, Angela said "*its strange…alien…even now a little bit is coming back up…that I buried"*. Nevertheless, Angela was very courageous and decided to engage in therapy because she was *"fed up of feeling like this"*. Part of the emotional preparation sessions involved reading the book *Emotional Processing: Healing Through Feeling* (Baker, 2007). Angela did so, and was *"…quite shocked how [she] could relate to it…me reacting to people, snapping at them – I didn't feel so alien – I am not the only one. There's hope."* Another part of the emotional preparation involved discussing the therapy with their support system. Angela asked her partner to read the book so he could empathise with her position, and he agreed.

Exposure Homework: Part of the exposure homework was to keep a diary and write-down memories to be re-lived in therapy. Angela found the writing down very hard as *"it brought up feelings that [she] didn't remember"*. Subsequent attempts to write the diary of her cancer experiences resulted in her *"crying her eyes out"* at home, and it was too emotional for her to cope, and there were several sessions where diaries were not completed. However, talking about the experiences was easier for her, but it was interesting to note that the act of talking was more factual, and there was a lot less emotion being communicated, in place of a *"numb"* feeling. The second homework involved exposure to her body-image by looking at her naked body in the mirror. However, this was too distressing for her and she could only look for a few minutes before disengaging. For Angela, the experience of engaging in both types of homework was *"weird"* and *"strange"*, as if the experience of these emotions was unfamiliar. When she was about to cry from the exposure, she noticed her mind say *"stop it!"* really loud. When asked what might happen if she didn't stop herself crying, she stated that she *"didn't wanna let the illness get the better of [her]"*, although even then she sounded uncertain about whether that was the case.

Exposure Sessions: After one session Angela began to notice that the flashbacks were increasing in recurrence. In the week she had woken up in panic from nightmares, and was understandably nervous about her second exposure. One important clinical finding was that when Angela recounted the memories from her diary in session, there was very little emotional engagement, but a rather numb demeanour. Angela confessed that this was how she coped with emotional pain and that it had a long history back to her childhood relationship to her parents and grandparents. However, gentle encouragement from RB and LP lead to Angela beginning to express deep sadness and cry, not just over her experience of cancer, but over the thought of burdening loved ones with the emotional pain she was in. By the end of the final

session, Angela was able to stand in front of the mirror for 20-minutes and look at her scars, and her emotional reaction was much less intense. She has decided to get tattoos to cover the scars to improve her body-image and start accepting and loving her body. Angela's flashbacks had ameliorated significantly as well *"Reaction isn't as strong now…not having as many flashbacks as I did"*.

4.1.3.2.3.5. Follow-Up

No follow-up could be conducted. Angela kept cancelling appointments. It was possible that she was avoiding the ending of therapy because it resembled another ended relationship.

4.1.3.2.4. Summary

Since the initial screening and assessment, it was apparent that Angela had endured several distressing events during the cancer episode. Finding the lump, having the scan/biopsy, and receiving the results, having the chemotherapy/side-effects, and the mastectomy, were all endorsed as traumatic and were experienced as sudden, unexpected, and very frightening. Though Angela described the traumatic experience of physical pain as part of the treatments, the worst for her was the systematic removal of body parts that made her female, destroying her gender identity. The events experienced by Angela were endorsed as traumatic and do meet DSM-IV PTSD Criterion A1 (APA, 2000), her appraisals throughout the illness were ones of helplessness and horror (Criterion A2).

Angela's coping strategy during the cancer episode was one of experiential and expressive suppression. This approach was used because of the effects that her distress would have on others, and great effort was made to ensure people did not see how distressed she was. This strategy had its roots in earlier attachment experiences with her own parents surrounding the loss of her grandparents. Angela described a scenario where she had very little emotional support because her father could not cope with her having cancer. Her one friend who supported her through the illness suddenly withdrew her support before the mastectomy. Angela's relationship with her partner was generally well-meaning and supportive, but his lack of empathy for her feelings and situation had further contributed to the lack of intimacy between them that stemmed from Angela's poor body-image.

Angela was initially nervous about emotional preparation because experiencing and expressing unpleasant feelings would be unfamiliar to her, but was willing to engage in the hope that she could recover. Angela had significant difficulty doing the exposure diaries as writing them was too emotionally overwhelming, whereas talking about them was easier, but was accompanied by numbing feelings. The exposure was stalled until the roots of the emotional numbing were explored, and emotion began to be expressed. By the end of treatment, the flashbacks had ameliorated significantly (but not completely), and Angela had begun to re-appraise her body image and take steps to improve it.

The conditions under which Angela experienced these events were such that she was unable to process receiving the diagnosis, or the need for treatment. A review of Angela's history, and her level of adaptation through the illness into survivorship, revealed that she had little opportunity to cognitively process her trauma, or, given her tendency to avoid emotional experiences and expression, the capacity to experience and process them herself. It is therefore possible that Angela's emotional processing style

interacted with the trauma and her recovery environment to predispose her to develop a chronic PTSD syndrome.

4.1.4. Discussion

Phase 2 required in-depth case studies of a patient's psychological adaptation throughout the cancer treatment. Both of the patients were selected to provide follow-up explanations for the findings in Phase 1, specifically related to variables that influenced the psychological adjustment to cancer throughout the treatment, and lead to their post-cancer presentation at the PTSD Clinic (subsyndromal/adjustment disorder *v* full PTSD). These variables were emotion regulation strategies, and the beliefs and schemas that may underpin their use.

4.1.4.1. Clinical Findings

In terms of the cancer experience, both patients were comparable in terms of the severity of their disease, the treatments administered, and the relative side-effects, and both patients found the experience horrifying and overwhelming. However, Angela, who in addition had her ovaries removed, appraised the entire experience up to that point as destroying her body and femininity. This was not a problem for Esther. Esther, while finding the cancer experience harrowing, did not endorse it as traumatic, but rather as "*an ordeal"*, from which she would emerge alive and comparatively unscathed.

From these case studies emerged two distinctive findings. First, the role of social support and/or constraints during and after the cancer, and second, how each patient reacted, including the strategies used to cope with the illness. Both Esther and Angela had little to no support from their immediate family, and limited empathy from their respective partners, although both did have at least one female friend who was supportive and inspirational. However Angela also lost this source of emotional support prior to her mastectomy. It is apparent from these case studies that the lack of emotional validation and support made a strong contribution to the chronic distress of both Esther and Angela. The implications here are that less support was associated with a greater likelihood of numbing responses and avoidant emotional processing styles, which was also observed in Angela's case. This is commensurate with previous research showing that social support is a negative correlate of PTSD symptom severity (Andrykowski & Cordova, 1998; Green et al., 2000; Jacobsen, 2002; Kornblith et al., 2003), and social constraints on talking about cancer predicts greater distress and PTSD symptoms (Cordova et al., 2007). Older studies have also demonstrated a powerful effect of social support on reducing emotional numbing and avoidance (Andrykowski et al., 2000). Research in other populations has revealed a link between social support and adjustment, in both military veterans (King et al., 1998), and bereaved mothers (Lepore, Silver, Wortman, & Wayment, 1996). In Lepore et al's study, mothers who experienced initially high levels of intrusions in unconstrained social systems exhibited a significant decrease in depression symptoms over time, compared to those with constrained social symptoms, who developed more severe depression symptoms over time.

Esther and Angela endorsed the use of one or more unhelpful strategies on the EPS-25/DSSI, and also described these at length during emotional preparation. Esther used distraction techniques throughout the

illness, but also complained of persistent rumination after her treatment that was related to her close relationships. Angela however, clearly endorsed the use of suppression, both experiential and expressive, resulting in a persistent sense of numbing throughout the therapy. The difference here was that even though Esther did endorse suppression on the EPS-25, it was clear from the sessions that she was rather using distraction out of a psychological awareness of her own sensitivity and need to mobilise resources to survive the treatment. In this sense, Esther's coping strategy allowed a more internal locus of control which facilitated a solution-focused approach. Angela, however, used suppression since diagnosis out of the fear that expression of sadness and tears was weak, and the concern that it would overwhelm her already strained support system. As such, her coping strategy appeared more emotion-focused, with the goal of disengaging from them altogether. These observations are consistent with prior research showing that an optimistic outlook and proactive support-seeking were associated with better quality of life and adjustment after cancer (Halstead & Fernsler, 1994), and that problem-focused engagement was negatively correlated with distress, whereas emotion-focused disengagement was positively correlated with distress, (Epping-Jordan et al., 1999). Similarly, cognitive avoidance predicted greater PTSD symptoms in women with cancer (Hampton & Frombach, 2000), and other studies have demonstrated that suppression exacerbated PTSD symptoms (Amir & Ramati, 2002). Also, Angela's alexithymic presentation appeared to be linked with the emotional numbing that she experienced during and after her cancer diagnosis. This may have clinical implications as the early use of dissociation has been shown to predict PTSD development in cancer patients (Kangas, Henry, & Bryant, 2005). It also was apparent from the sessions with Esther and Angela that rumination was partially responsible for the chronic distress that they were experiencing, and this is consistent with previous research showing that negative attention biases can manifest in rumination which predicts (and partially mediates) the severity of future PTSD symptoms (Chan et al., 2011).

One feature of the cancer experience that differentiates it from other traumas is its long duration, and that, as Phase 2 demonstrated, can often occur in an emotionally invalidating and unsupportive environment. The role of rumination and the persistent use of emotional avoidance strategies within an otherwise unhelpful social system suggested the possibility that as the cancer experience is long-term, patients have longer to evaluate their circumstances, facilitating persistent negative ruminations, and chronic processing of distress consistent with the major course diversions posited in dual representation theory. According to Brewin et al., (1996), chronic emotional processing can occur when individuals undergo prolonged traumas, and cannot process them effectively due to a) multiple stressors competing for resource allocation; b) secondary emotions (such as anger and shame), or c) successive traumatic events or threats that reactivate trauma memories. This course is likely when stressors are repeated; there has been previous trauma, and poor social support. These conditions are consistent with the cancer experiences that are described by Angela and Esther, who experienced additional life stress from their unhelpful social networks. However, despite that finding that both Angela and Esther appeared to take this course, only one developed chronic CR-PTSD.

The fundamental differences in presentation between Esther and Angela appeared twofold: first Esther did not report depression symptoms during or after the cancer, but Angela did. And second, both patients differed in their adjustment to the social constraints. As such, their presentations may be partially attributable to the combined influence of emotion processing styles *and* the degree of supportiveness in

their close relationships. These case studies have highlighted a possible interaction between avoidant emotional processing strategies and social constraints on talking about the cancer, and this is supported by social-cognitive processing models in the emotional adjustment to cancer (Lepore, 2001). Though Lepore's social-cognitive processing model (2001) is not diagnostically-focused, it is highly applicable to PTSD as previous clinician-researchers have indicated that not only is a lack of social support a strong predictor of PTSD (Brewin et al., 2000), but that the presence of social support, and the need to emotionally engage with the support by talking with others, is integral to the emotional processing of PTSD symptoms (Ehlers & Clark, 2000; Foa & Cahill, 2001). Though idiosyncratic coping strategies and social support are both identified as independent contributors to PTSD symptom severity, less has been said on the potential interaction between these two factors in the cancer population, though this has been identified in other trauma populations (Hoyt et al., 2010).

According to Lepore (2001), the availability of social support from close relationships during the cancer is a good predictor of the level of adjustment experienced by the patient. Those patients with support networks that are emotionally unavailable generally suffer from poorer adjustment. As such, Lepore argues that the social context of the recovery environment can influence adjustment. As already established in this thesis, the cancer experience from diagnosis through to recovery has the potential to be traumatic, and as such, can force patients and survivors to re-evaluate their beliefs regarding their own locus of control, mortality, and the quality of their relationships with others. The discrepancy of pre-existing schemas with peri and post traumatic experiences is often implicated in the degree of distress (Horowitz, 1986; Janoff-Bulman, 1992), and a period of adjustment is required to emotionally process these new experiences into existing schemas by assimilating (re-appraising experience to fit with existing understanding), or accommodating (changing beliefs to accept new experiences). Of course, this process is distressing, and requires a significant degree of emotional resources, of which much is expended on surviving the treatment. According to Lepore, the process of emotional adjustment (or emotional processing, in context of PTSD), can be achieved if experienced in a supportive social context. Talking with others provides the opportunity to process emotions, absorb new perspectives, mobilise coping resources, and learn new strategies. However, Lepore notes that the quality of the exchange moderates the processing of distress. Social networks that are critical/unsupportive of the survivor's distress may inadvertently increase their psychological symptoms. Lepore (2001) argues that the key element here is that if social constraints are present in the survivor's support network, the cancer survivor may *react* by using supressing or avoidant strategies. And, of course, this has the potential to prevent cognitive processing, and put the cancer survivor at risk of chronic distress.

It is the nature of these reactions/responses that was most evident in both case reports. Both patients described limited, unavailable, and emotionally invalidating relationships. However, the distinguishing feature was that Esther decided to respond by re-appraising her situation and do what she could to survive the illness. Angela, however, was completely overwhelmed, and reacted by shutting down emotionally to the point she could neither experience nor express the level of her distress to her significant others or fully engage with the therapy. This points to the general understanding of how avoidant strategies prevent processing, but also builds on existing knowledge because it highlights how the course of events and idiosyncratic coping strategies within the context of a support network may engender systemic interactions that result in vulnerability or resilience to PTSD. For example, clear, authentic open

expression may facilitate effective help-giving in a benign social context; poor support from a critical/invalidating system may trigger reactions in the individual that mean they deal with emotions themselves. Conversely, pre-existing withdrawing/avoidant strategies within the cancer survivor may prevent effective resource mobilisation from a caring support network that might otherwise serve as a buffer against the distress. Nevertheless, the robustness of this statement rests on the quality of the study, so we must consider the strengths and limitations of Phase 2.

4.1.4.2. Strengths

The key strength of Phase 2 is that it permitted the investigation of complex relationships intrinsic to the patients' degree of emotional adjustment throughout the cancer treatment that would have otherwise been impractical and unrealistic under a quantitative paradigm. In Chapter 1.2 it was argued that quantitative measurements of hypothetical risk factors often pre-suppose linear relationships and treat them as moderators by virtue of capturing it as a single measurement, and thus failing to capture the course of adaptation. These two clinical case studies provided rich, detailed, but nevertheless preliminary data, on a possible interaction of emotional processing styles and schemas with social support, and the potential effect on the chronicity of adjustment disorders or PTSD. It should also be noted that competently-written clinical case reports require qualified professionals to diagnose, treat, and observe the patient. Several steps were taken to ensure the quality of assessment and treatment. First, though the investigator administered a series of structured clinical interviews, their accuracy was verified by subsequent interviews conducted by a consultant clinical psychologist and oncology specialist who were blinded to the results of the initial screening; the diagnostic accuracy rate was 100%. Second, case history was abstracted from patient records, referral letters to the PTSD Clinic, as well as the patient. Third, the emotional processing therapy was trauma-focused and commensurate with NICE Guidance (2005) for the treatment for PTSD, and too was conducted by a consultant clinical psychologist. Fourth, the investigator is a qualified mental health para-professional with sufficient assessment skills, and additional knowledge of prolonged exposure therapy, administered for a range of anxiety disorders. The investigator is also well-versed in the emotional processing model (Baker, 2007), and its subsequent application to PTSD (Baker, 2010), having previously investigated the role of emotional processing in CBT (Baker et al., 2011), and contributed to the rationale for the emotional processing protocol used to treat PTSD (Baker, Gale, Abbey, & Thomas, 2013).

4.1.4.3. Limitations

Though these findings enjoy some support from established theory, practice, and quantitative research, it should first be noted that the two cases studies presented cannot, and do not, represent the full range of clinical presentations that may be encountered in practice, and therefore should not be generalized. However, they do serve to highlight broad differences between a classic case of adjustment disorder and a comparatively uncommon case of chronic CR-PTSD. These findings may be used to generate new research questions regarding the factors that differentiate subsyndromal and chronic cases of PTSD. Second, the qualitative nature of this phase always involves subjective interpretation. It is therefore possible these findings reflect *a priori* assumptions about relationships between different factors and the worldview of the investigator. However, the assessment and treatment of psychological disorder

is based in ‘talking therapy’. This method of information exchange, and the way in which client and therapist form an alliance and an understanding of the presenting issue, has its roots in social constructivism. By definition, the new knowledge gathered in therapy is neither discrete nor separate from the entity that communicated it, but rather an abstract phenomenon that emerges via social exchange. Given that PTSD and depression is highly co-morbid, much of the distress in these two patients is rooted in their relationship to themselves, the world and their future, of which these beliefs are learned and maintained in a social context (Beck, 1979). As such, the emergence and maintenance of these difficulties is based on idiosyncratic perceptions, and therefore socially constructed. In short, this level of subjectivity merely reflects the ontology (what can be said to exist, and the extent that constructs or discrete entities can be classified), and the epistemology (the nature of knowledge, and how it is learned), of the case study approach. Rather than imposing a limitation, this is the root of its strength and applicability. The final concern is therefore not of the appropriateness of this approach, but on how to synthesise quantitative and qualitative datasets that differ in their ontology and epistemology (Smith, 1983; Lincoln & Guba, 1985).

4.1.4.4. Future Improvements

However, the role of appropriate quantitative and qualitative synthesis is only valid if the qualitative data meet the ontological and epistemological assumptions of the approach (Bazeley, 2004). The first issue is that the idiosyncratic approach of case studies reduces generalisability. Two case reports do not reflect the diverse range of presentations for breast cancer survivors or survivors of cancer in general. Future investigators may consider a larger series of case studies in female breast cancer survivors and then move to other cancers. They might choose to explore post-treatment psychological maladjustment in women from different age groups following the evidence that younger age is a strong predictor of cancer-related distress. Similarly, they may choose to investigate if different treatment regimens affect the adjustment process of individual patients. A broader and richer perspective on post-treatment psychological maladjustment may be achieved by implementing a larger and more comprehensive case series which focus on the variables described in this thesis. However, questions remain regarding the validity and reliability of data analysis.

This particular phase involved case studies in clinical settings to reflect what a clinical psychologist and oncology specialist might see in practice, and thus demonstrate strong ecological validity. Nevertheless, the social constructivism intrinsic to qualitative enquiry may suggest that the idiosyncratic perceptions of the singular investigator may also have generated the findings. Validity may be improved in subsequent case studies by employing one or more highly experienced qualitative researchers to perform an interpretative phenomenological analysis (IPA) of assessment and therapy transcripts to elicit recurrent themes in patient experiences. This could be further improved with qualitative analysis software (Bazeley, 2004). This interpretative framework will facilitate the collection of broad categories and themes in a way that clinical observation cannot. Oncology research enjoys a diverse range of experienced researchers who have generated qualitative literature providing rich detail on patient’s experience of cancer care and psychological adjustment (BPOS, 2014). If enough clinical cases are gathered, themes regarding the traumaticity of cancer experiences, and challenges in psychological adjustment may provide knowledge to begin developing cheap, quick, and effective screening tools to detect those who are vulnerable to psychological maladjustment in survivorship.

4.1.4.5. Conclusions

Overall, each patient appraised the experience and aftermath of the treatment differently, and consistent with PTSD diagnostic criteria, this influenced the initial course of their adaptation to the illness. However, the differences in personal coping strategies and social support between the two cases suggest a pivotal role of social constraints on talking about the illness, and how the presence or absence of social support may trigger temporally precedent emotional processing styles. Regardless of the initial appraisals of cancer and treatment, it is apparent from these two cases that the combination of social constraints and avoidant emotional processing styles may predict chronic courses of maladjustment, and in extreme cases, CR-PTSD. However, extensive research is required to test substantiate this conclusion.

Chapter 5 - Discussion

This thesis employed a sequential-explanatory mixed methods design to elucidate the roles of social and cognitive factors in the development and maintenance of PTSD in cancer survivors. As such, this research was divided into two phases (1, quantitative; 2, qualitative), where the findings in the first phase (Chapter 3) were linked to the second in the form of follow-up explanations in phase 2 (Chapter 4). Phase 1 identified the low prevalence of CR-PTSD in cancer populations (Study 1) and also showed that cancer survivors with CR-PTSD or adjustment disorder (AD) were differentiated by clinically and statistically significant differences in problems experiencing, linking, and labelling emotions (Study 2). Similarly, the severity of CR-PTSD symptoms was independently predicted by problems experiencing, linking, and labelling emotions (Study 2). Consequently, Study 3 was conducted to test the ability of emotional processing styles such as suppression and behavioural avoidance (on the EPS-25), and beliefs about the experience and expression of emotions, to predict adjustment to stress. It was found that emotional processing styles did not predict adjustment, but rather beliefs about the incomprehensibility of emotion was the single best predictor. These findings required follow-up explanations. First, though many of these variables were significantly implicated in the differential diagnosis of CR-PTSD, a) much of the variance was unexplained; and b), the adjustment disorder group also suffered with clinically significant problems in experiencing, linking, and labelling, emotions, albeit to a less disabling extent. Second, it was unclear *how* these problems were implicated in the adjustment during and after the cancer. Study 4 was conducted to provide follow-up explanations for how problems experiencing, linking, and labelling emotions contributed to the development and maintenance of CR-PTSD.

Study 4 presented two case studies: one with cancer-related AD, and the other with CR-PTSD. Clinical observation of these two patients suggested that their deeply-held beliefs about the experience and expression of emotion influenced how they managed their distress during and after the cancer, but that this was also influenced by the emotional availability of their support system. This suggested (for these two patients), that the interaction of these elements was strongly implicated in their course of adjustment. Though Study 4 present two isolated instances (and therefore cannot be generalised), the presented findings are highly consistent with findings from military veterans (King et al., 1998), bereaved

mothers (Lepore et al., 1996), and social-cognitive processing models in adjustment to cancer (Lepore, 2001). This bears similarity to diathesis stress models for PTSD (McKeever & Huff, 2003), so it is prudent to consider the broad theoretical, clinical, service-based, and future research implications.

Chapters 3 and 4 did discuss the findings, strengths, limitations, and research implications of each study in their respective sections. But this thesis has yet to discuss these findings as a whole and consider the respective implications in a broader context. In order to do that, this thesis must first discuss the various ontologies and epistemologies in mixed methods, how this creates boundaries of inference and generalisation, and the implication implications for data interpretation and synthesis. This is followed by an overview of the findings and their emergent clinical and service-based implications. Finally, a range of research strategies are proposed to address the aforementioned implications.

5.1. The Ontological & Epistemological Orientations of Quantitative, Qualitative, and Mixed-Methods Approaches

5.1.1. Empiricism

First, the fundamental principle on which science is based originates in the philosophy of empiricism. That is, all knowledge (and the frameworks on which this knowledge is constructed) originates in experience, and any belief that is held can only be truly validated through experience. The epistemology of empiricism is that organisms can only experience (and therefore have knowledge of) their environment through the senses. Therefore, anything that can be known has to be directly observable. The scientific method rests on the requirement that all hypotheses must provide testable predictions that can be verified through independent observations of the phenomenon of interest. If a phenomenon is observable, then it is measurable. If it is not observable, then it is not falsifiable and therefore beyond the remit of scientific enquiry. This philosophy applies to both qualitative and quantitative paradigms, but there are significant differences between their respective epistemologies.

5.1.2. Positivism

In keeping with empiricism, the quantitative approach aims to gather knowledge regarding the aetiology of observed phenomena, and its respective degree of influence over these phenomena. This approach is rooted in positivism, where reality is regarded as independent of the self, and is therefore independent of the subjectivity of the observer. As such, the ontology of positivism is that the environment is able to be reliably observed, categorised, and quantified. Quantitative research is based on objective and dispassionate verification of testable predictions, but, unlike the epistemological roots of empiricism, minimises the role of the researcher's experience and subjectivity. This is often done by using empirically justifiable *a priori* suppositions, methods, and tests, to examine data to eliminate researcher bias. Quantitative approaches also utilise standardised methodologies, instruments, and statistical measurements, which lends a greater ease of replicability and data synthesis (Creswell, 2003). This allows the precise validation of theories through quantitatively testing the reliability of predictions made by theories on the aetiology and processes lying behind complex phenomena (Johnson & Onwuegbuzie, 2004), and, when samples are taken at random, may promote generalisation of findings to populations (Gall, Gall, & Borg, 2003).

5.1.3. Social Constructivism

Rather than supposing the environment is independently observable and measurable, the qualitative approach has its own set of assumptions that are based in social constructivism. The ontology of social constructivism is that reality (at least as far as human psychology is concerned) is not independent of its observer, and therefore cannot truly be known. As such, the experience of reality is regarded as a percept and is idiosyncratic in nature. If so, then social relationships and experience of environments are constructed out of these percepts. The epistemology of this approach is therefore that all knowledge is gathered through a medium that is socially constructed, and thus an emergent property of that exchange. The corollary here is that knowledge gathered from this approach is not characterised by independent observations - it is interpreted through the worldview of the researcher (Tashakkori & Teddlie, 1998). A qualitative researcher must therefore be aware that context is key in applying their findings (Sechrest & Sidani, 1995). So, the supposition that every individual's experience of reality is different means that it is not generalizable, and is thus opposed to the nomothetic stance intrinsic to the quantitative approach (Gall, Gall, & Borg, 2003).

5.1.4. Summary

As is evident, quantitative approaches gather numerical data, and measures of quantity are a universal constant, whereas qualitative approaches gather linguistic data from a language that is socially constructed. So, qualitative approaches too, are not without their limitations. To conduct qualitative research one must then assume that there is a shared meaning between participant and researcher, and this may not always be the case (Gall, Gall & Borg, 2003). To this end, researcher bias may inhibit the investigator from seeing other explanations that do not conform to their view (Krantz, 1995), or be selective in what they choose to report (Tashakkori & Teddlie, 1998). Out of these different epistemologies arises two complementary forms of knowledge. While quantitative describes the strength of outcome, qualitative aims to describe the process leading to an outcome. The process to be qualitatively observed exists in a context that is outside the realm of quantitative enquiry because qualitative philosophies acknowledge the fluctuating nature of reality, attributing to it dynamic systemic interactions that may be lost during the process of quantitative enquiry (Filstead, 1979). Consequently, qualitative methods are well-suited to describe complex phenomena, especially given the natural context and the environmental conditions contributing to the observed phenomenon (Johnson & Onwuegbuzie, 2004). In the context of this thesis, this allows clinician-researchers to understand the needs of their patients within their system, which will also aid the generation of new research questions that will facilitate new studies that may improve quality of life in this population (Tashakkori & Teddlie, 1998).

5.1.5. Pragmatism

Taking advantage of the strengths behind each approach is central to the use of mixed methods (MMR). MMR's foundation lies in the philosophy of pragmatism. In this approach, the ontological and epistemological roots of quantitative and qualitative paradigms are equally valid, and can therefore be synthesised according to the needs of the research question. Researchers are not restricted in their capacity to generate new knowledge by being bound to a particular ontology or epistemology, but rather the choice of research methods (and their synthesis) is dependent on the nature of the research question (Creswell, 2003; Hammond, 2005). This makes use of 'complementarity', where such a synthesis offsets the intrinsic limitations to each approach, by combining their strengths. However, it is likely that the most significant criticism of MMR is that the intrinsic ontological and epistemological differences lead to

unresolved debate regarding whether these data can be truly synthesised, or if it is indeed appropriate to merge data generated from two opposing epistemologies (Smith, 1983; Lincoln & Guba, 1985). Though this is an extreme view, it is supported by the fact that there is no consensus on the most appropriate procedure for mixing paradigms and how to interpret results that conflict during synthesis. This is first assuming that a) the researcher has decided which approach takes priority (qualitative or quantitative), b) whether the investigator is proficient in the methods to be combined, and c) whether MMR/SED is justified. Though the researcher is proficient in clinical case reports and general quantitative methods, the different ontological and epistemological stances inherent to quantitative and qualitative paradigms require a careful and robust approach.

5.1.6. Limitations of the Sequential Explanatory Methodology

The general rationale for employing MMR is that it combines the ontological and epistemological strengths of both approaches, while offsetting the limitations intrinsic to each paradigm. Thus, mixed-methods research provides a pragmatic (but sometimes impractical) solution to a complex problem. Though the practice is acceptable, it is still problematic even for relatively less complex designs which focus on explaining quantitative findings (Greene, Caracelli, & Graham, 1989), maybe because the perceived benefits overshadow the ontological and epistemological conflicts between quantitative and qualitative approaches. It then follows that the criterion of internal validity rests not on whether the two types of data can be linked, but on how, why, and whether it should be done (Miles & Huberman, 1994). According to Bazeley (2004), the validity of mixed-methods research rests on a) an awareness of the intrinsic limitations of each research method before they are integrated into MMR; and b), an appropriate level of generalisation after considering the chosen sample and methodology. As such, the validity of this thesis also rests on the rigour with which these methods are applied and the weighting of the evidence.

In keeping with Bazeley's (2004) criteria, the writer of this thesis provided extensive discussion on the general and specific methodological limitations of each study, followed by discussion on how to improve each study's validity and reliability, and suitably weighted conclusions. However, what has yet to be considered is how each study (in this context) fits into the MMR/SED paradigm. Study 1 (the random-effects meta-analysis), provided a powerful and representative view of the prevalence of CR-PTSD in the literature, but somewhat less conclusive evidence on particular moderators of these broad rates due to variability in data reporting in the included studies. However the findings did reflect what was found in study 2 in terms of sampling (how CR-PTSD was extremely uncommon [<25 CR-PTSD patients sampled over five years in a catchment area of approximately 300,000]), and the lack of power of disease and treatment variables to predict PTSD symptom severity. Study 2 provided evidence on statistical grounds that the differences between the two diagnostic groups were characterised by statistically (and clinically) significant differences in emotional impoverishment even when accounting for variance due to moderators emerging from the literature (mean age). These two studies were employed with the aim of capturing broad differences in the cancer survivor population, then more specific differences in the AD and CR-PTSD sub-populations. This then permitted follow-up explanations for these differences in the same population (and sub-populations) from which Study 2 (and to some extent study 1) were derived. The key strength of the qualitative phase was on moving from large and general, to small and specific samples from the same population, and choosing assessment methods based on the strengths and limitations from previous studies. It should be noted that SED has a particular

strength over other MMR designs as the quantitative emphasis also requires a range of a priori research questions, aims, designs, analysis choices, and a strict protocol though which this initial stage is implemented. This facilitated a robust and logical rationale for the sequential use of these methods that lead to valid conclusions from which the qualitative phase could progress.

However, questions remain on a) how and when the statistical findings in Phase 1 are used to focus follow-up explanations in Phase 2, and b) how these findings are weighted to generate final conclusions. Recall that SED generally provides two different types of rationale for *linking* quantitative results to subsequent qualitative exploration: the *participant selection model* and the *follow-up explanations model*. The two patients in Study 4 were approached subsequent to their referral to the PTSD clinic and were included as a case study if they a) consented to do so, and b) their primary diagnosis was a strong, clinically (and experimentally) valid diagnosis of either CR-PTSD or CR-AD. They were by no means selected based on the findings of Studies 1 & 2, simply because some patients with AD in Study 2 also had very high scores on the EPS-25, and some PTSD patients had very low scores, suggesting that problems processing emotions do not necessarily predict one course of adjustment over another. In this sense, using a participant selection model based on those results would have created a false distinction between the two diagnostic groups where findings at a sample level (clinical v non-clinical problems with emotional impoverishment) are generally true at a patient level. The refusal to select patients on this rationale permitted the exploration of how the patient's emotional processing might also be influenced by factors yet to be investigated in this sample.

Therefore, the follow-up explanations model was used to explore, in terms of process, how problems with emotional impoverishment are implicated in the development of these types of psychopathology. It should be noted that though the findings from the two case studies suggest a link between perceived social support or constraint and the activation of latent emotional processing styles, the role of social support / constraint was not considered prior to the analysis of the case studies. This might suggest that the finding likely *emerged* from the case studies, rather than being inserted into the qualitative data due to the researcher's *a priori* assumptions. This finding was subsequently validated in a new literature search which identified a social-cognitive processing theory of psychological adjustment in the same population (Lepore, 2001).

However in keeping with Bazeley's (2004) criteria, the issue of generalisation remains. Though it was suggested that a larger case series would provide a broader view of psychological presentations, it can be argued that because the cases were not randomly selected from the clinical sample (let alone the population) there is no guarantee that they even remotely represent the population of interest and thus cannot be generalised to the clinical populations of female breast cancer survivors – but generalisation is not the goal. As reflected in the weighting of the case studies, the aim was not to provide definitive answers, but to provide follow-up explanation and thus generate hypotheses for future study to improve the detection and adequate treatment provision for cancer survivors with trauma and stress-related psychopathology (APA, 2013; Kangas, 2013). Clinical observations in naturalistic samples can help generate research questions that are amenable to scientific testing. They can hold great clinical value in terms of their short-term benefits and long-term clinical and service-based implications for quality care, and this serves a vital role in evidence-based practice. In this sense, the major weighting lies first in the results of the meta-analysis, which state that CR-PTSD is uncommon but can and does emerge in the

cancer survivor population and second, that problems experiencing emotions are implicated in trauma and stress-related psychopathology, although this second hypothesis must first be investigated in larger samples and compared to a cancer survivor control group using gold-standard assessments before having the validity of the meta-analysis findings and informing clinical practice.

Finally, there are also practical disadvantages to the MMR/SED approach that may outweigh the potential methodological limitations (Creswell et al., 2003). In the case of this thesis, the qualitative component alone required 18 months for one investigator to collect, transcribe, and analyse, the therapy recordings for two patients over and above that of the quantitative phase, despite the relative lack of weighting in the final synthesis. Thus, the use of MMR/SED, though pragmatic in its use, was quite impractical. Investigator's choosing to replicate this research program might consider using a larger research team.

5.1.7. Discussion

In the case of this thesis, it is the view of this investigator that the use of MMR (specifically, SED), though impractical, was justified for the following reasons: First, systematic reviews, though identifying a range of factors related to PTSD prevalence in cancer survivors, were plagued by extreme heterogeneity in sampling and methodology in the retrieved literature, limiting the degree with which conclusions could be made regarding the traumaticity of cancer, and the factors contributing to its emergence (Gurevich et al., 2002; Kangas et al., 2002; Smith et al., 1999). For this reason, a quantitative synthesis was justified to empirically estimate the mean prevalence of CR-PTSD, and identify study-level moderators that contributed to the fluctuations in prevalence in cancer survivors. Second, as the prevalence of CR-PTSD was low, a more sensitive investigation of the factors contributing to CR-PTSD necessitated a clinical, rather than purely oncological, sample. Using a clinical sample of cancer survivors, the investigator found that PTSD symptom severity was predicted by problems linking and labelling emotions, and covert cognitive avoidance. However, the variance contribution, though significant, was small. Third, this suggested to the investigator that there were dynamic processes related to the use of avoidant strategies that were integral to the development of PTSD, and this necessitated a qualitative investigation. This is because while quantitative methods are excellent for the natural sciences (where phenomena exist separate from the self), such methods have significant limitations for the study factors contributing to the emergence of PTSD in medical settings. As discussed in Phase 2, mental health problems are related to perceptions of the self, and its relationship to the world and others. As such these percepts are idiosyncratic, so mental-health (or lack thereof) is a socially-constructed phenomenon. Furthermore, even if that assertion is debateable, the environment in which mental ill-health is both diagnosed and treated (that is, the therapeutic alliance), is socially-constructed. It is therefore highly appropriate to use research methods that reflect the environment and epistemology from which the concepts of mental health and traumatic stress were originally formed.

The use of the sequential explanatory design required the researcher to learn and implement multiple methods of data collection and analysis, ranging from meta-analysis to cross-sectional/prospective research, and detailed case reports. In terms of the follow-up explanations model, the literature reviews, meta-analysis, and cross-sectional data analysis of the PTSD Clinic revealed the problems discriminating between adjustment disorder and PTSD, and therefore informed our selection criteria of one subsyndromal/adjustment case, and one chronic PTSD case. Contrary to the cancer literature, Phase 1 did

not indicate that age, disease stage, or time since treatment were useful independent predictors of PTSD symptom severity, or diagnosable PTSD in adult breast cancer survivors. However, the results from Study 2 did suggest that those who met caseness on the Davidson's Trauma Scale were characterised by greater rumination and more impoverished emotional experiences. For this reason, the focus of the case reports was on the role of emotional processing strategies in the chronicity of distress. Therefore, the data gathered and analysed from Phase 1 (quantitative) provides a general overview of the clinical problem in diagnosing PTSD and identifying stable factors contributing to its emergence in adult cancer survivors. The data from Phase 2 was used explain the role of key variables from Phase 1 in the course of PTSD symptoms in two adult cancer survivors, and explores the systemic interaction of these variables in a clinical context. Though the aim of this study was to explain the relationship between quantitative variables and vulnerability to PTSD in the adult cancer survivors, the priority was given to the quantitative approach. This was due to the abundance of quantitative data in this thesis versus two clinical case studies, which cannot carry substantial weight, but rather facilitate the generation of new hypotheses for future investigation. Though the evidence is weighted to the results from Phase 1, the findings from Phase 2 will be used as examples by which the clinical implications of Phase 1 can be considered.

5.2. Clinical Implications

The evidence shows it is unlikely that traumatisation and PTSD is the common path for cancer patients. However, Study 1 revealed that a significant minority of cancer survivors will present with clinically significant symptoms or PTSD as a result of the cancer experience. But additional evaluation of Phase 1 suggests that having a patient endorse cancer as traumatic is insufficient on its own to meet Criterion A1. The literature to-date also revealed that diagnosing CR-PTSD is problematic due to the distinctive features of the stress reaction with which cancer patients and survivors present (Bruce, 2006; Kangas et al, 2002; Smith et al., 1999). The accuracy of diagnosing CR-PTSD will have clinical implications for choosing the correct interventions. It is therefore necessary to address these diagnostic issues (Kangas, 2013), and attempt to reconcile them with the understanding of PTSD using the findings presented in this thesis.

5.2.1. DSM-IV PTSD Criterion A: Cancer As A Trauma

The general consensus from early systematic reviews (Bruce, 2006; Kangas et al., 2002; Smith et al., 1999) was that cancer is not a discrete, demarcatable event. There are many stages from detection, to diagnosis, rigorous and often painfully invasive medical treatment, to follow-up appointments at treatment conclusion (Smith et al., 1999). During this period, the stressor can involve a threat to life at the time of diagnosis, and physical integrity through the treatment. As such, cancer is a multiple, interacting, protracted, and cumulative experience (Bruce, 2006; French-Rosas et al., 2011), that resembles both singular (Type I), and repeated (Type II) stressors (Terr, 1991). *Type I* trauma is defined as a "singular shock" (a simple event) from which one can recover. *Type II* trauma refers to repeated episodes. Type II is further sub-categorised into *IIr* and *IInr*. *Type IIr* is when the sufferer experiences repeated psychological shocks, but has sufficient emotional *resources* (both personally, but also within their social system) to cope. *Type IInr* refers to the experience of repeated shocks with little or *no resources* to cope and recover, resulting in the compounding of traumatic experiences, and subsequent vulnerability to

further post-traumatic responses. Though Kangas et al., (2002) acknowledge the evidence that prolonged, repeated, or multiple traumas can exacerbate PTSD symptoms compared to singular events, they (along with other investigators) highlight the significant difficulties in conceptualising cancer as a traumatic stressor, as many of the distinguishing features defy DSM-IV PTSD Criteria (APA, 1994; Smith et al., 1999).

As Smith et al., (1999) stated, the cancer itself is not necessarily an *imminent* threat to life. Though the cancer may be terminal if not treated, intervention at earlier stages of detection can be curative, or at least, extend the life of the patient. Advancements in screening methods and treatments have significantly improved prognosis and reduced mortality, with recent studies showing a 35% drop in breast cancer mortality for UK women aged <50 years (Autier, 2010). For those patients whose cancer may be curable, many do have a degree of control over their treatments. These points can be used to argue that the cancer experience is not a Criterion A stressor for the following reasons: First, PTSD Criterion A1 states that the person has to *witness*, *or be confronted with*, an event that involves actual or threatened death or serious injury (APA, 1994). This suggests imminence to the threat that is uncharacteristic of cancer. Second, PTSD Criterion A2 states that the individual has to react with fear, *helplessness* or horror (APA, 1994). Helplessness suggests a lack of controllability which is not characteristic of cancer treatment. However, these arguments rest on two suppositions: 1) that it is the prospect of death that is traumatic; and 2), that the presentation of treatment choice is synonymous with control as opposed to helplessness.

This is not supported by the data from studies 2 & 4. Of those patients who did have PTSD, the specific events that were endorsed as traumatic were related to the treatments themselves, the pain, and chronic cognitive, physical, and psycho-sexual side-effects that had to be endured in order to survive the illness. Phase 2 presented a Stage II breast cancer survivor (Angela) with chronic CR-PTSD. For Angela, it was not the threat of death that was traumatising, but rather the systematic removal of body parts that defined her femininity, and the associated physical pain, along with the overwhelming pace of the surgeries and treatment (Criterion A1). The additional helplessness and horror she experienced was not related to her apparent choice of intervention. For her, each subsequent surgery was performed to reduce her risk of cancer recurrence - she had no discernible option, and felt helpless as the cancer required her to remove everything that was female (Criterion A2). The picture that developed for Angela was that it was one traumatic stressor after another, culminating in the removal of her ovaries. Believing that they could take no more from her, Angela then began to develop the PTSD syndrome. This resembles a chronic Type II stressor.

Esther also provided a depth of insight into the apparent control over her treatment. Though she recalled being given several choices, she was also made aware which one would give her the best chance of survival, and the least likelihood of recurrence. It was this option that also had the most side-effects, and had the potential to cause serious health problems that in her mind were comparable to the breast cancer itself. Esther felt helpless because either option was going to destroy her body (Criterion A2). In summary, this had very little to do with death. The impression that was given was that the helplessness and horror experienced by these two women was in response to the imminent and unavoidable threat of physical pain and injury.

It is interesting to note that the qualitative data from Studies 2 and 4 is commensurate with the new DSM-5 PTSD Criterion A (APA, 2013), which states that cancer is not considered especially traumatic

unless what is experienced during the course of the illness includes some incidents characterised by other traumatic events. It is also of clinical relevance that neither of the women in Study 4 described the traumas they experienced in concise terms at the time of assessment. The entire cancer experience was deeply overwhelming for both, but the exact nature of the traumas was not revealed until exposure began, and unprocessed memories began to surface that were initially suppressed (either by distraction or numbing) during the disease. The clinical implications here are evident: while it is possible that the already low PTSD prevalence rates are inflated due to lack of differential diagnoses (Kangas, 2013), failure to explore the traumatic aspects of the experience may also result in failure to identify a cancer survivor with PTSD. For some mental health professionals, the mere disclosure of physical/sexual assault, near death experiences (accidents/natural disasters), combat exposure, or prolonged domestic violence, may be sufficient to endorse Criterion A1. But as is evident from this thesis, the level of trauma experienced from the cancer (if any), may not be immediately apparent from having endured the continuum from diagnosis, through to treatment and recovery. For this reason, this investigator suggests that oncology specialists or mental health professionals who encounter cancer survivors presenting with psychological distress related to their cancer, screen for *discrete* traumatic events along the cancer continuum. Information on traumatic experiences may be found in the content of sensory flashbacks, nightmares/distressing dreams, or specific avoidance patterns (e.g. avoiding wards, adverts for cancer charities). Though it is arguable that the cancer experience may consist of discrete traumatic events, there do remain a number of issues in assessing for the PTSD syndrome itself (Criteria B-D, APA, 1994).

5.2.2. DSM-IV PTSD Criterion B – Re-experiencing Symptoms

The common theme underlying the re-experiencing criteria is that they are a cluster of symptoms that manifest as intrusive memories, negative affect (extreme fear), nightmares, and dissociative flashbacks which are directly related to the experience of a traumatic event (Criterion A1) to which the individual reacted with intense fear helplessness or horror (Criterion A2). As such, these intrusive symptoms are qualitatively different from those in the other anxiety disorders, because the fear is anchored in past events, not anticipated future events. Kanags et al., (2002) argue that this presents a problem when assessing Criterion B in cancer patients and survivors, given that much of their intrusions are about future orientated fears regarding their health, or the fear-of-recurrence (FOR). FOR is a state of continued concern reported by cancer survivors, whereby they experience great anxiety about their cancer returning (Hodges & Humphris, 2009). FOR is extremely common among cancer patients, and is also present in some cancer samples that also have PTSD. Mehnert et al., (2009) found that FOR was positively correlated with PTSD symptom severity, supporting previous arguments that cognitive intrusions endorsed in PTSD better reflect future-orientated fears than by past threats being experienced as current. This poses a diagnostic problem: those patients who endorse intrusion symptoms may actually be reporting intrusive anxious cognitions rather than intrusive unprocessed memories. This has been found to inflate PTSD prevalence in cancer samples (Green et al., 1998).

There were some cases in Study 2 where the PTSD syndrome was anchored to FOR on the Davidson's Trauma Scale. But the clinical notes also suggested that in many cases the FOR was not related to fear of death, but rather of having to endure the chemotherapy again. In Phase 2, Angela did admit to a fear of recurrence, but this was in addition to the flashbacks and nightmares related to the chemotherapy and surgery. It was only via careful clinical assessment that the contents of flashbacks were

established and Criterion B was endorsed. This is essential, as previous investigators have stated that establishing PTSD caseness in cancer samples requires information that is not available from screening questionnaires (Shelby et al., 2005). Also, the use of screening tools to provide a tentative diagnosis will encourage improper endorsement of Criterion B because patients/survivors do not have the clinical knowledge to discriminate between intrusive cognitions and intrusive memories.

5.2.3. DSM-IV PTSD Criterion C – Avoidance Strategies

In their seminal review, Kangas et al. (2002) put forward an argument that the avoidance symptoms (Criterion C) are the hardest to endorse in the cancer population. For example, efforts to avoid environmental cues (Criterion C2) may be difficult (e.g. necessary follow-up clinics), and the continued presence of somatic sensations (e.g. side-effects of treatment/surgery), may prevent avoidance of internal reminders (Criterion C1). Also, a diminished interest in activities (Criterion C4) might better reflect the chronic fatigue that is experienced by some chemotherapy patients. The sense of a foreshortened future (e.g. no career or children, or a shorter lifespan; Criterion C7) may also be medically justified on the grounds that 24% of deaths worldwide are cause by cancer (WHO, 2014). Though cancer is curable, there is always a risk of recurrence, and in Angela's case (Study 4), the removal of ovaries meant that she could no longer have children. Also, this foreshortened future may be linked with latent FOR.

However, Study 4 suggested otherwise. Angela's behavioural avoidance patterns involved avoiding external reminders such as TV adverts, and oncology wards (Criterion C2), but most of the avoidance criteria were met through covert cognitive avoidance of thoughts and feelings (C1), feelings of detachment that were reported as a symptom and observed as a sign (C5), and as established much later in the therapy, a restricted range of affect (C7). All of these symptoms were experienced during and after (but not before) the cancer experience, and clinically speaking, had profound repercussions for her adaptation and recovery. This is very similar to Study 2, where the CR-PTSD group was distinguished by greater problems experiencing, linking, and labelling emotions than the non-PTSD group, and this has been observed in other trauma populations (Foa, Riggs, & Gershuny, 1995).

Studies 2 & 4 did suggest that symptoms relating to impoverished experience of emotions may provide a more specific indicator of PTSD in this population, but that these symptoms may also occur alongside clinical depression. In terms of factors that may perpetuate or exacerbate existing traumatic stress, oncology specialists may consider whether the cancer survivor presents with signs of alexithymia (poor labelling and linking of emotions to events), emotional numbness/a generally disengaged demeanour, given that research has shown that failure to experience emotions is highly elevated in PTSD populations (Frewen et al., 2008) and can affect the patient's ability to emotionally engage during assessment and therapy (Gendlin, 1981; Klein et al., 1969). Further information-gathering could ascertain whether this behavioural and cognitive avoidance is linked with strained or unavailable relationships. If so, social-cognitive processing may be a factor in the chronicity of the disturbance. As is evident from Study 2 & 4, the presence of rumination (another form of cognitive avoidance) is a predictor of PTSD symptom severity. But as Study 4 and Lepore (2001) suggest, may also be related to unsupportive interactions and social constraints on talking about the illness. It is possible that persistent ruminations related to the lack of support throughout the cancer will also trigger flashbacks from the trauma and thus perpetuate chronic processing. The presence of rumination, suppression, or chronic emotional numbing, may have significant implications for trauma-focused therapy in cancer settings. Though it can be argued

that the covert cognitive avoidance of unpleasant emotions has a detrimental effect on therapy (Leahy, 2002, 2007), the interventions used to improve engagement with emotions may be rendered less effective if the use of these strategies is triggered by the poor quality of one's support system.

5.2.4. DSM-IV PTSD Criterion D: Hyper-arousal Symptoms

The hyperarousal criteria may also reflect somatic sensations that are related to the cancer treatment and side effects. Arousal symptoms might be better explained by common side-effects of chemotherapy such as 'hot flashes', some of which include sensations of heat, sweating, and palpitations (Lipov et al., 2008; Sturdee, 2008). In addition, difficulty concentrating (Criterion D3) may be incorrectly endorsed due to chemotherapy inducing impairments in memory, processing speed, and executive functions (Thompson, 2011; Wefel et al., 2004), which affects between 16-75% of cancer patients (Argyriou et al., 2011). Kangas et al., (2002) quite rightly suggest that hyperarousal symptoms will be hard to differentially diagnose until the cancer treatment has finished. Refraining from diagnosing CR-PTSD until treatment completion may reduce incorrect symptom endorsement and improve sensitivity to PTSD. The lack of specificity in the hyperarousal criterion is also reflected in the general psychiatric co-morbidity associated with PTSD. Difficulties falling or staying asleep (Criterion D1) or concentrating (Criterion D3) are also symptoms of Major Depression (APA, 1994), which is highly co-morbid with PTSD in cancer populations (Palmer et al., 2004; Shelby et al., 2005), and also co-morbid with CR-PTSD in the second case-study (Study 4). Similarly, other criteria are subject to co-morbid endorsements - the avoidance of social activities characterised by both PTSD (Criterion C2) and depressive presentations. However, the reasons for these endorsements are different. Social isolation in PTSD is to avoid processing of trauma, whereas in depression it is due to lack of motivation, energy, and interest. It is for this reason that assessing clinicians should be aware of the side-effects of cancer treatment in order to perform a differential diagnosis (which in itself can be done by a clinical psychologist). This issue was addressed in Studies 3 & 4 by the joint assessment of an oncology specialist and a clinical psychologist, in which their combined expertise allowed a highly competent and comprehensive assessment of the cancer survivor's psychological needs.

5.2.5. DSM-IV PTSD Criteria E & F: Duration and Disturbance

Given the suppositions that CR-PTSD can be correctly diagnosed, and that the PTSD syndrome is present, there remains the issue of whether the syndrome has been of sufficient duration to warrant a full PTSD diagnosis, or failing that, an adjustment response. However, this first requires the clinician to ascertain when the cancer trauma has ended. This is notoriously difficult to establish. However, the findings in this thesis suggest that it may be feasible to consider post-treatment as post-trauma for the following reasons: 1) aspects of cancer treatment were generally endorsed as traumatic; 2) a peri-treatment PTSD diagnosis is not feasible given the overlap between psychological symptoms and physiological side-effects of cancer treatment; and 3), if PTSD was diagnosed peri-treatment, trauma-focused interventions may be counter-productive given that the patient needs their emotional resources to survive the treatment (c.f. Foa & Rothbaum, 1998). Therefore it may be clinically prudent to use watchful waiting and see how cancer survivors adjust following successful treatment. Follow-up sessions three months post-treatment can be used to screen for PTSD symptoms. During these sessions, it is advised that clinicians be aware of the nature of cancer as a potentially traumatic stressor, and carefully assess for any discrete events occurred during the treatment (that would also present in flashbacks) to warrant a PTSD

diagnosis; and in the absence of these, consider a diagnosis of adjustment disorder if the PTSD syndrome is present without Criterion A endorsement.

If in the majority of cases, cancer survivors only meet criteria for adjustment disorder; this may still not discount the possibility of PTSD development. Research to-date shows that a much larger proportion of cancer survivors present with subsyndromal symptoms (Green et al., 1998; Guglietti et al., 2010; Shelby et al., 2008), and that these symptoms, like PTSD, are associated with considerable psychological distress and functional impairments (Cordova et al., 1995; Shelby et al., 2008). While this too may lead clinicians to initially question the validity of a full PTSD diagnosis, it does not, according to the DSM-5, discount the possibility that these subsyndromal symptoms may be a predictor of future PTSD. New course specifiers state that PTSD may present with delayed expression, where only subsyndromal criteria are met before eventually meeting full criteria at least six months post-trauma (APA, 2013). This new specifier is founded on recent meta-analyses showing that delayed expression of PTSD is preceded by high stress sensitivity, additional life stressors, maladaptation to continued exposure to stress, and the steady accumulation of subsyndromal symptoms from the onset of the stressor (Andrews et al., 2009; Smid et al., 2009). This is of paramount clinical importance, as Study 4 has clearly demonstrated that not only are these features of the cancer experience, but that subsyndromal symptoms are present from very early on in the cancer treatment, fluctuate and persist throughout the course of the disease (Andrykowski et al., 2000), and, in Angela's case (Study 4), may reflect a delayed presentation. Though this presentation may be considered an adjustment response during treatment, this may develop into a PTSD syndrome if those factors prevent the cognitive processing of the illness. It is for this reason that any cancer survivor presenting this way must be given a comprehensive assessment to elucidate any social or cognitive factors that may impair adjustment and processing of the experience of cancer.

5.2.6. Social-Cognitive Processing and Emotion Schemas

Chapter 1.4 described and evaluated the role of schemas in PTSD. Generally speaking, their involvement in the development and maintenance of PTSD is focused on existential beliefs regarding life and justice, how that relates to the safety of the world and the self, and how contradictory or confirmatory schemas affect the assimilation or accommodation of trauma memories. As such, trauma-discordant beliefs such *"The world is safe"* and *"bad things don't happen to good people"* would be shattered and require extensive revision that would be achieved via the emotional processing cycle. The cognitive model also describes that the success of emotional processing is dependent on how trauma survivors *appraise* their distress (Ehlers & Clark, 2000). Those who cognitively appraise the intrusions as a *current threat* are those that systematically avoid processing memories, and thus develop chronic PTSD. As such, trauma-focused therapies are designed to formulate each individual case, and identify blocks to processing to facilitate therapeutic change.

The cognitive approach supposes one's thoughts affect what they feel - therefore, psycho-education about the symptoms of anxiety can improve engagement and thus improve processing. The additional use of Socratic dialogue or behavioural experiments can facilitate testable predictions and valid experiential experiments that serve to modify appraisal-driven emotional reactions to anxiety symptoms. In particular, these beliefs often refer to catastrophic assumptions about the duration of negative feelings, the potential worsening of symptoms, and the effect on the body that are based in simple misunderstandings and misattributions (e.g. *"I am fainting / having a heart attack"*). Addressing these cognitive appraisals is

relatively easy to do in therapy. For example, exposure therapy is used to prove that anxiety does not get worse and perpetuate if it is experienced, but rather reduces in intensity and ameliorates with engagement (Foa & Kozak, 1986). This approach is highly successful in the case of PTSD and enables cognitive change in trauma survivors (Chapter 1.4). Nevertheless, there are some subtle but clinically important differences between addressing fear avoidance due to appraisals about the consequences of fear, and addressing emotion avoidance due to negative emotion schemas.

It is commonly known that CBT is effective because appraisal-driven emotions can be changed by learning adaptive cognitive skills. However, Chapter 1.4. shows that not everyone with PTSD responds to exposure, and as such they do not process their memories. Emotion schemas surrounding the avoidance of emotion may be implicated in this failure because unlike cognitive appraisals, they are very hard to change, altering with steady integration of experience over time (Chapter 1.3.). Emotion schemas have additional qualities over and above the personal consequences of experiencing fear (or emotion in general) in that they reflect beliefs about the experience and expression of emotion in a *social context* (Gottman 1996; 1997; Leahy, 2002; 2007). It has been argued that emotion schemas are formed during early life and predict the social consequences of emotional expression (Baker, 2007). This is an element that is additional to clinical PTSD theories because negative beliefs about the experience of fear are related to the immediate effect of fear on the *self* (e.g. the fear will get worse and continue indefinitely), rather on its effect on their social network. There are additional implications in that unlike affect-laden cognitions (which are readily accessible given exposure to the appropriate triggers), core beliefs (and possibly emotion schemas) may not be consciously retrievable. In the context of diathesis stress models for PTSD (McKeever & Huff, 2003), emotional schemas (and respective emotion avoidance) may serve as a dormant intrapersonal vulnerability which is then activated in response to an interpersonal diathesis (social constraints), following the experience of a traumatic stressor. If so, it may be clinically prudent to screen for negative beliefs regarding the experience and expression of negative emotion in a social context.

5.2.7. CR-PTSD and Co-Morbid Depression:

Finally, there remains the issue of depression in cancer survivors with PTSD. In Chapter 1.5. it was established that major depression was often co-morbid with cancer-related PTSD symptoms. Study 2 supported this finding, as the CR-PTSD group revealed clinically significant differences in depression symptoms compared to the non-PTSD group. In addition, controlling for depression symptoms rendered the remaining differences non-significant, suggesting problems experiencing, linking, and labelling emotions are accounted for by the presence of depression symptoms. However, the frequency of depression symptoms at assessment did not predict the severity of PTSD symptoms at follow-up. Problems experiencing, linking, and labelling emotions at assessment predicted the outcome at follow-up, consistent with findings that the ability to emotionally engage at the beginning of therapy reflects the amount of therapeutic change at discharge (Klein, 1969).

This presents a conundrum for the treatment of PTSD with co-morbid depression because the NICE Guidelines (2005) state that in these circumstances, the PTSD should be treated first, given evidence that depression will ameliorate when PTSD symptoms decline. This may be appropriate when depression is related to the experience of PTSD, but given the relationship between the role of alexithymic (EPS-25 impoverished subscale) and depressive symptoms, the presence of depression may hinder therapeutic

engagement. It is possible that during the process of adjustment, depression developed prior to the PTSD, which may also reflect social constraints about talking about the illness. Though the evidence for this is comparatively anecdotal, it is worthy of note. In Study 4, Angela presented with significant alexithymic symptoms, which appeared related to her early attachment experiences and familial relationship patterns. Angela decided to numb her emotions. Similarly, Angela also has a history of depression, and clinical observation suggested that the combination of social constraints, emotional numbing, and depressive withdrawal served to reduce emotional engagement, which prevented the cognitive processing of the illness. For this reason, Angela received cognitive therapy for depression in conjunction with trauma-focused therapy for PTSD. Much more data needs to be gathered to examine the frequency of this presentation in the cancer population, but it is worth noting that such a presentation may indicate a vulnerability to chronic CR-PTSD.

5.2.8. Trauma and PTSD as a Model of Cancer Survivorship

Though this thesis presents strong evidence for the emergence of CR-PTSD, there remains the issue of whether the cancer experience is best described in a trauma framework – a question that has far-reaching implications for the assessment and treatment of psychopathology in survivors. As mentioned before (Chapter 1.5), the DSM-IV (APA, 1994) included life-threatening illness as a traumatic event, where the experience of cancer (from diagnosis to recovery) would have been sufficient to fulfil criterion A. However, the release of the DSM-5 presents a reformed trauma criterion, where "…a life-threatening illness or debilitating medical condition is not necessarily considered a traumatic event. Medical incidents that qualify as traumatic involve sudden catastrophic events" (APA, 2013, p.274). Therefore, the cancer continuum, as distressing as it may be, is no longer assumed as traumatic unless something horrific and unforeseen happens during the course of the disease. Adjustment disorders are now presented as the most appropriate diagnosis given that they are common reactions to medical illness and by extension are likely to be the dominant presentation in the majority of cancer survivors (APA, 2013).

These changes reflect the view that a trauma framework (and thus PTSD) does not adequately represent the distress experienced throughout cancer (Green et al., 1998; Palmer et al., 2004), in favour of longer-term psychological maladjustment. However, to conceptualise cancer survivorship using one recovery model (be it trauma-focused or otherwise) is to assume a homogeneity to the experience that does not exist in practice. The central issue is not which of these two trauma / stress-related disorders (or any one disorder) better represent cancer-related distress, but, as Kangas (2013) rightly suggests, whether differential diagnosis will aid in identifying cancer survivors who are experiencing chronic trauma and stress-related psychopathology. If so, patients who are incorrectly identified as having AD when they have CR-PTSD will not receive the NICE-recommended treatment (NICE, 2005). Similarly, patients incorrectly diagnosed with CR-PTSD (rather than AD), will receive inappropriate trauma-focused treatment, rather than other supportive approaches better suited to their psychological condition. Either of these unwelcome outcomes points to a protracted clinical and economic burden.

Though this discussion presents a range of indicators to aid in differential diagnosis, two issues remain: First, if the cancer experience is generally non-traumatic, then the fact that CR-PTSD can and does emerge in a significant minority of survivors suggests that factors other than diagnosis and treatment may exert a larger effect on coping and the cognitive processing of the illness. Second, these factors may

be implicated in course of adjustment towards AD or CR-PTSD, and as such, may provide predictive value, aiding in the early screening of patients vulnerable to trauma and stress-related psychopathology.

5.3. Service-Based Implications

Though this thesis has made some recommendations regarding the identification and assessment of survivors with trauma and stress-related psychopathology, there remains the issue of how these new guidelines will be applied in practice. According to the Department of Health's Cancer Reform Strategy (DoH, 2007), psychological assessment and support is part of routine post-treatment follow-up, but in practice these consultations are often conducted by relatively inexperienced doctors who are not familiar with the patient, and there is little time to assess the psychological needs of the patient given the immediate medical concerns. As is already established, the psychological sequelae of cancer survivorship are of considerable complexity. And in potential cases of CR-PTSD or CR-AD, require specialist knowledge and assessment. This is what the PTSD Clinic at Royal Bournemouth Hospital (RBH), Dorset, UK, aimed to achieve.

The Clinic ran an equitable service for those who were suffering from mental health difficulties related to surviving cancer. It was co-ordinated by two specialists, and ran for six hours a week with a very short waiting list. During this period, 63 patients were admitted into the clinic and were offered a range of therapeutic interventions appropriate to their psychological needs, ranging from counselling, prolonged exposure, to full trauma-focused therapy (NICE, 2005). The referral stream was manageable, and culminated in a small but significant caseload. The cancer sample (n=58), gathered over five years, reflects the uncommon, but nevertheless impairing, nature of CR-PTSD and CR-AD. The clinic initially received referrals from GPs and oncology specialists in the hospital's catchment area (population 550, 000), but the investigator also endeavoured to create care pathways with local NHS Primary Care Adult Mental Health services. This was because adults presenting to their GP with depression and anxiety symptoms were duly signposted to mental health services. Given the Clinic's oncology specialism and NICE-recommended treatment, the clinical lead for Dorset's Primary Care Adult Mental Health endorsed a collaborative care approach, by referring eligible cancer survivors to the PTSD Clinic, while continuing to treat pre-existing and co-morbid depression.

Collaborative care may be a cost-effective method of managing the support of this population. According to Huffman et al., (2014), collaborative care models for mental health problems in medical contexts have proven cheap, sustainable, and consistently improve the clinical outcomes for patients in research and clinical practice. In addition, these programs improve the experience of the patient, and generally lower the costs of healthcare. Collaborative care interventions have several components. First they routinely use validated screening questionnaires as a cheap, quick and effective initial assessment strategy. Second, patients who screen positive are referred for a clinical assessment. Third, if they meet eligibility criteria for the collaborative care program, care managers/nursed can monitor symptoms over time using screening questionnaires. Fourth, nurses can also provide psycho-education and evidence based psychotherapy to patients, and recommend pharmacotherapy to primary care providers. Finally, these programs will include stepped care recommendations from a clinical lead who reviews each case with the respective care manager/nurse. Thus far, collaborative care interventions for depression in cancer patients have been showed to be effective cheap, acceptable, and feasible (Strong et al., 2008).

It may be prudent to develop a similar model of care for cancer survivors presenting with trauma / stress-related disorders. In this model, it is likely that oncology nurses and specialists will be the first point of clinical contact for those presenting with AD/PTSD symptoms related to cancer (c.f. DoH, 2007), and can provide initial psychological support (Kwekkeboom & Seng, 2002), although referral to a clinical psychologist may be warranted (Thompson, 2011). These referrals and initial assessments were based on the breadth of its care pathways and the three levels of screening for eligibility. The first step was using flashbacks and nightmares from PTSD Criterion B (APA, 2000). The second step was using the Davidson's Trauma Scale to identify those with clinically significant symptoms, and the third was the Emotional Processing Scale to identify cognitive avoidance strategies. Finally, there was a full clinical interview with the consultant clinical psychologist.

The results from Study 2 showed that these inclusion criteria were highly effective at screening in those with CR-AD and CR-PTSD. This is clinically important as the literature shows that even those who do not have CR-PTSD but suffer from subsyndromal symptoms (CR-AD) are still functionally impaired (Chapter 1.5). Furthermore, the EPS-25 was able to provide additional clinical data to distinguish these diagnostic groups. Study 2 presented the positive and negative predictive values of both the Davidson' and the EPS-25 to correctly categorise CR-PTSD and SS-PTSD/CR-AD groups, and the EPS-25 had superior PPV and NPV's. This is interesting, as PPV and NPV are largely affected by prevalence. When the prevalence of a condition or disease is low (as it is for this population), PV fluctuates widely. But, Study 2 used selective criteria to improve sensitivity and specificity to CR-PTSD. As such, the prevalence of CR-PTSD in this sample was extremely high, which enabled a higher PPV/NPV. Therefore the additional use of the EPS-25 impoverished subscale may enable more effective screening given that problems experiencing, linking, and labelling emotions differentiated the diagnostic groups – although further research with larger samples and a psychologically healthy control group will be required to test this hypothesis.

This screening procedure may prove effective at reducing workload, but also for screening in all those who do have the condition. Given this information it is likely that NHS oncology departments could provide a small, equitable, and specialised, psychology service that is also feasible and sustainable. In order for individual trusts to implement this, there needs to be a financial assessment to enable the employment of staff, funds for their training and education, clinic space, support services and administrative support. The RBH PTSD Clinic ran for one day a week, treating 3 patients, requiring 1 hour per patient, although with a six-eight week waiting-list. A two-day clinic would reduce the waiting list and create extra time for staff training. Based on the costs for a one-day clinic at RBH, the projected annual costs for a two day clinic would be as follows: First, a consultant clinical psychologist would require £25,000, and a nurse consultant in oncology would require an additional £5061.37 (based on 20.0848 per hour x 6 a week x 42 weeks). Second, the support staff would fall into two categories a) secretarial support, which would cost £514 (£10.28 per hour x 1 hour per week x 50 weeks); and b) clinic management & notes retrieval, which would cost £1,656 (Band 2 @ £7.36 per hour x 45mins per patient x 6 patients a week x 50 weeks). In total, this would require an annual funding of £31,771.37 to run the service at 2 sessions per week with an average of six patients per week. This may suggest a manageable, cost-effective, service that is able to screen, assess, and treat mental health problems in cancer survivors.

5.4. Improving Reporting Standards

Given the clinical and service-based implications of these findings, this section will present a range of strategies and methods that can be used to improve the understanding, prediction, assessment, and treatment, of cancer survivors with trauma/stress-related presentations to improve research and thus inform evidence-based practice. First, Study 1 demonstrated the marked between-study heterogeneity of the CR-PTSD literature, and how variations on reporting styles and standards may have prevented a better-quality synthesis of data that could contribute to knowledge of variables that are associated with CR-PTSD. Future meta-analyses of data from this literature may benefit from the following recommendations. In order to assess the risk of PTSD in this population, it may be necessary to report, or make readily available, the raw data on PTSD event rates by a) disease stage b) cancer type, c) treatment regimens, d) any traumatic events during the course of the disease, e) age at diagnosis, and f) report the mean time of assessment in relation to time post diagnosis *and* time post treatment. It may also be prudent to establish a committee that can draw a consensus on reporting standards for the CR-PTSD literature. This strategy has proven beneficial for standardising the conduct and reporting of systematic reviews and meta-analyses (PRISMA; Moher et al., 2009), case studies (CARE; Gagnier et al., 2013), and may standardise reporting sufficiently to improve the sensitivity of meta-regression of study-level moderators and risk factors. This will support stronger systematic reviews and meta-analyses in the future that may provide additional evidence for the factors implicated in the development and maintenance of CR-PTSD. In the meantime, this thesis presents broad clinical and service-based implications for the screening, diagnosis, and treatment of trauma and stressed-related disorders. But there does remain the issue of how to effectively screen for patients who may be vulnerable to CR-PTSD rather than AD.

5.5. Future Research

5.5.1. Towards a Systemic Diathesis-Stress Model for CR-PTSD and AD.

The clinical and service-based implications point to new research avenues on the course of adjustment in cancer. It has been argued that 'less traumatic' stressors can be expected to trigger PTSD in vulnerable individuals who have limited resources to manage their response to stress (Marshall et al., 2008), and that the apparent insufficiency of some events to meet PTSD criterion A may reflect a lack of consideration for the interactive effect of personal stress reactivity (King et al., 1998; King et al., 1999), and also the effect of allostatic load on the strength of the stressor (Brewin et al., 2009).

There are substantial data on the role of psychiatric history, and demographic, and disease variables in PTSD prevalence (Kangas et al., 2002), but much less information on what variables influence coping during the diagnosis and treatment of the disease. This thesis has presented evidence for the role of social-cognitive variables in this population. For example, social support and/or constraints on talking about the illness (Andrykowski & Cordova, 1998; Cordova et al., 2007; Green et al., 2000; Jacobsen et al., 2002; Kornblith et al., 2003; Lepore & Helgeson, 1998; Manne, 1999; Widows et al., 2000), emotion regulation strategies such as dissociation (Kangas et al., 2005), emotional suppression and avoidance (Amir & Ramati, 2002; Hampton & Frombach, 2000), and rumination (Chan et al., 2011). These have all been identified as possible predictors and maintainers of PTSD symptoms. Most recently, emotional processing styles have been implicated in the development of post-natal depression (Wilkins et al., 2009), and now PTSD (Baker, 2010; Baker et al., 2013).

Given the protracted nature of the cancer experience, it is possible that social/systemic variables and their relationship to beliefs about the experiencing and expressing of emotions may influence their processing over the course of disease, and may be implicated in the emergence of PTSD in this population. Therefore, the CR-PTSD literature and this thesis suggest that additional investigation is warranted to explore the role of emotion schemas, coping strategies, and the effectiveness of the patient's support network on psychological adjustment, and how these factors may interact to predict the development and maintenance of CR-PTSD over and above AD. A series of investigations may lead to a preliminary vulnerability model for CR-PTSD. However, this thesis itself does not provide sufficient data to warrant strong conclusions on the general role of social-cognitive factors in the development and maintenance of CR-PTSD and AD. But, the quantitative weighting of the sequential explanatory design does provide data that significantly contributes to knowledge which can be used to propose a new investigation.

5.5.2. A New Research Program

In order to achieve these new research aims, investigators may need to conduct a longitudinal study that collects quantitative data at a series of time points from diagnosis though to treatment and follow-up during the first few years after treatment conclusion (c.f. Lepore et al., 1996 methodology). This thesis provides some evidence for the following factors: 1) specific cancer experiences endorsed as traumatic; 2), beliefs about emotional experience and expression; 3) problems experiencing, linking, and labelling, emotion; and 4), social constraints on talking about the illness (in the context of spousal, familial, and platonic, relationships). In order to evaluate the role of these variables, a linear regression model with three predictors (social constraints, problems linking and labelling emotions, and negative emotion schemas), combined with acceptable Type I [α =.05], and Type II [β = .95] error rates, and an expected moderate effect size [F^2 = .15], would require a minimum sample size of n = 120. Additionally, a binary logistic regression may be developed using these variables and provide a testable framework for predicting those most vulnerable to future CR-PTSD or AD, compared to cancer survivors with no clinically-significant psychopathology.

However, such a study would require a strong *a priori* rationale, and a large evenly-distributed sample of cancer survivors divided into CR-PTSD, AD, and no trauma/stress-related disorder groups. This presents future investigators with a problem: Study 1 identified that the prevalence of CR-PTSD was generally low, where approximately 1/17 survivors will present with current CR-PTSD. In order to achieve acceptable statistical power [β = .80], a CR-PTSD group of n = 60 (with the additional n = 60 in the AD group, and another 60 in the non-trauma/stress group) would require the screening of at least n = 1020 cancer survivors just to identify those with CR-PTSD– not accounting for attrition. Such a proposal will require new sampling methods on the grounds that a single research centre may have insufficient resources (e.g. funds, clinical psychologists) to assess this volume of patients in a realistic time-frame. However, a multicentre approach conducted in separate oncology services (either in the UK or trans-nationally) would reduce the clinical load, and the time-frame required to track patients through their treatment and follow-up.

The successful development of a preliminary vulnerability model may provide the empirical basis for the development of a screening tool which, like most screening questionnaires, would be quick, cost-effective, and easy to administer either as a self-report questionnaire, or administered by an oncology

specialist. If the tool was clinician-administered, it may improve the sensitivity and specificity of detection, and would only require minimal training of oncology staff. Such a tool may be able to assess vulnerability to trauma and stress-related disorders on a number of levels throughout the illness and flag need for appropriate future intervention. Finally, in keeping with the service-based implications, these multi-centre studies could also refer those with CR-PTSD or AD into a randomized clinical trial that assesses the effectiveness of standard trauma-focused therapies for PTSD (NICE, 2005) compared to EPT (Baker et al., 2013). This would present data regarding the relative benefits of emotion preparation stages for those with chronic psychopathology.

Chapter 6 – Conclusions

The main aim of this study was to address the clinical/diagnostic, service, and research-based, issues in the diagnosis and treatment of cancer-related PTSD (CR-PTSD), before beginning a clinical trial of trauma-focused treatment in cancer populations. A legitimate clinical trial of a trauma-focused therapy is contingent upon a) experiencing a trauma (something atypical of cancer), and b) presenting with full symptom criteria for PTSD (which is uncertain given the overlap with treatment side-effects). A narrative review, meta-analysis, cross-sectional analysis of a mental health sample of cancer survivors, and clinical case studies of breast cancer survivors with adjustment disorder (AD) and CR-PTSD, elucidated a number of issues regarding the diagnosis and treatment of cancer survivors, and also practical issues in study implementation.

This thesis has made several new contributions to our existing knowledge of trauma. Firstly, it has established the prevalence of CR-PTSD (as diagnosed by clinical interviews), and clinically-significant symptoms, in an attempt to establish the amount of CR-PTSD that can be expected to present at an oncology service. The prevalence was sufficiently low to suggest that traumatisation was uncommon. This revealed it was unfeasible to conduct a single-centre clinical trial for CR-PTSD given that a very large number of cancer survivors would have to be screened and assessed just to achieve a sufficient statistical power. Similarly, the sampling methods used in the literature yielded very small rates of CR-PTSD, and as shown in Study 1, it was not possible to conduct powerful analyses in order to detect factors related to this diagnosis. This necessitated a new sampling method to enable examination of variables contributing to CR-PTSD over and above sub-clinical presentations. Secondly, Study 2 provided evidence that CR-PTSD was distinguished from AD by above-threshold emotional avoidance and numbing symptoms (EPS-25 impoverished subscale), whereas the AD group was characterised by sub-threshold scores. This is clinically relevant, given that the EPS-25 scoring system is calibrated to distinguish between psychologically healthy and disordered samples. As such, clinically significant symptoms of emotion avoidance and alexithymic symptoms may be the factors that distinguish the similar presentations of CR-PTSD and AD in cancer survivors.

This evidence is compelling, and there is a great need for this study to be replicated in other samples. If covert emotion avoidance is found to be a reliable and distinctive feature of CR-PTSD over and above AD, then short questionnaires may be devised that inherit the excellent sensitivity of PTSD screeners (e.g.

PCL-C, PDS, and DTS), and the promising specificity of the EPS-25, to separate statistically normal emotion avoidance from clinically-significant avoidance. Not only will this improve sensitivity to trauma/stress-related symptoms (PTSD & AD), but it will also add another level of screening that may aid in differential diagnosis - and hence triage - for post-treatment psychological intervention. These findings may also be taken further: if pathological emotion avoidance distinguishes these disorders, then pre-existent emotionally-avoidant traits may increase the risk of developing CR-PTSD after traumatic medical events within the course of the disease. In this study, emotion avoidance and the associated strategies (suppression, rumination, and dissociation) only accounted for a significant minority of variance in the severity of symptoms, which suggests that additional factors may influence the course of adjustment throughout the cancer treatment.

Arguably the next factor for study is that of social support, or social constraints on talking about cancer. This variable has been identified as a powerful and reliable predictor across trauma populations, but is also especially implicated in adjustment to cancer due to its integral role in helping the patient cope with the illness. Study 4 suggested the hypothesis that social constraints may interact with covert emotion avoidance. This covert emotion avoidance, which may reveal itself on the EPS-25 subscales, may be rooted in emotion schemas related to the consequences of experiencing and expressing emotions in a social context. For example, social constraint (from family and friends) and reactive emotion avoidance (from the cancer survivor), may impair the cognitive processing of the illness, and also prevent the cancer survivor from replenishing the psychological resources required to cope. The extent to which this leads to the development and maintenance of PTSD is beyond the scope of this study.

It is possible that this hypothesis has even wider implications beyond that of cancer survivors. This study has demonstrated that cancer is not a typical, short-lived, delineated traumatic event; rather, it is a long-term episode of extreme stress, in which coping responses, and environmental or social factors, create additional stress with which some cancer patients are insufficiently-resourced to cope. As such, cancer may provide new knowledge about what makes an experience traumatic. Diathesis stress models typically cite intrapersonal (e.g. PTSD Criterion A2; APA, 2000) and environmental diatheses (PTSD Criterion A1; APA, 2000) as sufficient to define traumatisation. However, one could argue that stress is on a continuum, where extreme stress is where the environmental demand is too great for one's psychological resources to overcome. Therefore, trauma (arising from extreme stress), may also be contingent on resource allocation. In this sense, cancer, though extremely stressful, may not be typically traumatic, but may be experienced as such if cognitive (emotion avoidance), environmental (cancer stressor), and systemic diatheses (social constraint) interact to prevent resource replenishment and normal cognitive processing. Cancer-related trauma may be a useful avenue for the study of allostatic load and its implications for traumatisation across a range of complex, protracted events – including military combat exposure, and domestic abuse scenarios. But given that extreme stressors are inherently unpredictable and unethical to control or manipulate, research has focused on populations where traumatisation is predictable and likely (e.g. military combat). It may be prudent to continue this research in the cancer population, given that it is a predictable stressor that is amenable to longitudinal research. This may eventually facilitate a preliminary, systemic, and integrative, vulnerability model for trauma and stress-related disorders – one that can be applied to more than one population.

Continued research into CR-PTSD and CR-AD will certainly advance the psychological aftercare of cancer patients, and facilitate their adjustment into survivorship. New post-cancer cognitive-behavioural approaches that focus on teaching healthy ways to regulate and process emotion may prove beneficial to cancer survivors (Baker et al., 2013). Maybe those who are cancer-free can then enter into this survivorship not just with renewed physical health, but with a renewed emotional wellness that stems from post-traumatic growth – the goal of emotional processing.

Chapter 7 – References

Abbey, G., Thompson, S.B.N., Hickish, T., & Heathcote, D. (2014). A meta-analysis of prevalence rates and moderating factors for cancer-related post-traumatic stress disorder. *Psycho-Oncology,* doi:10.1002/pon.3654.

Adams, R. E. & Boscarino, J. A. (2006). Predictors of PTSD and delayed PTSD after disaster: The impact of exposure and psychosocial resources. *Journal of Nervous and Mental Disease, 194*, 485–493.

Aldao, A. & Nolen-Hoeksema, S. (2010). Specificity of cognitive emotion regulation strategies: a transdiagnostic examination. *Behaviour Research & Therapy, 48* (10), 974-983.

Aldao, A., Nolen-Hoeksema, S., & Schweizer, S. (2010). Emotion-regulation strategies across psychopathology: a meta-analytic review. *Clinical Psychology Review*, 30 (2), 217-237.

Alter, C.L., Pelcovitz, D., Axelrod, A., Goldenberg, B., Harris, H., Meyers, B., Grobois, B., Mandel, F., Septimus, A., & Kaplan, S. (1996). Identification of PTSD in cancer survivors. *Psychosomatics, 37*, 137 – 143.

American Psychiatric Association. (1986). *Diagnostic and statistical manual of mental disorders (3th ed.)* Washington, DC: Author.

American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders (4th ed.)* Washington, DC: Author.

American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders (4th ed., text rev.)*. Washington, DC: Author.

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Washington, DC: Author.

Amir, M. & Ramati, A. (2002). Post-traumatic symptoms, emotional distress, and quality of life in long-term survivors of breast cancer: a preliminary research. *Anxiety Disorders, 16*, 191-206.

Andrews, B., Brewin, C.R., Philpott, R., & Stewart, L. (2007). Delayed-onset posttraumatic stress disorder: a systematic review of the evidence. *American Journal of Psychiatry, 164* (9), 1319-1326.

Andrews, B., Brewin, C. R., Rose, S., & Kirk, M. (2000). Predicting PTSD symptoms in victims of violent crime: The role of shame, anger and child abuse. *Journal of Abnormal Psychology, 109*, 69-73.

Andrews, B., Brewin, C.R., Stewart, L., Philpott, R., & Hejdenberg, J. (2009). Comparison of immediate-onset and delayed-onset posttraumatic stress disorder in military veterans. Journal of Abnormal Psychology, 118, 767-777.

Andrykowski, M. A., & Cordova, M. J. (1998). Factors associated with PTSD symptoms following treatment for breast cancer: test of the Andersen model. *Journal of Traumatic Stress, 11*, 189–203.

Andrykowski, M.A., Cordova, M.J., McGrath, P.C., Sloan, D.A., & Kenady, D.E. (2000). Stability and change in posttraumatic stress disorder symptoms following breast cancer treatment: a 1-year follow-up. *Psycho-Oncology, 9*, 69-78.

Andrykowski, M.A., Cordova, M.J., Studts, J.L., & Miller, T.W. (1998). Diagnosis of posttraumatic stress disorder following treatment for breast cancer. *Journal of Consulting and Clinical Psychology, 66*, 586-590.

Andrykowski, M. A., Cordova, M. J., Studts, J. L., & Miller, T. W. (1998). Posttraumatic Stress Disorder after treatment for breast cancer: prevalence of diagnosis and use of the PTSD Checklist—Civilian Version (PCL-C) as a screening instrument. *Journal of Consulting & Clinical Psychology, 66*, 586–590.

Argyriou, A.A., Assimakoloulos, K., Iconomou, G., Giannakopoulou, F., Kalofonos, H.P. (2011). Either called "chemobrain" or "chemofog", the long-term chemotherapy-induced cognitive decline in cancer survivors is real. *Journal of Pain and Symptom Management, 41* (1), 126-139.

Atkinson, P. & Delamont, S. (2006). In the roiling smoke: Qualitative inquiry and contested fields. *International Journal of Qualitative Studies in Education, 19* (6), 747-755.

Autier, P., Boniol, M., La Vecchia, C., Vatten, L., Gavin, A., Héry, C., & Heanue, M. (2010). Disparities in breast cancer mortality trends between 30 European countries: retrospective trend analysis of WHO mortality database. *British Medical Journal, 341*: c3620.

Baker, R. (2007). *Emotional processing: Healing through feeling.* Oxford: Lion Hudson.

Baker, R. (2010). *Understanding Trauma: How to overcome Post-Traumatic Stress.* Lion Hudson: Oxford.

Baker, R. (2011). *Understanding panic attacks and overcoming fear.* (3rd ed). Oxford: Lion Hudson.

Baker, R., Allen, H., Gibson, S., Newth, J. & Baker, E. (1998). Evaluation of a primary care counselling service in Dorset. *British Journal of General Practice, 48*, 1049-1053.

Baker, R., Gale, L., Abbey, G., & Thomas, S. (2013). Emotional processing therapy for post-traumatic stress disorder. *Counselling Psychology Quarterly*, *26*, 362-385.

Baker, R., Holloway, J., Thomas, P.W., Thomas, S., & Owens, M. (2004). Emotional processing and panic. *Behaviour Research & Therapy, 42*, 1271-1287.

Baker, R., Owens, M., Thomas, S., Whittlesea, A., Abbey, G., Gower, P., Tomsular, L., Corrigan, E., & Thomas, P. (2012). Does CBT facilitate emotional processing? *Behavioural and Cognitive Psychotherapy, 40*, 19-37.

Baker, R., Thomas, S., Thomas, P.W., Gower, P., Santonastaso, M., & Whittlesea, A. (2010). The emotional processing scale: scale refinement and abridgement (EPS-25). *Journal of Psychosomatic Research, 68* (1), 83-88.

Baker, R., Thomas, P.W., Thomas, S., Santonastaso, M. & Corrigan, E. (2014). *The Emotional Processing Scale.* Hogrefe: Oxford.

Baron, R.M., Kenny, D.A. (1986). The moderator-mediator variable distinction in social psychological research: conceptual, strategic, and statistical considerations. *Journal of Personality & Social Psychology, 51,* 1173–1182.

Bartlett, F. (1932). *Remembering: A study in Experimental and Social Psychology.* Cambridge: Cambridge University Press.

Bazeley, P. (2004). Issues in Mixing Qualitative and Quantitative Approaches to Research. In R. Buber, J. Gadner, & L. Richards (Eds). *Applying qualitative methods to marketing management research.* UK: Palgrave Macmillan, pp141-156.

Beck, J. (1995). *Cognitive Therapy: Basics and Beyond.* New York: Guilford Press.

Beck, A.T., Rush, A.J., Shaw, B.F., & Emery, G. (1979). *Cognitive Therapy of Depression*. New York: The Guilford Press.

Bedford, A. & Foulds, G. A. (1978). *Manual of the Delusions-Symptoms-States Inventory (DSSI).* Windsor: NFER-Nelson.

Behar, E., Dimarco, I.D., Hekler, E.B., Mohlman, J., Staples, A.M. (2009). Current theoretical models of generalized anxiety disorder (GAD): conceptual review and treatment implications. *Journal of Anxiety Disorders, 23*, 1011-1023.

Berking, M., Wupperman, P., Reichhardt, A., Pejic, T., Dippel, A., & Znoj, H. (2008). Emotion-regulation skills as a treatment target in psychotherapy. *Behaviour Research & Therapy, 46*, 1230-1237.

Blake, D.D., Weathers, F.W., Nagy, L.M., Kaloupek, D.G., Gusman, F.D., Charney, D.S., Keane, T.M. (1995). The development of a clinician-administered PTSD scale. *Journal of Traumatic Stress, 8,* 75-90.

Blanchard, E.B., Jones-Alexander, J., Bucklet, T.C., & Forneris, C.A. (1996). Psychometric properties of the PTSD Checklist (PCL). *Behavioral Research & Therapy, 34*, 669-673.

Boden, M.T., Westermann, S., McRae, K., Kuo, J., Alvarez, J., Kulkarni, M.R., Gross, J.J., & Bonn-Miller, M.O. (2013). Emotion Regulation and Posttraumatic Stress Disorder: A Prospective Investigation. *Journal of Social and Clinical Psychology, 32*, (3), 296-314.

Bomyea, J., Risbrough, V., & Lang, A.J. (2012). A consideration of select pre-trauma factors as key vulnerabilities in PTSD. *Clinical Psychology Review, 32* (7), 630-641.

Borenstein, M., Hedges, L.V., Higgins, J.P.T., & Rothstein, H.R. (2009). Introduction to Meta-Analysis. John Wiley & Sons Ltd.

Borkovec, T. D. (1994). The nature, functions, and origins of worry. In: G. Davey & F. Tallis (Eds.), *Worrying: perspectives on theory assessment and treatment* (pp. 5–33). Sussex, England: Wiley & Sons.

Borkovec, T. D., & Newman, M. G. (1998). Worry and generalized anxiety disorder. In A. S. Bellack & M. Hersen (Series Eds.), & P. Salkovskis (Ed.), *Comprehensive clinical psychology: Vol. 6. Adults: Clinical formulation and treatment.* (p. 439 – 459). Oxford, United Kingdom: Pergamon Press.

Borkovec, T.D., & Roemer, L. (1995). Perceived functions of worry among generalized anxiety disorder subjects: Distraction from more emotionally distressing topics? *Journal of Behavior Therapy and Experimental Psychiatry, 26*, 25–30.

Borkovec, T.D., & Ruscio, A.M. (2001). Psychotherapy for generalized anxiety disorder. *Journal of Clinical Psychiatry, Perception of emotion in self and others 62*, 37–45.

Boyer, B.A., Bubel, D., Jacobs, S.R., Knolls, M.L., Harwell, V.D., Goscicka, M. & Keegan, A. (2002). Post-traumatic stress in women with breast cancer and their daughters. *American Journal of*

Family Therapy, 30, 323-338.

Brady, K.T., Killeen, T.K., Brewerton, T., & Lucerini, S. (2000). Comorbidity of psychiatric disorders and posttraumatic stress disorder. *Journal of Clinical Psychiatry, 61*, 22-32.

Breslau, N., Davis, G.C., Andreski, P., & Peterson, E. (1991). Traumatic events and posttraumatic stress disorder in an urban population of young adults. *Archives of General Psychiatry, 48*, 216-222.

Breslau, N,, Kessler, R., Chilcoat, H., Schultz, L., Davis, G., & Andreski, P. (1998). Trauma and posttraumatic stress disorder in the community: The 1996 Detroit Area Survey of Trauma. *Arch Gen Psychiatry, 55*, 626-32.

Breslau, N. & Kessler, R. (2001). The stressor criterion in DSM-IV posttraumatic stress disorder: An empirical investigation. *Biological Psychiatry, 50* (9), 699-704.

Brewin, C.R., Andrews, B., & Valentine, J.D. (2000). Meta-analysis of risk factors for posttraumatic stress disorder in trauma-exposed adults. *Journal of Consulting and Clinical Psychology, 68* (5), 748-766.

Brewin, C.R., Dalgleish, T., & Joseph, S. (1996). A dual representation theory of post-traumatic stress disorder. *Psychological Review, 103*, 670–686.

Brewin, C.R. & Holmes, E.A. (2003). Psychological theories of posttraumatic stress disorder. *Clinical Psychology Review, 23*, 339-376.

Brewin, C.R., Lanius, R.A., Novac, A., Schnyder, U., & Galea, S. (2009). Reformulating PTSD for DSM-V: Life after criterion A. *Journal of Traumatic Stress*, 22, 366-373.

British Psychosocial Oncology Society (2014), Abstracts for the 2014 annual conference: the emotional impact of cancer. *Psycho-Oncology, 23*, 1–16. doi: 10.1111/pon.3495

Brown, T.A., Barlow, D.H., & Liebowitz, M.R. (1994). The empirical basis of generalized anxiety disorder. *American Journal of Psychiatry, 151*, 1272–1280.

Bruce, M. (2006). A systematic and conceptual review of posttraumatic stress in childhood cancer survivors and their parents. *Clinical Psychology Review, 26*, 233-256.

Bryant, R.A. & Panasetis, P. (2001). Panic symptoms during trauma and acute stress disorder. *Behavior Research and Therapy, 39*, 961-966.

Bucci, W. (1997). *Psychoanalysis and Cognitive Science: A Multiple Code Theory.* Guildford Press: New York.

Bucci, W. (2001). Pathways of emotional communication. *Psychoanalytic Enquiry, 21*, 40-70.

Buckley, T.C., Blanchard, E.B., & Neill, W.T. (2000). Information processing and PTSD: A review of the empirical literature. *Clinical Psychology Review, 20*, 1041-1065.

Butler, L.D., Koopman, C., Classen, C., & Spiegel, D. (1999). Traumatic stress, life events, and emotional support in women with metastatic breast cancer: cancer-related traumatic stress symptoms associated with past and current stressors. *Health Psychology, 18* (6), 555-560.

Cancer Research UK. *Lifetime Risk Of Cancer. In Cancer Stats: Cancer Statistics for the UK*. Retrieved September 27, 2013, from http://www.cancerresearchuk.org/cancer/info/cancerstats/incidence/risk/.

Cahill, S.P. & Foa, E.B. (2007). Psychological theories of PTSD. In Friedman, M.J., Keane, T.M., & Resick, P.A. (Eds), *Handbook of PTSD: Science & Practice* (pp. 55-77). New York: Guilford Press.

Campbell, D.G., Felker, B.L., Liu, C.F., Yano, E.M., Kirchner, J.E., Chan, D., Rubenstien, L.V., Chaney, E.F. (2007). Prevalence of depression-PTSD comorbidity: implications for clinical practice

guidelines and primary care-based interventions. *Journal of General Internal Medicine, 22*, (6), 711-718.

Cantor, N. & Mischel, W. (1977). Traits as prototypes: Effects on recognition memory. *Journal of Personality and Social Psychology, 35* (1), 38-48.

Castonguay, L.G., Goldfried, M.R., & Hayes, A.M. (1996). Predicting the effect of cognitive therapy for depression: A study of unique common factors. *Journal of Consulting and Clinical Psychology, 64*, 497-504.

Chan, M.W.C., Ho, S.M.Y., Tedeschi, R.G., & Leung, C.W.L. (2011). The valence of attentional bias and cancer-related rumination in posttraumatic stress and posttraumatic growth among women with breast cancer. *Psycho-Oncology, 20*, 544-552.

Cioffi, D., & Holloway, J. (1993). Delayed costs of suppressed pain. *Journal of Personality and Social Psychology, 64* (2), 274-282.

Clark, D.M. Ball, S., & Pape, D. (1991). An experimental investigation of thought suppression. *Behaviour Research and Therapy, 29* (3), 253-257.

Cloitre, M., Koenan, K.C., Cohen, L.R., & Han, H. (2002). Skills training in affective and interpersonal regulation followed by exposure: a phase-based treatment for PTSD related to childhood abuse. *Journal of Consulting and Clinical Psychology, 70,* 1067-1074.

Cloitre, M., Stovall-McClough, K.C., Nooner, K., Zorbas, P., Cherry, S., Jackson, C.L., Gan, W., & Petkova, E. (2010). Treatment for PTSD related to child abuse: a randomized controlled trial. *The American Journal of Psychiatry, 167,* 915-924.

Comprehensive Meta-Analysis (Version 2) [Computer software]. Englewood, NJ: Biostat.

Cook, J.M., Shnurr, P.P., & Foa, E.B. (2004). Bridging the gap between posttraumatic stress disorder research and clinical practice: the example of exposure therapy. *Psychotherapy: Theory , Research, Practice, Training, 41*, 374-387.

Coolican, H. (2004). *Research Methods and Statistics in Psychology* (4th Edition). UK: Routledge.

Cordova, M.J., Andrykowski, M.A., Kenady, D.E., McGrath, P.C., Sloan, D.A., & Redd, W.H. (1995). Frequency and correlates of posttraumatic-stress-disorder-like symptoms after treatment for breast cancer. *Journal of Consulting and Clinical Psychology, 63* (6), 981-986.

Cordova, M.J., Giese-Davis, J., Golant, M., Kronenwetter, C., Chang, V., & Spiegel, D. (2007). Breast cancer as trauma: posttraumatic stress and posttraumatic growth. *J Clin Psychol Med Settings, 14*, 308-319.

Cordova, M.J., Studts, J.L., Hann, D.M., Jacobsen, P.B., & Andrykowski, M.A. (2000). Symptom structure of PTSD following breast cancer. *Journal of Traumatic Stress, 13*, 301-319.

Costa-Requena, G. & Gil, F. (2010). Posttraumatic stress disorder symptoms in cancer: psychometric analysis of the Spanish Posttraumatic Stress Disorder Checklist-Civilian version. *Psycho-Oncology, 19*, 500-507.

Creamer, M., Burgess, P., & McFarlane, A.C., (2001) Post-traumatic stress disorder: findings from the Australian National Survey of Mental Health and Well-being. *Psychological Medicine* **31**(7), 1237-1247.

Creswell, J. (2003). *Research design: qualitative, quantitative, and mixed methods approaches (2nd ed.).* Thousand Oaks, CA: SAGE.

Creswell, J. & Miller, D. (2000). Determining validity in qualitative inquiry. *Theory Into Practice, 39* (3), 124-130.

Creswell, J. & Plano Clark, V.L. (2006). *Designing and Conducting Mixed Methods Research.* Sage Publications: USA.

Creswell, J. W., Plano Clark, V. L., Gutmann, M. L., & Hanson, W. E. (2003). Advanced mixed methods research designs. In A. Tashakkori & C. Teddlie (Eds.), *Handbook of mixed methods in social and behavioural research* (pp. 209–240).Thousand Oaks, CA: Sage.

Dalgleish, T. (2004). Cognitive theories of posttraumatic stress disorder: The evolution of multi-representational theorizing. *Psychological Bulletin, 130*, 228-260.

Dalgleish, T. & Power, M.J. (2004). Emotion-specific and emotion-non-specific components of posttraumatic stress disorder (PTSD): implications for a taxonomy of related psychopathology. *Behaviour Research and Therapy, 42* (9), 1069–1088.

Davidson, J.R.T., Book, S. W., Colket, J. T., & Tupler., L. A. (1997). Assessment of a new self-rating scale for post-traumatic stress disorder. *Psychological Medicine: A Journal of Research in Psychiatry and the Allied Sciences, 27* (1), 153-160.

Davidson, J.R.T. & Foa, E.B. (1991). Diagnostic issues in posttraumatic stress disorder: Considerations for the DSM-IV. *Journal of Abnormal Psychology, 100* (3), 346-355.

Department of Health. (2007). *Cancer Reform Strategy.* Department of Health.

De Jong, J.T.V.M., Komproe, I.H., Van Ommeren, M., (2003). Common mental disorder in postconflict settings. *The Lancet, 361* (9375), 2128-2130.

Dorenwend, B.P., Turner, J.B., Turse, N.A., Adams, B.G., Koenen, K.C., & Marshall, R. (2006). The psychological risks of Vietnam for U.S. veterans: a revisit with new data and methods. *Science, 313*, 979-982.

Dougall, A.L., Herberman, H.B., Inslicht, S.S., Baum, A., & Delahanty, D.L. (2000). Similarity of prior trauma exposure as a determinant of chronic stress responding to an airline disaster. *Journal of Consulting and Clinical Psychology, 68* (2), 290-295.

Ehlers, A. & Clark, D.M. (2000). A cognitive model of posttraumatic stress disorder. *Behaviour Research and Therapy, 38*, 319–345.

Ehlers, A., Clark, D.M., Hackmann, A., McManus, F., & Fennell, M. (2005). Cognitive therapy for post-traumatic stress disorder: development and evaluation. *Behaviour Research and Therapy, 43*, 413-431.

Elwood, L.S., Hahn, K.S., Olatunji, B.O. & Williams, N.L. (2009). Cognitive vulnerabilities to the development of PTSD: a review of four vulnerabilities and the proposal of an integrative vulnerability model. *Clinical Psychology Review, 29*, 87-100.

Epping-Jordan, J.E., Compas, B.E., Osowiecki, D.M., Oppedisano, G., Gerhardt, C., Primo, K., & Krag, D.N. (1999). Psychological adjustment in breast cancer: processes of emotional distress. *Health Psychology, 18* (4), 315-326.

Epstein, S. (1985). The implications of cognitive-experiential self-theory for research in social psychology and personality. *Journal of the Theory of Social Behaviour, 15*, 283-310.

Epstein, S. (1994). Integration of the cognitive and the psychodynamic unconscious. *American Psychologist, 49*, 709-724.

Everett, B.S. & Howell. (2005). Encyclopedia of Statistics in Behavioural Science (Vol. 3). UK: Wiley

Falsetti, S.A., Resnick, H.S., & Davis, J.L. (2008). Multiple-channel exposure therapy for women with PTSD and comorbid panic attacks. *Cognitive Behaviour Therapy, 37* (2), 117-130.

Farnsworth, J. K., & Sewell, K. W. (2011). Fear of emotion as a moderator between PTSD and firefighter social interactions. *Journal of Traumatic Stress, 24* (4), 444–450.

Fear, N.T., Jones, M., Murphy, D., Hull, L., Iversen, A.C. Coker, B., Machel, L., Sundin, J., Woodhead, C., Jones, N., Greenberg, N., Landau, S., Dandecker, C., Rona, R.J., Hotopf, M., & Wessely, S. (2010) What are the consequences of deployment to Iraq and Afghanistan on the mental health of the UK armed forces? A cohort study. *Lancet* 375 (9728), 1783-1797.

Feeny, N.C. (2003). Myths regarding exposure therapy for PTSD. *Cognitive and Behavioral Practice, 10* (10), 85-90.

Feeny, N.C., Zoellner, L.A., & Foa, E.B. (2002). Treatment outcome for chronic PTSD among female assault victims with borderline personality characteristics: A preliminary examination. *Journal of Personality Disorders, 16*, 30-40.

Ferry, F., Bolton, D., Bunting, B., Devine, B., McCann, S., & Murphy, S. (2008). Trauma, health and conflict in Northern Ireland. A study of the epidemiology of trauma related disorders and qualitative investigation of the impact of trauma on the individual. The Northern Ireland Centre for Trauma and Transformation and the Psychology Research Institute, University of Ulster.

Filstead, W. J. (1979). Qualitative methods - a needed perspective in evaluation research. In Cook, T. D. and Reichardt, C. S. (eds). *Qualitative and Quantitative Methods in Evaluation Research (pp. 33-48).* London: Sage.

First M.B., Spitzer, R.L., Gibbon, M., & Williams, J.B.W. (2002). *Structured Clinical Interview for DSM-IV-TR Axis I Disorders, Research Version, Non-patient Edition. (SCID-I/NP.)* New York: Biometrics Research, New York State Psychiatric Institute. #

Fiske, S. T., & Linville, P. W. (1980). What does the schema concept buy us? *Personality and Social Psychology Bulletin, 6*, 543–557.

Fiske, S. T., & Taylor, S. E. (1991). *Social cognition* (2nd ed.). New York: McGraw-Hill

Foa, E.B. & Cahill, S.P. (2001). Emotional processing in psychological therapies. In N.J. Smelser & P.B. Bates (Eds), *International Encyclopaedia of the Social and Behavioural Science* (pp. 12363-12369). New York: Elsevier.

Foa, E.B., Hembree, E.A., & Rothbaum, B.O. (2007). *Prolonged exposure therapy for PTSD: Emotional processing of traumatic experiences. Therapist guide.* New York, NY: Oxford University Press.

Foa, E.B., Huppert, J.D., & Cahill, S.P. (2006). Theoretical conceptualisations of pathological anxiety. In E.B. Foa (Ed), *Pathological Anxiety: Emotional Processing in Etiology and Treament.* (pp. 3-24). New York: Guildford Press.

Foa, E.B. & Kozak, M.J. (1986). Emotional processing of fear - exposure to corrective information. *Psychological Bulletin, 99*, 20-35.

Foa, E.B. & Meadows, E.A. (1997). Psychosocial treatments for posttraumatic stress disorder: A critical review. *Annual Review of Psychology, 48*, 449-480.

Foa, E.B., Riggs, D.S., & Gershuny, B.S. (1995). Arousal, numbing, and intrusion: symptom structure of

PTSD following assault. *Am J Psychiatry, 152*, 116-20.

Foa, E.B., Riggs, D.S., Massie, E.D., & Yarczower, M. (1995). The impact of fear activation and anger on the efficacy of exposure treatment for posttraumatic stress disorder. *Behaviour Therapy, 26*, 487-499.

Foa, E.B. & Rothbaum, B.O. (1998). *Treating the trauma of rape: cognitive behavioral therapy for PTSD.* New York: Guilford Press.

Foa, E.B. Steketee, G., & Rothbaum, B.O. (1989). Behavioural/cognitive conceptualisations of posttraumatic stress disorder. *Behaviour Therapy, 20*, 155-176.

Foster, C., Wright, D., Hill, H., Hopkinson, J. & Roffe, L. (2009). Psychosocial implications of living 5 years or more following a cancer diagnosis: a systematic review of the research evidence. *European Journal of Cancer Care 18*, 223–247.

Frans, Ö., Rimmö, P.-A., Åberg, L., & Fredrikson, M. (2005). Trauma exposure and post-traumatic stress disorder in the general population. *Acta Psychiatrica Scandinavica, 111* (4), 291-290.

Frazier, P.A., Gavian, M., Hirai, R., Park, C., Tennen, H., Tomich, P., & Tashiro, T. (2011).Prospective predictors of posttraumatic stress disorder symptoms: Direct and mediated relations. *Psychological Trauma: Theory, Research, Practice, and Policy, 3* (1), 27-36.

French-Rosas, L.N., Moye, J., Naik, A.D. (2011). Improving the recognition and treatment of cancer-related posttraumatic stress disorder. *Journal of Psychiatric Practice, 17* (4), 270-276.

Freuh, B.C., Grubaugh, A.L., Yeager, D.E., & Magruder, K.M. (2009). Delayed-onset post-traumatic stress disorder among war veterans in primary care clinics. *The British Journal of Psychiatry, 194*, 515-520.

Frewen, P.A., Dozois, D.J.A., Neufeld, R.W.J., Lanius, R.A. (2008). Meta-analysis of alexithymia in posttraumatic stress disorder. *Journal of Traumatic Stress, 21*, 243-246.

Friedman, M.J. & Yehuda, R. (1995). Post-traumatic Stress Disorder and comorbidity: Psychobiological approaches to differential diagnosis. In M.J. Friedman, D.S. Charney, & A.Y. Deutch (Eds), *Neurobiological and clinical consequences of stress: From normal adaptation to Post-Traumatic Stress Disorder* (pp. 429-446). Philadelphia, PA: Lippincott-Raven Publishers.

Gagnier, J.J., Kienle, G., Altman, D.G., Moher, D., Sox, H., & Riley, D. (2013). The CARE Guidelines: Consensus-Based Clinical Case Reporting Guideline Development. *The Journal of Head and Face Pain, 53*, 1541-1547.

Gall, M., Gall, J., & Borg, W. (2003). *Educational Research.* Boston: Pearson Education, Inc.

Gandubert, C., Carriere, I., Escot, C., Soulier, M., Hermes, A., Boulet, P., Ritchie, K., & Chaudieu, I. (2009). Onset and relapse of psychiatric disorders following early breast cancer: a case-control study. *Psycho-Oncology, 18*, 1029-1037.

Gendlin, E. (1982). *Focusing.* Bantam

Golden-Kreutz, D.M. & Andersen, B.L. (2004). Depressive symptoms after breast cancer surgery: relationships with global, cancer-related, and life event stress. *Psycho-Oncology, 13*, 211-220.

Goncalves, V., Jayson, G., & Tarrier, N. (2011). A longitudinal investigation of posttraumatic stress disorder in patients with ovarian cancer. *Journal of Psychosomatic Research, 70*, 422-431.

Gottman, J. & DeClaire, J. (1997). *Raising an emotionally intelligent child: The heart of parenting.* Fireside; New York.

Green, B.L., Epstein, S., Krupnick, J.L., & Rowland, J. (1997). Trauma and medical illness: Assessing trauma-related disorders in medical settings. In *Assessing Psychological Trauma and PTSD*, Wilson, J. & Keane, T. (Eds). Guilford Press: New York; 160-191.

Green, B.L., Krupnick, J.L., Rowland, J.H., Epstein, S.A. Stockton, P., Spertus, I., & Stern, N. (2000). Trauma history as a predictor of psychologic symptoms in women with breast cancer. *Journal of Clinical Oncology, 18* (5), 1084-1093.

Green, B.L., Lindy, J.D., Grace, M.C., & Leonard, A.C. (1992). Chronic posttraumatic stress disorder and diagnostic comorbidity in a disaster sample. *Journal of Nervous and Mental Disease, 180*, 70-766.

Green, B.L. Rowland, J.H., Krupnick, J.L., Epstein, S.A., Stockton, P., Stern, N.M., Spertus, I.L., & Steakley, C. (1998). Prevalence of posttraumatic stress disorder in women with breast cancer. *Psychosomatics, 39*, 102-111.

Greenberg, L.S. & Safran, J.D. (1987). *Emotion in psychotherapy: Affect, cognition, and the process of change.* New York, NY: The Guildford Press.

Greenberg, L.S. & Safran J.D. (1989). Emotion in psychotherapy. *American Psychologist, 44*, 19-29.

Greene, J. C., Caracelli, V. J., & Graham, W. F. (1989). Toward a conceptual framework for mixed-method evaluation designs. *Educational Evaluation and Policy Analysis, 11*, 255-274.

Guglietti, C.L., Rosen, B., Murphy, K.J., Laframboise, S., Dodge, J., Ferguson, S., Katz, J., Ritvo, P. (2010). Prevalence and predictors of posttraumatic stress in women undergoing an ovarian cancer investigation. *Psychological Services, 7*, 266-274

Gurevich, M., Devins, G.M., Rodin, G.M. (2002). Stress response syndromes and cancer: conceptual and assessment issues. *Psychosomatics, 43,* 259-281.

Halstead, M. T., & Fernsler, J. I. (1994). Coping strategies of long-term cancer survivors. *Cancer Nursing, 17*, (2), 94-100.

Hammond, C. (2005). The wider benefits of adult learning: An illustration of the advantages of multi-method research. *International Journal of Social Research Methodology, 8* (3), 239-255.

Hampton, M.R. & Frombach, I. (2000). Women's experience of traumatic stress in cancer treatment. *Healthcare for Women International, 21*, 67-76.

Harvey, A.G., & Bryant, R.A. (2002). Acute stress disorder: a synthesis & critique. *Psychological Bulletin, 128*, 886-902.

Hayes, S.C., Wilson, K.G., Gifford, E.V., Follette, V.M., & Strosahl, K. (1996). Experiential avoidance and behavioral disorders: A functional dimensional approach to diagnosis and treatment. *Journal of Consulting and Clinical Psychology 64* (6): 1152–68.

Helzer, J.E., Robins, L.N., & McEvoy, L. (1987). Post-traumatic stress disorder in the general population: Findings of the Epidemiologic Catchment Area survey. *New England Journal of Medicine, 317* (26), 1630-1634.

Hembree, E.A. Foa, E.B., Dorfan, N.M, Street, G.P., Kowalski, J., & Tu, X. (2003). Do patients drop out prematurely from exposure therapy for PTSD? *Journal of Traumatic Stress, 16*, 555-562.

Hennekens, C.H. & Buring, J.E. (1987). *Epidemiology in Medicine.* Lippincott Williams & Wilkins.

Herschbach, P., Berg, P., Dankert, A., Duran, G., Engst-Hastreiter, Waadt, S., Keller, M., Ukat, R., & Henrich, G. (2005). Fear of progression in chronic diseases: Psychometric properties of the Fear of

Progression Questionnaire. *Journal of Psychosomatic Research, 58* (6), 505-511.

Hodges, L. & Humphris, G. (2009). Fear of recurrence and psychological distress in head and neck cancer patients and their carers, *Psycho-Oncology, 18* (8), 841-848.

Holloway, I. (2008). *A-Z of Qualitative Research in Healthcare* (2nd Edition). UK: Blackwell.

Holloway, I. & Wheeler, S. (2010). *Qualitative Research in Nursing & Healthcare* (3rd Edition). UK: Wiley-Blackwell.

Hooper, L., Stockton, P., Krupnick, J., & Green, B., (2011). Development, use, and psychometric properties of the Trauma History Questionnaire. *Journal of Loss and Trauma, 16*, 258-283.

Howell, D.C. (2013). *Statistical Methods for Psychology* (8th Edition). Wadsworth Cengage Learning.

Horowitz, M.J. (1986). *Stress response syndromes (2nd ed.)*. Northvale, NJ: Jason Aronson.

Hoyt, T., Pasupathi, M., Smith, B.W., Yeater, E.A., Kay, V.S., & Tooley, E. (2010). Disclosure of emotional events in groups at risk for posttraumatic stress disorder. *International Journal of Stress Management, 17* (1), 78-95.

Huffman, J.C., Niazi, S.K., Rundell, J.R., Sharpe, M., & Katon, W.J. (2014). Essential articles on collaborative care models for the treatment of psychiatric disorders in medical settings: a publication by the academy of psychosomatic medicine research and evidence-based practice committee. *Psychosomatics, 55*, 109-122.

Hunt, M.G. (1998). The only way out is through: emotional processing and recovery after a depressing life event. *Behaviour Research and Therapy, 36*, 361-384.

Ingram, R.E., & Price, J.M. (2001). *Vulnerability to psychopathology: Risk across the lifespan*. New York, NY: The Guilford Press

Jacobsen, P. B., Sadler, I.J., Booth-Jones, M., Soety, E., Weitzner, M.A., & Fields, K.K. (2002). Predictors of post-traumatic stress disorder symptomatology following bone marrow transplantation for cancer. *Journal of Consulting and Clinical Psychology, 70* (1), 235-240.

Jacobsen, P.B., Widows, M.R.., Hann, D.M., Andrykowski, M.A., Kronish, L.E., & Fields, K.K. (1998). Posttraumatic stress disorder symptoms asfter bone marrow tranasplantation for breast cancer. *Psychosomatic Medicine, 60,* 366-371.

Janoff-Bulman, R. (1989). Assumptive worlds and the stress of traumatic events: Applications of the schema construct. *Social Cognition, 7*, 113-136.

Janoff-Bulman, R. (1992). *Shattered assumptions: Towards a new psychology of trauma.* New York: Free Press.

Jaycox, L.H. & Foa, E.B. (1996). Obstacles in implementing exposure therapy for PTSD: Case discussions and practical solutions. *Clinical Psychology and Psychotherapy, 3*, pp. 176–184.

Jim, H.S., Andrykowski, M.A., Munster, P.N., & Jacobsen, P.B. (2007). Physical symptoms/side effects during breast cancer treatment predict posttreatment distress. *Ann Behav Med, 34* (2), 200-208.

Johnson, R. B. & Onwuegbuzie, A. J. (2004). Mixed methods research: A research paradigm whose time has come. *Educational Researcher, 33* (7), 14-26.

Joseph S. (2000). Psychometric evaluation of Horowitz's Impact of Event Scale: A review. *Journal of Traumatic Stress*, *13*, 101–113.

Kangas, .M. (2013). DSM-5 trauma and stress-related disorders: implications for screening for cancer-related stress. *Front. Psychiatry*, doi: 10.3389/fpsyt.2013.00122

Kangas, M., Henry, J.L., & Bryant, R.A. (2002). Posttraumatic stress disorder following cancer. A conceptual and empirical review. *Clinical Psychology Review, 22*, 499-524.

Kangas, M., Henry, J.L., & Bryant, R.A. (2005a). Predictors of posttraumatic stress disorder following cancer. *Health Psychology, 24*, 579-585.

Kangas, M., Henry, J.L., & Bryant, R.A. (2005b). The relationship between acute stress disorder and posttraumatic stress disorder following cancer. *Journal of Consulting and Clinical Psychology, 73* (2), 360-364.

Kangas, M., Henry, J.L., & Bryant, R.A. (2005c). Correlates of acute stress disorder in cancer patients. *Journal of Traumatic Stress, 20* (3), 325-334.

Kessler, R.C., Sonneha, A., Bromet, E., Hughes, M., & Nelson, C.B. (1995). Posttraumatic stress disorder in the National Co-morbity Survey. *Archives of General Psychiatry 52* (12), 1048-1060.

Kessler, R. C., Zhao, S., Katz, S. J., Kouzis, A. C., Frank, R. G., Edlund,M. & Leaf, P. (1999a). Past-year use of outpatient services for psychiatric problems in the National Co-morbidity Survey. *American Journal of Psychiatry 156*, 115±123.

Kessler, R.C., Chiu, W.T., Demler, O. et al. (2005). Prevalence, severity, and co-morbidity of 12-month DSM-IV disorders in the National Comorbidity Survey Replication. *Archives of General Psychiatry, 62* (6), 617-627.

Kilpatrick, D.G., Resnick, H.S., & Acierno, R. (2009). Should PTSD Criterion A be retained? *Journal of Traumatic Stress, 22* (5), 374-383.

Kilpatrick, D.G., Resnick, H.S., Freedy, J.R., Pelcovitz, D., Resick, F, Roth, S., & van der Kolk, B. (1997). The posttraumatic stress disorder field trial: Evaluation of the PTSD construct: Criteria A through E. In T A., Widiger, A. J., Frances, H. A., Pincus, M. B., First, R. Ross, & W. Davis (Eds.) *DSM-IV Sourcebook (Volume IV)*. Washington, DC: American Psychiatric Press.

Kilpatrick, D. G., Saunders, B. E., Best, C. L., & Von, J. M. (1987). Criminal victimization: Lifetime prevalence, reporting to police, and psychological impact. *Crime & Delinquency, 33*, 479-489.

Kilpatrick, D. G., Saunders, B. E., Amick-McMullan, A., Best, C. L., Veronen, L. J., & Resnick, H. S. (1989). Victim and crime factors associated with the development of crime-related post-traumatic stress disorder. *Behavior Therapy, 20*, 199-214.

Kilpatrick, D.G., Veronen, L.J., & Best, C.L. (1985). Factors predicting psychological distress among rape victims. In C.R. Figley (Ed.), *Trauma and its Wake* (pp. 113-141). New York: Brunner.

King, D.W., King, L.A., Fairbank, J.A., Keane, T.M., & Adams, G. (1998). Resilience-recovery factors in post-traumatic stress disorder among female and male Vietnam veterans: Hardiness, postwar social support, and additional stressful life events. *Journal of Personality and Social Psychology, 74* (2), 420-434.

King, D.W., King, L.A., Foy, D.W., Keane, T.M., & Fairbank, J.A. (1999). Posttraumatic stress disorder in a national sample of female and male Vietnam veterans: Risk factors, war-zone stressors, and resilience-recovery variables. *Journal of Abnormal Psychology, 108* (1), 164-170.

Klein, C. (2008). The metastasis cascade. *Science, 321*, 1785-1787.

Klein, M.H., Mathieu, P.L., Gendlin, E.T., & Kiesler, D.J. (1969). *The experiencing scale: a research and training manual* (Vol 1). Madison: University of Wisconsin.

Klein, S. & Alexander, D.A. (2006). Epidemiology and presentation of post-traumatic disorders.

Psychiatry, 5 (7), 225-227.

Klein, S. & Alexander, D.A. (2009). Epidemiology and presentation of post-traumatic disorders. *Psychiatry,* 8 (8), 282-287.

Koenen, K.C., Stellman, J.M., Stellman, S.D., & Sommer Jr., J.F. (2003). Risk factors for course of posttraumatic stress disorder among Vietnam veterans: A 14-year follow-up of American legionnaires. *Journal of Consulting and Clinical Psychology, 71* (6), 980-986.

Koopman, C., Butler, L.D., Classen, C., Giese-Davis, J., Morrow, G.R., Westerndorf, J., Banerjee, T., & Spiegel, D. (2002). Traumatic stress symptoms among women with recently diagnosed primary breast cancer. *Journal of Traumatic Stress, 15* (4), 277-287.

Kornblith, A. B., Herndon, J. E., Weiss, R. B., Zhang, C., Zuckerman, E. L., Rosenberg, S., Mertz, M., Payne, D., Massie, M. J., Holland, J. F., Wingate, P., Norton, L., & Holland, J. C. (2003). Long-term adjustment of survivors of early-stage breast carcinoma, 20 years after adjuvant chemotherapy. *Cancer, 98*, 679-689.

Koutrouli, N., Anagnostopoulos, F., & Potamianos, G. (2012). Posttraumatic stress disorder and posttraumatic growth in breast cancer patients: A systematic review. *Women & Health, 52*, 503-516.

Kraemer, H.C., Stice, E., Kasdin, A., Offord, D., & Kupfer, D. (2001). How do risk factors work together? Mediators, moderators, and independent, overlapping, and proxy risk factors. *American Journal of Psychiatry, 158*, 848-856.

Krantz, D.L. (1995). Sustaining vs. resolving the quantitative-qualitative debate. *Evaluation and Program Planning, 18* (1), 89-96.

Kubany, E. S., & Manke, F. P. (1995). Cognitive therapy for trauma-related guilt: Conceptual bases and treatment outlines. *Cognitive and Behavioral Practice, 2*, 27-61.

Kulka, R.A., Schlenger, W.E., Fairbank, J.A., Hough, R.L., Jordan, B.K., & Marmar, C.R. (1990). *Trauma and the Vietnam War generation: Report of findings from the National Vietnam Veterans Readjustment Study* (Vol. 29). New York: Brunner/Mazel.

Kwekkeboom, K.L. & Seng, J.S. (2002). Recognising and responding to post-traumatic stress disorder in people with cancer. *Oncology Nursing Forum, 29* (4), 643-650.

Lang, P.J. (1977). Imagery in therapy: An information-processing analysis of fear. *Behaviour Therapy, 8*, 862-886.

Lang, P.J. (1984). Cognition in emotion: Concept and action. In C. Izard., J. Kagan, & R. Zajonc (Eds)., *Emotion, Cognition, & Behavior* (pp. 193-206). New York: Cambridge University Press.

Langer, E. J., & Abelson, R. P. (1974). A patient by any other name . . .: Clinician group difference in labeling bias. *Journal of Consulting and Clinical Psychology, 42*, 4–9.

Lazarus, R., & Folkman, S. (1984). *Stress, appraisal, and coping.* New York: Springer Publishing Company.

Leahy, R.L. (2002). A model of emotional schemas. *Cognitive and Behavioural Practice, 9* (3), 177-190.

Leahy, R.L. (2007). Emotional schemas and resistance to change in anxiety disorder. *Cognitive and Behavioural Practice, 14, 36-45.*

Lee, D., Scragg, P., & Turner, S. (2001). The role of shame and guilt in traumatic events: A clinical model of shame-based and guilt-based PTSD. *British Journal of Medical Psychology, 74*, 451-466.

Lepore, S.J. (2001). A social-cognitive processing model of emotional adjustment to cancer. In A. Baum,

& B. Andersen (Eds.). *Psychosocial Interventions for Cancer* (pp. 99-118). Washington, DC: APA Books.

Lepore, S. J., & Helgeson, V. S. (1998). Social constraints, intrusive thoughts, and mental health after prostate cancer. *Journal of Social and Clinical Psychology, 17*, 89-106.

Lepore, S.J., Silver, R.C., Wortman, C.B., & Wayment, H.A. (1996). Social constraints, intrusive thoughts, and depressive symptoms among bereaved mothers. *Journal of Personality and Social Psychology, 70* (2), 271-282.

Lethborg, C.E., Kissane, D., Burns, W.I., & Snyder, R. (2000). "Cast adrift": The experience of completing treatment among women with early stage breast cancer. *Journal of Psychosocial Oncology, 18*, 73-90.

Leventhal, H. (1979). A perceptual-motor processing model of emotion. In P. Pilner, K. Blankenstein, & I.M. Speigel (Eds), (Vol. 5, pp.1-46) New York, NY: Plenum.

Levine, E.G., Eckhardt, J., & Targ, E. (2005). Change in post-traumatic stress symptoms following psychosocial treatment for breast cancer. *Psycho-Oncology, 14*, 618-635.

Liebschutz, J., Saitz, R., Brower, V., Keane, T.M., Lloyd-Travaglini, C., Averbuch, T., & Samet, J.H. (2007). PTSD in urban primary care: High prevalence, low physician recognition. *Society of General Internal Medicine, 22*, 719-726.

Lincoln, Y. S., & Guba, E. G. (1985). *Naturalistic inquiry.* Beverly Hills, CA: Sage.

Lipov, E.G., Joshi, J.R., Sanders, S. (2008). Effects of stellate-ganglion block on hot flushes and night awakenings in survivors of breast cancer: a pilot study. *Lancet Oncology, 9*, 523-532.

Luecken, L.J., Dausch, B., Gulla, V., Hong, R. & Compas, B.E. (2004). Alterations in morning cortisol with PTSD in women with breast cancer. *Journal of Psychosomatic Research, 56*, 13-15.

Manne, S. L. (1999). Intrusive thoughts and psychological distress among cancer patients: The role of spouse avoidance and criticism. *Journal of Consulting and Clinical Psychology, 67*, 539-546.

Menard, S. (1995). *Applied Logistic Regression Analysis* (2nd Edition). London: Sage.

Marks, I. (1979). Exposure therapy for phobias and obsessive compulsive disorders. *Hospital Practice, 14*, 101-108.

Marshall, R.D., Amsel, L., & Suh, E.J. (2008). Response to McNally and Breslau (2008). *American Psychologist,* 63, 283–285.

Marshall, R.D., Spitzer, R., & Liebowitz, M.R. (1999). Review and critique of the new DSM-IV diagnosis of acute stress disorder. *American Journal of Psychiatry, 156*, 1677-1685.

Matsuoka, Y., Inagaki, M., Sugawara, Y., Imoto, S., Akechi, T., & Uchitomi, Y. (2005). Biomedical and psychosocial determinants of intrusive recollections in breast cancer survivors. *Psychosomatics, 46*, 203-211.

McDonald, S. D., & Calhoun, P. S. (2010). The diagnostic accuracy of the PTSD checklist: A critical review. *Clinical Psychology Review, 30*, 976-987.

McFarlane, A.C. (1997). The prevalence and longitudinal course of PTSD. Annals of the New York Academy of Sciences, 821:10-23. doi:10.1111/j.1749-6632.1997.tb48265.x. *The Psychobiology of Post-traumatic Stress Disorder, 821,* 10-23.

McFarlane, A.C. & De Girolamo, G. (2007). The nature of traumatic stressors and the epidemiology of posttraumatic reactions. In van der Kolk, B.A., McFarlane, A.C., & Weisaeth, L. (Eds), *Traumatic*

Stress: The Effects of Overwhelming Experience on Mind, Body, and Society. (pp.129-154). New York: Guilford Press.

McGrath, J. E. (1976). Stress and behavior in organizations. Chap. 31 in Dunnette, M. (Ed.), *Handbook of Industrial Organizational Psychology.* Chicago: Rand McNally Co., Inc. pp. 1351–1395.

McKeever, V.M. & Huff, M.E. (2003). A diathesis-stress model of posttraumatic stress disorder: Ecological, biological, and residual stress pathways. *Journal of General Psychology, 7* (3), 237-250.

McManus, S., Meltzer, H., Brugha, T., Bebbington, P., & Jenkins, J. (2009) *Adult psychiatric morbidity in England, 2007: Results of a household survey.* The Health & Social Care Information Centre.

McNally, R.J. (2001). Vulnerability to anxiety disorders in adulthood. In R.E. Ingram, & J.M. Price, (Eds), *Vulnerability to psychopathology: Risk across the lifespan* (pp. 304-321). New York, NY: The Guilford Press.

McNally, R.J., Bryant, R.A., & Ehlers, A. (2003). Does early psychological intervention promote recovery from posttraumatic stress? *Psychological Science in the Public Interest, 4*, 45-79.

Meeske, K.A., Ruccione, K., Globe, D.R., & Stuber, M.L. (2001). Posttraumatic stress, quality of life, and psychological distress in young adult survivors of childhood cancer. *Oncology Nursing Forum, 28* (3), 481-489.

Mehnert, A., Berg, P., Henrich, G., & Herschbach, P. (2009). Fear of cancer progression and cancer-related intrusive cognitions in breast cancer survivors. *Psycho-Oncology, 18*, 1273-1280.

Mehnert, A. & Koch, U. (2007). Prevalence of acute and post-traumatic stress disorder and comorbid mental disorders in breast cancer patients during primary cancer care: A prospective study. *Psycho-Oncology, 16*, 181-199.

Mehnert, A. & Koch, U. (2008). Psychological co-morbidity and health-related quality of life and its association with awareness, utilization, and need for psychosocial support in a cancer register-based sample of long-term breast cancer survivors. *Journal of Psychosomatic Research, 64*, 383-391.

Menard, S. (1995). Applied logistic regression analysis. Thousand Oaks: Sage University paper series on quantitative applications in the social sciences, 07-106.

Miles, M. B., & Huberman, A. M. (1994). *Qualitative data analysis: An expanded sourcebook.* Thousand Oaks, CA: Sage.

Moher, D., Liberati, A., Tetzlaff, J., & Altman, D.G. (2009). The PRISMA Group. Preferred Reporting Items for Systematic Reviews and Meta-Analyses: The PRISMA Statement. BMJ, 339, 2535, doi: 10.1136/bmj.b2535

Monson, C.M., Price, J.L. Rodriguez, B.F., Ripley, M.P., & Warner, R.A. (2004). Emotional deficits in military-related PTSD: an investigation of content and process disturbances. *Journal of Traumatic Stress, 17* (3) 275-279.

Morrill, E.F., Brewer, N.T., O'Neill, S.C. Lillie, S.E., Dees, E.C., Carey, L.A. & Rimer, B.K. (2008). The interaction of post-traumatic growth asnd post-traumatic stress symptoms in predicting depressive symptoms and quality of life. *Psycho-Oncology, 17*, 948-953.

Mundy, E. A., Blanchard, E. B., Cirenza, E., Gargiulo, J., Maloy, B., & Blanchard, C. G. (2000). Posttraumatic stress disorder in breast cancer patients following autologous bone marrow transplantation or conventional cancer treatments. *Behaviour Research & Therapy, 38*, 1015–1027.

Myers, R.H. (1990). *Classical and Modern Regression Application* (2nd Edition). Boston: Duxbury Press.

Mystakidou, K., Parpa, E., Tsilika, E., Panagiotou, I., Roumeliotou, A., Galanos, A., & Gouliamos, A. (2012). Traumatic experiences of patients with advanced cancer. *Journal of Loss and Trauma, 17,* 125-136.

Naidich, J. B., & Motta, R. W. (2000). PTSD-related symptoms in women with breast cancer. *Journal of Psychotherapy in Independent Practice, 1*, 35–54.

National Collaborating Centre for Mental Health (2005) *Post-traumatic Stress Disorder: the Management of PTSD in Adults and Children in Primary and Secondary Care.* Leicester and London: The British Psychological Society and the Royal College of Psychiatrists. [Full guideline]

National Institute of Clinical Excellence (2005) Post Traumatic Stress Disorder (PTSD): The management of PTSD in adults and children in primary and secondary care. Clinical Guidance 26. March 2005.

Newman, M.G., Castonguay, L.G., Borkovec, T.D., Fisher, A.J., Nordberg, S.S. (2008). An open trial of integrative therapy for generalized anxiety disorder. *Psychotherapy: Theory, Research, Practice, Training, 45* (2), 135-147.

Nir, Y. (1995). Post-traumatic stress disorder in children with cancer. In *Posttraumatic Stress Disorder in Children* (S. Eth & R. Pynoos Eds). American Psychiatric Press: Washington DC, PP. 123-132.

Nishith, P., Mechanic, M.B., Resick, P.A. (2000). Prior interpersonal trauma: The contribution to current PTSD symptoms in female rape victims. *Journal of Abnormal Psychology, 109* (1), 20-25.

Nixon, R.D.V. & Bryant, R.A. (2003). Peritraumatic and persistent panic attacks in acute stress disorder. *Behavior Research and Therapy, 41*, 1237-1242.

Nolen-Hoeksema, S. (1991). Responses to depression and their effects on the duration of depressive episodes. *Journal of Abnormal Psychology, 100*, 569-582.

Norris, F.H. (1992). Epidemiology of trauma: Frequency and impact of different potentially traumatic events on different demographic groups. *Journal of Consulting and Clinical Psychology, 60* (3), 409-418.

Norris, F.H., Murphy, A.D., Baker, C.K.., Perilla, J.L., Rodriguez, F.G., Rodriguez, J.J.G. (2003). Epidemiology of trauma and posttraumatic stress disorder in Mexico. *Journal of Abnormal Psychology, 112* (4), 646-656.

Norris, F.H. & Murrell, S.A. (1988). Prior experience as a moderator of disaster impact on anxiety symptoms in older adults. *American Journal of Community Psychology, 16*, 665-683.

Norris, F.H. & Sloane, L.B. (2007). The epidemiology of trauma and PTSD. In Friedman, M.J., Keane, T.M., & Resick, P.A. (Eds), *Handbook of PTSD: Science & Practice* (pp. 78-98). New York: Guilford Press.

O'Connor, M., Christense, S., Jensen, A.B., Møller, S., & Zachriae, R. (2011). How traumatic is breast cancer? Post-traumatic stress symptoms (PTSS) and risk factors for severe PTSS at 3 months and 15 months after surgery in a nationwide cohort of Danish women treated for primary breast cancer, *British Journal of Cancer, 104* (3): 419–426.

O'Connor, M., Lasgaard, M., Spindler, H., & Elklit, A. (2007). The impact of different diagnostic criteria on PTSD prevalence A comparison of PTSD and prevalence using the DSM-IV and ICD-10 PTSD criteria on a population of 242 Danish social work students. *Nordic Psychology, 59* (4), 317-

331.

Onwuegbuzie, A., & Leech, N. (2005). On becoming a pragmatic researcher: The importance of combining quantitative and qualitative research methodologies. *International Journal of Social Research Methodology, 8* (5), 375-387.

Orlinsky, D.E. & Howard, K.I. (1986). The relation of process to outcome in psychotherapy. In S.L. Garfield & A.E. Bergin (Eds), *Handbook of Psychotherapy and behaviour change (3rd Ed)*. New York: Wiley.

Ozer, E.J., Best, S.R., Lipsey, T.L., & Weiss, D.S. (2003). Predictors of posttraumatic stress disorder and symptoms in adults: A meta-analysis. *Psychological Bulletin, 129* (1), 52-73.

Ozer, E. J., & Weiss, D. S. (2004). Who develops posttraumatic stress disorder? *Current Directions in Psychological Science, 13*, 169–172.

Palmer, S.C., Kagee, A., Coyne, J.C., & DeMichele, A. (2004). Experience of trauma, distress, and posttraumatic stress disorder among breast cancer patients. *Psychosomatic Medicine, 66*, 258-264.

Parikh, R., Mathai, A., Parikh, S., Sekhar, G.C., & Thomas, R. (2008). Understanding and using sensitivity, specificity, and predictive values. *Indian Journal of Ophthalmology, 56* (1), 45-50.

Perconte, S.T. & Griger, M.L. (1991). Comparison of successful, unsuccessful, and relapsed Vietnam veterans treated for post-traumatic stress disorder. *The Journal of Nervous and Mental Disease, 179*, 558-562.

Perkonigg, A., Kessler, R.C., Storz, S., & Wittchen H-U. (2000). Traumatic events and post-traumatic stress disorder in the community: prevalence, risk factors and comorbidity. *Acta Psychiatr Scand, 101*, 46-59.

Perrin, M.A., DiGrande, L., Wheeler, K., Thorpe, L., Farfel, M., & Brackbill, R. (2007). Differences in PTSD prevalence and associated risk factors among world trade center disaster rescue and recovery workers. *American Journal of Psychiatry, 164*, 1385-13984.

Pitman, R. K., Altman, B., Greenwald, E., Longpre, R. E., Macklin, M. L., Poire, R. E., & Steketee, G. S. (1991). Psychiatric complications during flooding therapy for post-traumatic stress disorder. *Journal of Clinical Psychiatry, 52*, 17-20

Pitman, R.K., Lanes, D.A., Williston, S.K., Guillaume, J.L., Metzger, L.J., Gehr, G.M., & Orr, S.P. (2001). Psychophysiologic assessment of posttraumatic stress disorder in breast cancer patients. *Psychosomatics, 42*, 133-140.

Posluszny, D.M., Edwards, R.P., Dew, M.A., & Baum, A. (2011). Perceived threat and PTSD symptoms in women undergoing surgery for gynaecologic cancer or benign conditions. *Psycho-Oncology, 20*, 783-787.

Price, J. L., Monson, C. M., Callahan, K., & Rodriguez, B. F. (2006). The role of emotional functioning in military-related PTSD and its treatment. *Journal of Anxiety Disorders, 20* (5), 661–674.

Rachman, S. (1980). Emotional processing. *Behaviour Research and Therapy, 18* (1) 51-60.

Rachman, S. (2001). Emotional processing, with special reference to post-traumatic stress disorder. *International Review of Psychiatry*, *13* (3), 164-171.

Rauch, S. & Foa, E. (2006). Emotional processing theory (EPT) and exposure therapy for PTSD. *Journal of Contemporary Psychotherapy, 36*, 61-65.

Reiss, S. (1991). Expectancy model of fear, anxiety, and panic. *Clinical Psychology Review, 11*, 141-153.

Resick, P.A., Nishith, P., Weaver, T.L., Astin, M.C., & Feuer, C. (2002). A comparison of cognitive-processing therapy with prolonged exposure and a waiting condition for the treatment of chronic posttraumatic stress disorder in female rape victims. *Journal of Consulting and Clinical Psychology, 70*, 867-879.

Resnick, H.S., Kilpatrick, D.G., Dansky, B.S., Saunders, B.E., & Best, C.L. (1993). Prevalence of civilian trauma and posttraumatic stress disorder in a representative national sample of women. *Journal of Consulting and Clinical Psychology, 61* (6), 984-991.

Riskind, J.H. (1997). Looming vulnerability to threat: A cognitive paradigm for anxiety. *Behavior Research and Therapy, 35*, 793-802.

Riskind, J.H. & Kleiman, E.M. (2012). Looming Cognitive Style, Emotion Schemas, and Fears of Loss of Emotional Control: Two Studies. *International Journal of Cognitive Therapy, 5*, 392-405.

Riskind, J.H., Williams, N.L., & Joiner, T.E. (2006). The looming cognitive style: a cognitive vulnerability for anxiety disorders. *Journal of Social and Clinical Psychology, 25*, 779-801.

Robichaud, M. & Dugas, M.J. (2005). Negative problem orientation (part II): construct validity and specificity to worry. *Behaviour Research & Therapy, 43* (3), 403-412.

Rubonis, A. & Bickman, L. (1991). Psychological impairment in the wake of disaster: The disaster-psychopathology relationship. *Psychological bulletin, 109*, 384-399.

Salsman, J.M., Segerstrom, S.C., Brechting, E.H., Carlson, C.R., & Andrykowski, M.A. (2009). Posttraumatic growth and PTSD symptomatology among colorectal cancer survivors: a 3-month longitudinal examination of cognitive processing. *Psycho-Oncology, 18*, 30-41.

Saracci, R. (2010). *Epidemiology: A Very Short Introduction.* UK: OUP Oxford.

Schroevers, M.J., Helgeson, V.S., Sanderman, R., & Ranchor, A.V. (2010). Type of social support matters for prediction of posttraumatic growth among cancer survivors. *Psycho-Oncology, 19*, 46-53.

Sechrest, L. & Sidani, S. (1995). Quantitative and qualitative methods: Is there an alternative? *Evaluation and Program Planning, 18* (1), 77-87.

Shalev, A.Y., Freedman, S., Peri, T., Brandes, D., Sahar, T., Orr, S.P., & Pitman, R.K. (1998). Prospective study of posttraumatic stress disorder and depression following trauma. *American Journal of Psychiatry, 155*, 630-637.

Shalev, A.Y., Peri, T., Canetti, L., & Schreiber, S. (1996). Predictors of PTSD in injured trauma survivors: a prospective study. *The American Journal of Psychiatry, 153* (2), 219-225.

Shelby, R.A., Golden-Kreutz, D.M., & Andersen, B.L. (2005). Mismatch of posttraumatic stress disorder (PTSD) symptoms and DSM-IV symptom clusters in a cancer sample exploratory factor analysis of the PTSD Checklist-Civilian Version. J*ournal of Traumatic Stress, 18*, 347-357.

Shelby, R.A. Golden-Kreutz, D.M., & Andersen, B.L. (2008). PTSD diagnoses, subsyndromal symptoms, and co morbidities contributing to Impairments for breast cancer survivors. *Journal of Traumatic Stress, 21* (2), 165-172.

Silberstein, L.R., Tirch, D., Leahy, R.L., McGinn, L. (2012). Mindfulness, Psychological Flexibility and Emotional Schemas. *International Journal of Cognitive Therapy, 5,* 406-419.

Smid, G.E., Mooren, T.T., van der Mast, R.C., Gersons, B.P., & Kleber, R.J. (2009). Delayed posttraumatic stress disorder: systematic review, meta-analysis, and meta-regression analysis of prospective studies. *The Journal of Clinical Psychiatry, 70* (11), 1572-1582.

Smith, J. (1983). Quantitative versus qualitative research: An attempt to clarify the issue. *Educational Researcher, 12* (3), 6-13.

Smith, E.M. & North, C.S. (1993). Post-traumatic stress disorder in natural disasters and technological accidents. In J.P. Wilson & B. Raphael (Eds). *International Handbook of Traumatic Stress Syndromes (pp. 405-419)*. New York: Plenum Press.

Smith, M. Y., Redd, W. H., Peyser, C., & Vogl, D. (1999). Post-traumatic stress disorder in cancer: A review. *Psycho-Oncology, 8*, 521-537.

Snow, B.R., Stellman, J.M., Stellman, S.D., & Sommer, J.F. (1988). Post-traumatic stress disorder among American Legionnaires in relation to combat experience in Vietnam: Associated and contributing factors. *Environmental Research, 47*, 175-192.

Stanfield, J. (2006). The possible restorative justice functions of qualitative research. *International Journal of Qualitative studies in Education, 19* (6), 723-727.

Steinberg, A.M., Brymer, M.J., Kim, S., Ghosh, C., Ostrowski, S.A., Gulley, K., Briggs, E.C., & Pynoos, R.S. (2013). Psychometric properties of the UCLA PTSD Reaction Index: Part 1, *Journal of Traumatic Stress, 26* ; 1-9.

Steinlin, M., Imfeld, S., Zulauf, P., Boltshauser, E., Lövblad, K. O., RidolfiLüthy, A., Perrig, W., & Kaufmann, F. (2003). Neurological long-term sequelae after posterior fossa tumour resection during childhood. *Brain, 126*, 1998-2008.

Stevens, N.R., Gerhart, J., Goldsmith, R.E., Heath, N.M., Chesney, S.A., & Hobfoll, S.E. (2013). Emotion regulation difficulties, low social support, and interpersonal violence mediate the link between childhood abuse and posttraumatic stress symptoms, *Behavior Therapy, 44*, (1), 152-161.

Strong, V., Waters, R., Hibberd, C., Murray, G., Wall, L., Walker, J., McHugh, G., Walker, A., Sharpe, M. (2008). Management of depression for people with cancer (SMaRT oncology 1): a randomised trial. *Lancet, 372*, 40-48.

Stuber, M.L., Kazak, A.E., Meeske, K., & Barakat, L. (1998). Is post-traumatic stress a viable model for understanding responses to childhood cancer? *Child and Adolescent Psychiatric Clinics of North America, 7*, 169-182.

Sturdee, D.W. (2008). The menopausal hot flush--anything new?. *Maturitas, 60*, 42–9

Sullivan, M.J.L., Rouse, D., Bishop, S., & Johnston, S. (1997). Thought suppression, catastrophizing, and pain. *Cognitive Therapy and Research, 21* (5), 1573-2819.

Swann, W. B., & Read, S. J. (1981). Acquiring self-knowledge: The search for feedback that fits. *Journal of Personality and Social Psychology, 41*, 1119–1128.

Tarrier, N., Sommerfield, C., Pilgrim, H., & Faragher, B. (2000). Factors associated with outcome of cognitive-behavioural treatment of chronic post-traumatic stress disorder. *Behaviour Research and Therapy, 38,* 191-202.

Tashakkori, A. & Teddlie, C. (1998). *Mixed methodology: Combining qualitative and quantitative approaches (Vol. 46).* (L. Bickman, & D. Rog, Eds.) Thousand Oaks, CA: SAGE.

Taylor, S.m, Thordarson, D.S., Maxfield, L., Fedoroff, I.C., Lovell, K., & Ogrodniczuk, J. (2003). Comparative efficacy, speed, and adverse effects of three PTSD treatments: Exposure therapy, EMDR, and relaxation training. *Journal of Consulting and Clinical Psychology, 71* (2), 330-338.

Tedstone, J.E. & Tarrier, N. (2003). Posttraumatic stress disorder following medical illness and

treatment. *Clinical Psychology Review, 23*, 409-48.

Terr, L.C. (1991). Childhood traumas: An outline and overview. *American Journal of Psychiatry, 148*, 10–20.

Tirch,D.D., Leahy, R.L., Silberstein, L.R., & Melwani, P.S. (2012). Emotional Schemas, Psychological Flexibility, and Anxiety: The Role of Flexible Response Patterns to Anxious Arousal. *International Journal of Cognitive Therapy, 5,* 380-391.

Thompson, S.B.N. (2011). *Psychology of Trauma: Clinical Reviews, Case Histories, Research.* Portsmouth: Blackwell-Harvard-Academic.

Thompson, S.B., Eccleston, L., Hicksh, T. (2011). Post-traumatic stress disorder in cancer survivors: Recognising and acknowledging the symptoms. *WebmedCentral Oncology, 2* (8): WMC002062

Tjemsland, L., Søreide, J.A., & Malt, U.F. (1996). Traumatic distress symptoms in early breast cancer I: Acute response to diagnosis. *Psycho-Oncology, 5*, 1-8.

Tull, M.T., Barrett, H.M., McMillian, E.S., & Roemer, L. (2007). A preliminary investigation of the relationship between emotion regulation difficulties and posttraumatic stress symptoms. *Behaviour Therapy, 38*, 303-313.

Tull, M. T., Jakupcak, M., McFadden, M. E., & Roemer, L. (2007). The role of negative affect intensity and the fear of emotions in posttraumatic stress symptom severity among victims of childhood interpersonal violence. *Journal of Nervous and Mental Disease, 195* (7), 580–587.

Turk, C.L., Heimberg, R.G., Luterek, J.A., Menin, D.S., & Fresco, D.M. (2005). Emotion dysregulation in generalized anxiety disorder: A comparison with social anxiety disorder. *Cognitive Therapy and Research, 5*, 89–106.

Turner, C. D., Rey-Casserly, C., Liptak, C. C., & Chordas, C. (2009). Late Effects of Therapy for Pediatric Brain Tumor Survivors. *J Child Neurol 24*: DOI: 10.1177/0883073809341709

Vachon, M. (2006) Psychosocial distress and coping after cancer treatment. *American Journal of Nursing, 106*, 26-31.

van der Kolk, B.A., & Greenberg, M.S. (1987). Psychobiology of the trauma response: Hyperarousal, constriction, and addiction to trauma re-exposure. In van der Kolk, B.A. (Ed), *Psychological Trauma*. (pp. 63-88). Washington, DC: American Psychiatric Press.

van der Kolk, B.A. & van der Hart, O. (1989). Pierre Janet & the breakdown of adaptation in psychological trauma. *American Journal of Psychiatry, 146* (12), 1530-1540.

van der Kolk, B.A., Weisaeth, L., & van der Hart, O. (2007). History of trauma in psychiatry. In van der Kolk, B.A., McFarlane, A.C., & Weisaeth, L. (Eds), *Traumatic Stress: The Effects of Overwhelming Experience on Mind, Body, and Society.* (pp.47-74). New York: Guilford Press.

Van Etten, M.L. & Taylor, S. (1998). Comparative efficacy of treatments for post-traumatic stress disorder: A meta-analysis. *Clinical Psychology & Psychotherapy, 5*, 126-144.

Varra, A.A. & Follette, V.M. (2004). ACT with posttraumatic stress disorder. In S.C. Hayes, & K.D. Strosahl (Eds)., *A Practical Guide to Acceptance and Commitment Therapy.* (pp. 133-152). New York: Springer.

Vogt, D.S., King, D.W., & King, L.A. (2007). Risk pathways for PTSD. Making sense of the literature. In Friedman, M.J., Keane, T.M., & Resick, P.A. (Eds), *Handbook of PTSD: Science & Practice* (pp. 99-115). New York: Guilford Press.

Walker, J., Sawhney, A., Hansen, C.H., Ahmed, S., Martin, P., Symeonides, S., Murray, G., & Sharpe, M. (2013). Treatment of depression in adults with cancer: a systematic review of randomized controlled trials. Psychological Medicine, 19, 1-11.

Watson, J.C. & Bedard, D.L. (2006). Clients' Emotional Processing in Psychotherapy: A Comparison between Cognitive –Behavioural and Process-Experiential Therapies. *Journal of Consulting and Clinical Psychology 74*, 152-159.

Watson, C.G., Juba, M.P., Manifold, V., Kucala, T., & Anderson, P.E.D. (1991). The PTSD Interview: Rationale, description, reliability, and concurrent validity of a DSM-III-based technique. *Journal of Clinical Psychology, 47,* 179-188.

Weathers, F. W., Huska, J. A., & Keane, T. M. (1991). *PCL-C for DSM-IV*. Boston: National Center for PTSD, Behavioral Science Division.

Wefel, J.S. Lenzi, R., Theriault, R.L. Davis, R.N., Meyers, C.A. (2004). The cognitive sequelae of standard dose adjuvant chemotherapy in women with breast carcinoma: results of a prospective, randomized, longitudinal trial. *Cancer, 100*, 2292-2299.

Weiss, D.S. (2007). The Impact of Event Scale: Revised. *Cross-Cultural Assessment of Psychological Trauma and PTSD International and Cultural Psychology Series,* 219-238.

Weiss, N.H., Tull, M.T., Davis, L.T., Dehon, E.E., Fulton, J.J., & Gratz, K.L. (2012). Examining the association between emotion regulation difficulties and probable posttraumatic stress disorder within a sample of African Americans. *Cognitive Behaviour Therapy, 41* (1), 5-14.

Wells, A. (1995). Meta-cognition and worry: A cognitive model of generalized anxiety disorder. *Behavioural and Cognitive Psychotherapy, 23*, 301–320.

Whelton, J.W. (2004). Emotional processes in psychotherapy: evidence across therapeutic modalities. *Clinical Psychology and Psychotherapy, 11*, 58-71.

Whitaker, K.L., Brewin, C.R., Watson, M. (2008). Intrusive cognitions and anxiety in cancer patients. *Journal of Psychosomatic Research, 64*, 509-517.

Widows, M.R., Jacobsen, P.B., & Fields, K.K. (2000). Relation of psychological vulnerability factors to posttraumatic stress disorder symptomatology in bone marrow transplant recipients. *Psychosomatic Medicine,* 62**,** 873–882.

Wilkins, C., Baker, R., Bick, D., & Thomas, P. (2009). Emotional processing in childbirth: a predictor of postnatal depression? *British Journal of Midwifery*, *17,* 154-159.

World Health Organisation (2014). *Cancer Country Profiles - United Kingdom*. Retreived from http://www.who.int/cancer/country-profiles/gbr_en.pdf?ua=1.

Yehuda, R. (2004). Risk and resilience in posttraumatic stress disorder. *Journal of Clinical Psychiatry, 65*, 29-36.

Zigmond, A.S. & Snaith, R.P. (1983). The hospital anxiety and depression scale. A*cta Psychiatr Scand, 67*, 361-370.

Zlotnick, C., Davidson, J., Shea, MT., & Pearlstein, T. (1996). The Validation of the Davidson Trauma Scale (DTS) in a sample of survivors of childhood sexual abuse. *Journal of Nervous and Mental Disease, 184*, 255-257.

Zlotnick, C., Johnson, J., & Kohn, R. (2006). Epidemiology of trauma, post-traumatic stress disorder (PTSD) and co-morbid disorders in Chile. *Psychol Med, 36*, 1523-1533.

Glossary

ACT	Acceptance and Commitment Therapy
AD	Adjustment Disorder
ABMT	Autologous Bone Marrow Transplant
ANCOVA	Analysis of Co-Variance
ANOVA	Analysis of Variance
APA	American Psychiatric Association
APMS	Adult Psychiatric Morbidity Survey
ASD	Acute Stress Disorder
BMT	Bone Marrow Transplant
CAPS	Clinician-Administered PTSD Scale
CARE	Case Report Guidelines
CBT	Cognitive Behavioural Therapy
CMA	Comprehensive Meta-Analysis
CR	Conditional Risk
CR-PTSD	Cancer-related PTSD
DAS	Detroit Area Survey
DOH	Department of Health
DRT	Dual Representation Theory
DSM	Diagnostic and Statistical Manual
DSSI	Delusions Symptoms States Inventory
DTS	Davidson's Trauma Scale
DV	Dependent Variable
EP	Emotional Processing
EPS	Emotional Processing Scale
EPT	Emotional Processing Therapy
FOR	Fear of Recurrence
GAD	Generalized Anxiety Disorder
HADS-[A/D]	Hospital Anxiety and Depression Scale – [Anxiety / Depression Subscales]
ITEC	Integrative Training of Emotional Competencies Program
IV	Independent Variable
LESS	Leahy Emotional Schema Scale
MANOVA	Multi-variate Analysis of Variance
MDD	Major Depressive Disorder
MMR	Mixed-methods research
NCCMH	National Collaborating Centre for Mental Health
NCS	National Co-Morbidity Survey
NHS	National Health Service
NICE	National Institute for Health and Care Excellence
NVVRS	National Vietnam Veteran Readjustment Study

NPV/	Negative Predictive Value
PCL-C	Post-traumatic Stress Disorder Checklist – Civilian Version
PD	Panic Disorder
PDS	Post-Traumatic Stress Diagnostic Scale
PPV	Positive Predictive Value
PRISMA	Preferred Reporting Items for Systematic Reviews and Meta-Analyses
PTSD	Post-traumatic Stress Disorder
R&D	Research and Development Committee
RBH	Royal Bournemouth Hospital
RCT	Randomized Clinical / Controlled Trial
REC	NHS Research Ethics Committee
RTA	Road Traffic Accident
SAM	Situationally Accessible Memory
SCID	Structured Clinical Interview for DSM
SED	Sequential Explanatory Design
SEM	Structural Equation Modelling
SES	Socio Economic Status
SRT	Stress Response Theory
SS-PTSD	Subsyndromal PTSD
THQ	Trauma History Questionnaire
VAM	Verbally Accessible Memory
WHO	World Health Organisation

Appendices

Appendix A – Additional Declaration and Disclosure

The contents of this thesis are entirely the authors own work. However, it should be noted that from 2008-2012, all of data from the PTSD Clinic (Study 2) was collected by Professor Roger Baker and Nurse Consultant in Oncology Lin Purandare prior to the commencement of this PhD. All Clinic data from 2013 onwards was collected by the author of this thesis. Professor Roger Baker and Lin Purandare are included as authors on the second paper (see below), but this is because they were the assessing clinicians who founded the clinic and collected the initial data. They had no input into the analysis or writing of the article, and therefore no conflict of interest, Several sections of this work have also been presented or published prior to this submission. First, Chapters 1.1-1.4 were presented as lectures at a conference for therapists and counsellors accredited by the British Association of Behavioural and Cognitive Psychotherapists (BABCP) in July 2013. An abstract based on Chapter 3.1 was presented and published at the February 2014 conference in Preston, UK, for the British Psycho-Oncological Society (BPOS), and published in the proceedings for the American Society of Clinical Oncology (ASCO) for the May 2014 conference in Chicago, USA. As such, this abstract is published in *Psycho-Oncology* and the *Journal of Clinical Oncology*. The full study (Chapter 3.1.) is currently in press for full publication in *Psycho-Oncology*. The second study (Chapter 3.2) is also submitted to *Psycho-Oncology* and under review. Finally, the second case study (Chapter 4) has been cited and used in the published Instruction Manual for the Emotional Processing Scale (EPS-25; Baker, 2014).

Appendix B – Publications and Conference Presentations

1. **Baker, R., Gale, L., Abbey, G., & Thomas, S.** (2013). Emotional processing therapy for post-traumatic stress disorder. *Counselling Psychology Quarterly, 26, 362-385*.

This article was written, submitted, and published during the PhD timeframe, *but is not submitted as part of the PhD*, although the topic is of particular relevance to the development of this thesis.

2. **Abbey, G., Thompson, S.B.N., Hickish, T., & Heathcote, D.** (2014). A meta-analysis of prevalence rates and moderating factors for cancer-related post-traumatic stress disorder. *Psycho-Oncology,* DOI: 10.1002/PON.3654

This article was based on the first study from this thesis. It was submitted in November 2013 and accepted in July 2014. The accepted submission (prior to typesetting) is presented on the following page. All figures and tables in this article are also presented in this thesis, and thus all references to said tables and figures corresponded to table and figure numbers in this thesis.

Similarly, in an attempt to disseminate information both nationally and internationally, the meta-analysis was submitted as an abstract and presented by poster for two conferences: 1) The Annual Meeting for the **British Psychosocial Oncology Society (BPOS**), in Preston, UK, February 2014. **"**The emotional impact of cancer"; and 2), **The American Society of Clinical Oncology (ASCO)** 50th Annual General Meeting, in Chicago, Illinois, USA, May 2014.

3. **Abbey, G., Thompson, S.B.N., Hickish, T., Baker, R. & Purandare, L**. (submitted). Differential diagnosis of cancer-related adjustment disorder (CR-AD) and cancer-related post-traumatic stress disorder (CR-PTSD) in breast and colorectal cancer survivors. *Journal of Psychosocial Oncology*

Following the acceptance of the first study, this article (which was based on the second study in this thesis), was submitted to *Psychosocial Oncology* to follow-up on the discussion points raised in the previous publication. This article only included the cross-sectional analysis of patients and not the evaluation of Prof. Baker's new therapeutic approach. This was to prevent a conflict of interest. Please also note that neither RB or LP were involved in the analysis or the writing of this article, but they are credited due to their excellent work in developing and managing the clinic, and the initial data collection.

A meta-analysis of prevalence rates and moderating factors for cancer-related post-traumatic stress disorder.

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Abstract

Objective: Systematic reviews highlight a broad range of CR-PTSD prevalence estimates in cancer survivors. This meta-analysis was conducted to provide a prevalence estimate of significant CR-PTSD symptoms and full diagnoses to facilitate the psychological aftercare of cancer survivors.

Methods: A systematic literature search was conducted for studies using samples of cancer survivors using validated clinical interviews and questionnaires to assess the prevalence of CR-PTSD (k=25, n=4189). Prevalence estimates were calculated for each assessment method using random-effects meta-analysis. Mixed-effects meta-regression and categorical analyses were used to investigate study-level moderator effects.

Results: Studies using the Posttraumatic Stress Disorder Checklist – Civilian Version (PCL-C) yielded lower event rates using cut-off (7.3%, 95% CI=4.5-11.7, k=10) than symptom cluster (11.2%, 95% CI=8.7-14.4, k=9). Studies using the Structured Clinical Interview (SCID) yielded low rates for lifetime (15.3%, 95% CI=9.1-25, k=5) and current CR-PTSD (5.1%, 95% CI=2.8-8.9, k=9). Between-study heterogeneity was substantial (I^2=54-86%). Studies with advanced-staged samples yielded significantly higher rates with PCL-C cluster scoring (p=.05), and when assessing current CR- PTSD on the SCID (p=.05). The effect of mean age on current PTSD prevalence met significance on the SCID (p=.05). SCID lifetime prevalence rates decreased with time post-treatment (R^2= .56, p<.05).

Discussion: The cancer experience is sufficiently traumatic to induce PTSD in a minority of cancer survivors. Post-hoc analyses suggest that those who are younger, are diagnosed with more advanced disease, and recently completed treatment, may be at greater risk of PTSD. More research is needed to investigate vulnerability factors for PTSD in cancer survivors.

Key words: Cancer, Oncology, PTSD, prevalence, meta-analysis, DSM-5

1. Introduction

Systematic reviews show that long-term cancer survivorship is accompanied by co-morbid depression, anxiety, and symptoms of posttraumatic stress disorder (PTSD) [1-4]. The PTSD Field Trials for the Diagnostic and Statistical Manual, Fourth Edition (DSM-IV) [5] revealed that 22% of cancer survivors present with lifetime cancer-related PTSD (CR-PTSD) [6], endorsing cancer diagnosis and treatment as a traumatic stressor. This inspired research into the prevalence and presentation of CR-PTSD in cancer populations [8].

According to DSM-IV [5], PTSD can develop when an individual experiences a traumatic event that threatens their psychological and/or physical integrity (Criterion A1), and they react with helplessness or horror (Criterion A2). They typically re-experience the memories, sensations, and emotions from the event through sensory flashbacks, and nightmares (Criterion B), avoid trauma reminders, can be emotionally numb (Criterion C), anxious, irritable, and hyper-vigilant (Criterion D). These symptoms are pathological when their duration is more than one month post-trauma (Criterion E), and causes socio-occupational impairments (Criterion F). Using DSM-IV criteria, studies to-date identified lifetime rates from 5% [8] to 35% [9]. However, the recent publication of the DSM-5 [10] challenges this position. The new trauma criterion states "…a life-threatening…or debilitating medical condition is not necessarily considered a traumatic event. Medical incidents that qualify as traumatic events involve sudden, catastrophic events [e.g., waking during surgery, anaphylactic shock]" (p.274).

A recent article by Kangas [11] discussed how the new PTSD diagnostic criteria would affect the psychological aftercare of individuals who have completed cancer treatment - a population we will define as *cancer survivors*. Consequently, a PTSD diagnosis is less likely to be given to those who present with PTSD symptoms, in favour of DSM-5's adjustment disorder (AD). AD is a diagnosis given to individuals who either a) present with subsyndromal PTSD, or b), experience the complete PTSD syndrome in response to an event that is not considered traumatic [10]. Kangas [11] argues that relatively few DSM-IV studies investigated the rates of AD *and* PTSD in cancer samples, and thus could not determine if PTSD was the primary disorder. This casts doubt on the

appropriateness of PTSD as a diagnosis for cancer survivors, but emphasises the need to correctly diagnose a cancer survivor who presents with trauma/stress-related symptoms.

It does not, however, address the needs of oncology specialists to know the proportion of patients who would likely develop PTSD or AD, or of the factors contributing to their presentation. In systematic reviews to-date [1-4], efforts to achieve a prevalence estimate have been impeded due to the substantial variability in rates, which is often attributed to extreme between-study heterogeneity in assessment methods (e.g. questionnaire *v* clinical interview), assessment points (post-diagnosis or post-treatment), and sample characteristics such as severity of disease, or mean age [12].

Firstly, many studies use different methods of estimating (or diagnosing) PTSD. The most obvious example of this is the use of clinical interviews, such as the 'gold standard' Clinician-Administered Posttraumatic Stress Scale (CAPS) [13], or the Structured Clinical Interview (SCID) [14] which yield a full DSM-IV PTSD diagnosis (Criteria A-F), versus screening questionnaires such as the PTSD Checklist–Civilian Version (PCL-C) [15], which assess symptom severity (Criteria B-D). Though the PCL-C has been criticised for not establishing the fulfilment of PTSD Criterion A [16] it has been administered to cancer patients and survivors in the context of experiencing cancer as a traumatic event (Criterion A1). It can also assess PTSD symptoms over the past month (Criterion E), and has been used frequently to assess PTSD symptom severity and provide a *tentative* PTSD diagnosis. Andrykowski et al., [17] validated the PCL-C against the SCID [14] and its factor structure has been corroborated in cancer survivors [18-19]. Therefore the PCL-C is arguably a useful questionnaire to assess PTSD symptom severity, and has sufficient sensitivity and specificity to screen for a probable PTSD diagnosis. Nevertheless, like other screening questionnaires, the PCL-C has been known to inflate PTSD prevalence rates due to a) the endorsement of PCL-C items that are confounded by disease symptoms [20]; b) the inability to assess functional impairment (Criterion F); and c) whether cut-off scores are used, or using a symptom cluster method by imitating the DSM-IV-TR PTSD diagnostic criteria [7]. The proximity of psychological assessment (either by questionnaire or structured interview) to the end of diagnosis *or* treatment may also contribute to prevalence. Evidence for this is equivocal; some studies have shown that PTSD persists even 20 years after the cancer experience [21], whereas others reveal no relationship between PTSD symptom severity and time since diagnosis [6, 22-25] or time since treatment [26-27].

Secondly, heterogeneity may emerge from sample characteristics. Younger age at cancer diagnosis is associated with more severe PTSD symptoms [16, 18, 23, 24, 26, 28-31] although a some studies have not found a significant association [21, 25, 27]. Recent meta-analyses have demonstrated that younger samples of medical trauma survivors are associated with higher PTSD prevalence rates [32]. The disease characteristics of each study sample may also contribute. Gurevich et al., [1] argued that many studies which report no relationship between disease severity and CR-PTSD prevalence rates are actually skewed towards early disease stages [6, 16, 26, 28-30]. However, samples that are more evenly distributed report significant effects of disease staging on prevalence rates [9, 27, 33, 34]. In order to counter this heterogeneity, it has been argued that future studies of PTSD in cancer populations would benefit from larger, broader, samples [35]. Therefore, a meta-analysis of studies reporting CR-PTSD prevalence statistics was conducted in order to find an empirical estimate of the proportion of adult cancer survivors who will present with a) clinically significant PTSD symptoms, or b) a full diagnosis of PTSD under DSM-IV criteria. Our aims were:

1. To establish a mean PTSD prevalence estimate for CR-PTSD in adult cancer survivors, with regards to the use of diagnostic interview methods and screening questionnaires. This will provide an estimate of the presentation of full-PTSD, and caseness-level PTSD symptoms indicating the need for a full assessment, respectively.

2. Establish what percentage of variance in prevalence estimates can be explained by between-study heterogeneity, with a focus on disease stage sample characteristics, mean sample age, and time since diagnosis or treatment. This will provide evidence for patient variables that may be risk factors for the development of a PTSD.

2. Method

Search Strategy

Seven online databases (MEDLINE, PsycARTICLES, PsycINFO, CINAHL Complete, CINAHL Plus, Academic Search, and E-Journals) were searched systematically. Studies published between 1994 (the publication of the DSM-IV) until 11

June 2013 were included. The search terms *(cancer) AND (PTSD) AND (prevalence)* were used for every database. It was decided to adopt such broad terms to capture as many studies as possible that are focused on the number of CR-PTSD cases in their sample. Dissertations were included in the search, but case studies, and studies that were unavailable in English, were excluded. Systematic reviews from 1999 onwards concerning CR-PTSD in survivors of adult cancers were examined, and any relevant references from these reviews that were not returned in online databases were abstracted. The following inclusion/exclusion criteria were compiled by the primary and secondary authors following an investigation of issues identified in the above systematic reviews, and were agreed on by consensus of all the authors.

Inclusion & Exclusion Criteria

Articles were included if they met these criteria: 1) they were conducted with, and specify, a sample of cancer *patients* (those who are in treatment) *or survivors* (those who have completed treatment). Note that the search strategy does not specify any particular cancer due to the systematic reviews arguing the scarcity of evidence for the effect of disease variables on CR-PTSD prevalence rates; 2) they provide PTSD prevalence estimates (that is, the number of people in the sample who meet caseness); 3) the prevalence statistics are in reference to *cancer diagnosis and treatment* as the traumatic stressor (CR-PTSD); 4) they provide summary statistics on a) disease stages in the sample, b) gender, c) mean age of sample, and d) mean time post-diagnosis *or* post-treatment; 5) they use both/either a structured clinical interview such as the CAPS [13], SCID [14], Watson's PTSD Inventory [36], or a validated screening questionnaire that is based on DSM-IV PTSD criteria such as the PCL-C [15], or PTSD Reaction Index, [37]; and 6) use cross-sectional, or longitudinal/prospective, methods. Articles were excluded if they 1) used the Impact of Events Scale as a *standalone measure* of probable PTSD [38]; 2) specifically used samples of adult survivors of childhood cancers, or 3) used the same sample as another included study.

Study Selection

After the removal of duplicates entries, the full texts of the remaining records were read by the primary author. Studies that clearly met exclusion criteria (e.g. did not use cancer samples, or used the IES as a standalone PTSD measure) were excluded by the primary author. All authors read the remaining full-text articles, and selected from the

remaining studies those that they believed met inclusion criteria, and discrepancy between authors was resolved by full consensus.

Data Abstraction

All prevalence figures, sample characteristics, and study methods, were abstracted onto a spread sheet for review by the primary author, re-checked four times to ensure accurate data abstraction, and a random sample of the included studies were independently checked by a clinical psychology postgraduate (Table 1). There was full agreement. Though most of the data are easily abstracted in its presented form (e.g. mean age, gender, event rates, assessment method), some statistics were calculated manually. For example, in order to assess the moderating effect of disease stage distribution on CR-PTSD prevalence rates, a summary statistic on the skew of the sample distribution had to be used. Given that many studies are skewed towards low disease stages, the mode (0, 1, 2, 3, or 4) was used to depict the disease stage that was most frequent in each sample. There was k=4 studies that were bimodal; in these cases, all bimodal distributions were characterised by either I-II, II-III, or III-IV, so given the nature of cancer-stage progression, the mean was used (1.5, 2.5, and 3.5, respectively). In some cases, the sample size reported in abstracts did not reflect those in the final analysis, so our abstracted *n*'s reflected only those participants who completed the study. Finally, some authors reported time since diagnosis or treatment using different units (days/weeks rather than months). All studies were converted to the number of months. In all cases where it was reported in days or weeks, a month was treated as 30 days, and weeks were multiplied by seven, and then divided by 30 to get the number of months.

A-priori Statistical Methods & Analysis

All statistical analyses and graphical presentations were conducted using Comprehensive Meta-analysis (CMA) [39]. Prevalence statistics were depicted using the *event rate*. Ninety-five percent confidence intervals were calculated within the CMA software using the sample size (*n*) and standard error. When prospective studies were included in the analysis, the Time 1 measurements (being temporally associated with study-level moderators such as time since diagnosis and/or treatment) were used to calculate the event rates. In cases where the T1 measurements were not post-treatment, the T2 prevalence estimates (and the mean time post-treatment) were used. This was the case for one study only [34]. All meta-analyses were conducted under a random-effects

model due to the substantial methodological heterogeneity identified in systematic reviews. Mixed-effects meta-regression was used to examine the association between continuous variables such as time since diagnosis *or* treatment end and mean age of sample, on CR-PTSD prevalence rates. Categorical analyses were conducted on studies where samples were characterised by either early (I-II) or advanced (III-IV) disease stages, and also on population studies (USA/Canada, UK/Europe, and Eastern Countries). Finally, publication bias was assessed using funnel plots. These plots depict the spread of prevalence rates according to a) interviews and screeners, b) symptom cluster and cut-off scoring for screening questionnaires, and c) current v lifetime CR-PTSD for clinical interviews. In order to calculate the variance contribution of statistically significant study-level moderators on PTSD event rates, we calculated the R^2 statistic using the following formula: [R^2=1–($T^2_{unexplained}$ / T^2_{total})].

3. Results

Literature Search & Study Characteristics

We identified 257 articles. After duplicates were removed, 109 articles remained. A combined total of 138 studies were recovered from online databases and systematic reviews. Figure 2 depicts the PRISMA search strategy [40]. Table 3 presents the abstracted prevalence data and study/sampling characteristics for the final dataset (k=25). The aggregated sample was n=4189, of which 88% were female, with Mystakidou et al., [41] being the only study to represent both genders. In terms of methodology, k=18 studies were cross-sectional, and k=7 were longitudinal/prospective. The majority of studies were conducted in the United States (k=18), with one in Japan, five in Europe & the UK, and one in Israel. Though there were a range of cancers included, 21 studies used exclusively female breast cancer samples, with one study using ovarian [42], one gynaecological [34] and two inclusive of breast cancer, but with the addition of others [6, 41]. Due to the low n for cancers other than breast, an analysis of the moderating effects of cancer type could not be conducted. Therefore a separate analysis was conducted on breast cancer alone, as this is the most frequently studied cancer population. Also, the moderating effects of gender were not analysed due to the predominance of females over males in the final dataset.

FIGURE 2 HERE

CR-PTSD Prevalence Rates

The results of the meta-analysis are available in Table 4. The table presents prevalence statistics for the whole pool of cancer studies and for breast cancer alone, grouped by assessment strategy, from clinical interview methods (SCID, CAPS, Watson's PTSD Inventory), to questionnaires (PCL-C, PDS, PTSD RI, PTSD Scale). Separate statistics are provided for the PCL-C and SCID given their use in the majority of studies. See supplemental online figures 2-6 for forest and funnel plots.

All cancer types: Studies that used the PCL-C yielded lower event rates using the cut-off method [7.3%, 95% CI = 4.5-11.7, *k*=10] compared to the symptom cluster method [11.2%, 95% CI=8.7-14.4, *k*=9]. Studies using the SCID alone yielded higher lifetime diagnoses [15.3%, 95% CI=9.1-24.7, *k*=5] than current diagnoses [5.1 %, 95% CI=2.8-8.9, *k*=9]. Studies using a clinical interview method yielded a combined event rate of 6.4% [95% CI=4.1-9.9, *k*=12] for current CR-PTSD, and 12.6% [95% CI=7.5-20.7, *k*=7] for lifetime CR-PTSD. Studies that used a cut-off score screening method used the PCL-C exclusively. All other screening tools used a symptom cluster method, and yielded similar prevalence estimates to the PCL-C cluster method [13.8%, 95% CI=9.5-19.6, *k*=11]. No comparison could be done between interview methods, or between screening tools, because only the PCL-C and the SCID was used more than once. Though the point estimates for each assessment method are comparable, each is characterised by considerable between-study heterogeneity across cancer samples [Table 4; I^2=54-86%].

TABLE 3 HERE

TABLE 4 HERE

Breast cancer: Studies that used the PCL-C yielded lower event rates using the cut-off method [6.4%, 95% CI=4.2-9.7, *k*=9] compared to the symptom cluster method [11.2%, 95% CI=8.7-14.4, *k*=9]. These figures are similar to those found across cancers, although the cut-off event rate for breast cancer alone is 1% less. The rates for the symptom cluster method here are the same for breast cancer as for all cancers. Studies using the SCID alone yielded lower but nevertheless comparable event rates for current CR-PTSD [4.1%, 95% CI=2-8.5, *k*=7]. Much higher rates were found for lifetime CR-PTSD [14.2%, 95% CI=7.7-24.9, *k*=4]. Studies that used a clinical interview method yielded a combined event rate of 5.8% [95% CI=3.3-10, *k*=10] for current CR-PTSD, and

11.5% [95% CI=6.3-20.1, k=6] for lifetime CR-PTSD. Studies using the cut-off scoring method used the PCL-C exclusively (see above results). All other screening tools used a symptom cluster method, and yielded remarkable similar prevalence estimates to the PCL-C cluster method [12.1%, 95% CI=9.3-15.7, k=10]. Similar between-study heterogeneity was observed for breast cancer samples [I^2=54.4-81.5%].

Sources of Heterogeneity

Mean sample age, mean time since diagnosis, and mean time since end of treatment were entered into a meta-regression for their variance contribution to CR-PTSD prevalence rates. Early or advanced modal disease staging and population of origin (USA, UK & Europe, Japan, and Israel) were analysed categorically to assess group differences in prevalence.

Time post-diagnosis was not significant for any analyses. Time post-treatment was not significant when using the SCID, PCL-C, or the questionnaire method, but was significant when including other clinical interviews to assess lifetime CR-PTSD [Q_{model}(1)=3.84, $T^2_{unexplained}$=0.22 k=3, p=.05], with CR-PTSD event rates decreasing when time since treatment increases, and a variance contribution of 56% [R^2=.56, p<.05]. The contribution of mean sample age to prevalence was not significant for breast cancer samples, or when using the cut-off and cluster screening methods, the clinical interview method, or when using the SCID to assess lifetime CR-PTSD in all cancers. However, it was significant when using the SCID to assess current CR-PTSD [Q_{model} (1)=.43, p=.05].

Disease stage was significant when using the SCID to assess current CR-PTSD, [Q(1)=10.23, p=.05]. Studies characterised by early stages (I-II) yielding markedly lower prevalence rates (4.2%, 95% CI=2.1-8.1, k=8, n=737], than those with advanced stage (III-IV) disease [11.4%, 95% CI=9.6-13.5, k=1, n=989]. This was also true for the interview method [Q(1)=6.07, p=.01], with the only difference in effect coming from the additional interview methods other than the SCID in the early-stage group [5.7%, 95% CI=3.4-9.6, k=11, n=970]. Finally, the use of the questionnaire method with cluster scoring also yielded a significant difference [Q(1)=3.71, p=.05] with early stages yielding significantly lower prevalence rates [11.6, 95% CI=8.8-13.5, k=9, n=1894], than advanced stage samples [31.3 95% CI=11.8-61, k=2, n=112]. This moderator was not significant for pure breast cancer samples.

Population was not a significant moderator when using the questionnaire method or on the PCL-C, or the SCID, alone, either in the full cancer sample, or breast cancer

samples. But, significant differences were found when using the clinical interview method to diagnose current CR-PTSD [$Q(3)=9.2$, $p=.03$], with Israel having significantly higher rates [18%, 95% CI=8.8-33.2, $k=1$] than the US [5.6%, 95% CI=2.6-11.6, $k= 8$] Europe [6%, 95% CI=2.4-14, $k=3$], or Japan [3.9%, 95% CI=1.8-8.4, $k=1$]. Population was also significant for lifetime CR-PTSD [$Q(2)=11.28$, $p=.03$], with the USA [15.4%, 95% CI=8.4-26.7. $k=5$], and Japan [14.8%, 95% CI=10-21.3, $k=1$] having higher rates than Europe [2.8%, 95% CI=1.1-7.2, $k=1$]. Population was significant in exclusively breast cancer samples when diagnosing lifetime CR-PTSD with the interview method [$Q(2)=10.83$, $p<.01$], with the UK & Europe [2.8%, 95% CI=1.1-7.2] having a significantly lower prevalence than the USA [14.2%, 95% CI=6.8-27.5 $k=4$] and Japan [14.8, 95% CI=10-21.3, $k=1$]. Similar trends in breast cancer were found when diagnosing current CR-PTSD [$Q(3)=11.43$, $p<.01$], with Israel yielding higher rates [18%, 95% CI=8.8-33.2, $k=1$] than Japan [3.9%, 95% CI=1.8-8.4, $k=1$], UK & Europe [4.1%, 95% CI=2.2-7.6, $k=2$], and USA [5.7%, 95% CI=2.5-12.6, $k=6$].

4. Discussion

This is thought to be the first meta-analysis to investigate the prevalence of CR-PTSD in cancer survivors, and the contribution of between-study heterogeneity to the fluctuation of these rates. Our data for all cancers indicated that studies using clinical interview methods yield a mean prevalence of 6.4% for current CR-PTSD, and 12.6% for lifetime CR-PTSD. Screening methods that used cluster scoring indicated a prevalence of 13.8% for clinically significant CR-PTSD symptoms. The PCL-C yielded lower event rates of clinically significant CR-PTSD symptoms using the cut-off (7.3%) compared to symptom cluster (11.2%), whereas the SCID yielded predictably higher lifetime diagnoses (15.3%) than current diagnoses (5.1%) for CR-PTSD.

These rates are similar to those found by Alter et al., [6] in the DSM-IV Field Trials (4%), Green et al., [8] and Palmer et al, [35] which identify a current CR-PTSD prevalence of 2.5-5%. Epidemiological surveys reveal the lifetime conditional risk (CR) of PTSD for women in the US general population to be between 10.4% (95% CI=8.8-11.7%) [50] to 13% (95% CI=9.9-16.1%) [51]. Nevertheless, the lifetime CR-PTSD rate for our 100% female US sample was 15.4% (95% CI=8.4-26.7%), which is higher than the CR for the USA, but within the confidence intervals for Breslau's survey [51]. If 5-

12% of cancer survivors develop CR-PTSD, a trauma framework may not represent the distress experienced by most patients – as argued by Green et al., [8], Palmer et al., [35], and now the DSM-5 task force [10]. This supports Kangas' criticism that the lack of differential diagnoses in semi-structured clinical interviews challenges the validity of PTSD diagnosis [11]. It does not, however, take into account that the literature fails to record the presence/absence of discrete catastrophic events within the course of the disease that would make the cancer experience traumatic.

We recommend that clinicians consider the full range of presentations, be aware of the nature of cancer as a stressor, and assess for discrete events that occurred during the treatment to warrant a PTSD diagnosis; and in the absence of these, consider a diagnosis of AD. If cancer survivors meet AD criteria; this does not discount the possibility of PTSD development. The literature shows that cancer survivors present with subsyndromal symptoms [8, 47, 52], which are also associated with considerable functional impairments [26, 47]. While this too may lead clinicians to initially question the validity of a CR-PTSD diagnosis, it does not discount the possibility that these subsyndromal symptoms may be a predictor of future PTSD. DSM-5 course specifiers state that PTSD may present with *delayed expression*, where only subsyndromal criteria are met before eventually meeting full criteria at least six months post-trauma [10]. This is supported by recent meta-analyses that show delayed expression is preceded by high stress sensitivity, additional life stressors, maladaptation to continued exposure to stress, and the steady accumulation of subsyndromal symptoms from the onset of the stressor [53, 54]. These are features of cancer, and subsyndromal symptoms are also present at the beginning of cancer treatment, fluctuate and persist throughout the course of the disease, and too may reflect a delayed PTSD presentation [16]. Though rightly considered an adjustment response during treatment, this may develop into CR-PTSD if those factors prevent the cognitive processing of the illness.

In our moderator analysis, screening questionnaires yielded higher rates of CR-PTSD symptoms, than did the use of clinical interviews for current CR-PTSD. The reasons for this may be that while interviews diagnose disorder, questionnaires detect clinically significant symptoms, are not clinician-administered, and do not screen for disorders that better explain the symptoms [7]. However, the PCL-C has demonstrated its reliable sensitivity and specificity in correctly identifying clinical cases [12], but there is still debate on where to place the cut-off score [7]. The Davidson's Trauma Scale [55] can be used as an alternative to screen for CR-PTSD. It has a cut-off score which moves

depending on the prevalence of PTSD in the specific population. As this meta-analysis revealed a current CR-PTSD prevalence of 6.4%, the Davidson's cut-off for the cancer survivor population would be 47.

When lifetime CR-PTSD was assessed using the SCID, prevalence decreased with time post-treatment. This finding should be interpreted with caution. First, this trend was only observed for lifetime CR-PTSD, and second, this factor is a proxy variable for time post-trauma. Since there is no agreement as to whether the diagnosis or the treatments are the definitive traumas, investigators adopted one of the two indices; therefore, these moderators could not be combined, resulting in low statistical power. However, this finding reflects the epidemiologic trend that non-cancer PTSD sufferers do recover naturally over time, though a minority remain symptomatic after many years [50]. This has implications for the survivors, as potential cases may not be identified during routine follow-up, placing them at risk of PTSD months to years after their treatment.

Younger-aged samples were associated with higher current CR-PTSD event rates on the SCID. This trend was just significant, so these results too must be interpreted cautiously. The majority of research into this relationship concentrates on age at diagnosis – our study-level moderator was the mean age of the sample. Several of the samples were many months, or sometimes years, post diagnosis, and this likely reduced the sensitivity of our analysis – but as it addressed the impact of younger age on the prevalence rates of lifetime CR-PTSD (not on symptom severity) - it provides a singular contribution.

The final aim was to synthesise disease-stage data to establish whether it is a risk factor for PTSD following comments that skewed samples characterised the research [1]. Our post-hoc analyses suggested that advanced disease is related to an increase in CR-PTSD event rates on PCL-C and on the SCID. This is commensurate with studies that show a positive relationship between disease severity and CR-PTSD [9, 27, 33, 34], and may provide some preliminary support for this conclusion, but we advise cautious interpretation. Though the differences in event rates were significant, additional factors may have contributed. The advanced disease sample in one study [41] was from a different culture, and set in palliative care, which introduces environmental factors into the development of CR-PTSD that are uncharacteristic of early-stage cancer samples. The degrees of freedom for the early-stage group were larger than for the advanced-stage group, and reflect the abundance of earlier stage cancers in the majority of studies.

Publication Bias

The funnel plots revealed substantial biases. However, this is not the bias one would expect from FDP. Publication bias is identified by a skew in the distribution towards higher effect sizes, at the bottom of the funnel plot. The presence of this type of bias is not visually depicted in our plots.

Strengths & Limitations

There are limitations that constrain our conclusions. The findings from the moderator analysis are tentative because several findings come from retaining k=1 comparisons. This is problematic as the Q test has low power to detect heterogeneity when k is low [56]. Nevertheless, this highlights the need for concise, standardised, and transparent, reporting to facilitate future meta-analytic studies. Also, only study-level moderators were included. Many studies did not assess psychiatric history or additional life stress at the time of assessment, so intrapersonal vulnerability factors were unaccounted for. The substantial variability in reporting styles, and limitations of using CMA, may have introduced variance into analyses that is not attributable to the moderators of interest. Studies that have used questionnaires may over-inflate rates of CR-PTSD due to symptom endorsement being confounded by artefacts of cancer drugs, and medical conditions [5], and realistic fears of cancer recurrence being endorsed as the acceptance of a foreshortened future. Nevertheless, the sensitivity and specificity of the PCL-C against the SCID for CR-PTSD is sufficient to screen for those survivors who may be suffering from an adjustment disorder or CR-PTSD. Out of the studies that have used interviews, few have assessed the rates of co-morbid disorders, meaning CR-PTSD might not be the primary disorder. Also, all of the studies included in this meta-analysis used DSM-IV criteria, not the new DSM-5. Early epidemiological studies documented the inflation of PTSD prevalence due to the revised DSM-IV criteria, so PTSD prevalence may decrease due to the new DSM-5 PTSD criterion A. The strengths of this investigation are that it included 25 studies and a substantial patient pool across several populations. This affords our analysis generalizability that the individual studies could not achieve [35]. Precision was enhanced by including data for symptoms anchored to the experience of adult cancer. This does not account for differential diagnoses, but does account for stress-related symptoms due to an extreme stressor. In this case, the findings may reflect the proportion of survivors who meet criteria for AD *and* PTSD. This is more clinically useful than focusing on PTSD alone.

Future Research

If the cancer experience is not generally traumatic, then the fact that CR-PTSD does emerge in a significant minority of survivors suggests other factors affect adjustment and the cognitive processing of the illness. It has been argued that 'less traumatic' stressors can be expected to trigger PTSD in vulnerable individuals who have limited resources to manage their response to stress [57], and that the insufficiency of some events to meet PTSD criterion A may reflect failure to consider the role of personal stress reactivity [58-59], and the effect of allostatic load [59]. As such, there is less information on variables that influence this adjustment. Variables such as social support and/or constraints on talking about the illness [28, 30, 21, 61-65], emotion regulation strategies such as dissociation [22], emotional suppression and avoidance [49, 66], and rumination [67] have been identified as predictors of PTSD symptoms in cancer populations. Most recently, emotional processing styles have been implicated in post-natal depression [68], and PTSD [69-70]. Given the protracted nature of cancer, it is possible that social variables and their relationship to beliefs about the experiencing and expressing of emotions may influence cognitive processing over the course of disease, and predict the emergence of CR-PTSD. This necessitates further investigation the role of these variables in multiple oncology services both nationally and internationally. But first, in order to assess the risk of CR-PTSD in this population, it may be necessary to report, or make readily available, the raw data on CR-PTSD event rates by a) disease stage b) cancer type, c) treatment regimens, d) any traumatic events during the course of the disease, e) age at diagnosis, and f) report the mean time of assessment in relation to time post diagnosis *and* time post treatment.

Conclusions

Prevalence rates from questionnaires reveal a minority of cancer survivors with present with clinically significant symptoms due to cancer. In these cases, we recommend that oncology specialists recognise the possibility of CR-PTSD and refer their patient to mental health services. Prevalence rates from clinical interviews show that a minority of cancer survivors meet (or have previously met) full DSM-IV criteria for CR-PTSD after the conclusion of treatment, and that those who are younger, diagnosed with more advanced disease, or recently completed treatment, may be at greater risk of CR-PTSD. Methodological heterogeneity prohibits robust conclusions about the expected prevalence of CR-PTSD as a primary disorder, but does provide some clinical justification for the

diagnosis of a CR-PTSD in a minority of cases. Given the release of the DSM-5, we recommend caution in diagnosing CR-PTSD, but advise further investigation into whether traumatic occurrences were experienced during the course of the disease.

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Conflicts of Interest

None declared.

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5. References

1. Gurevich M, Devins GM, Rodin GM. Stress response syndromes and cancer: conceptual and assessment issues. *Psychosomatics,* 2002; **43** : 259-281. DOI: 10.1176/appi.psy.43.4.259
2. Kangas M, Henry JL, Bryant RA. Posttraumatic stress disorder following cancer. A conceptual and empirical review. *Clinical Psychology Review,* 2002; **22** : 499-524. DOI: 10.1016/S0272-7358(01)00118-0
3. Koutrouli N, Anagnostopoulos F, Potamianos G. Posttraumatic stress disorder and posttraumatic growth in breast cancer patients: A systematic review. *Women & Health,* 2012; **52** : 503-516. DOI: 10.1080/03630242.2012.679337
4. Smith MY, Redd WH, Peyser C, Vogl D. Post-traumatic stress disorder in cancer: A review. *Psycho-Oncology,* 1999; **8** : 521-537. DOI: 10.1002/(SICI)1099-1611(199911/12)8:6<521::AID-PON423>3.0.CO;2-X
5. American Psychiatric Association. *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author, 1994.

6. Alter CL, Pelcovitz D, Axelrod A, Goldenberg B, Harris H, Meyers B, Grobois B, Mandel F, Septimus A, Kaplan S. Identification of PTSD in cancer survivors. *Psychosomatics,* 1996; **37** : 137 – 143. DOI: 10.1016/S0033-3182(96)71580-3
7. Thompson SB, Eccleston L, Hickish T. Post-traumatic stress disorder in cancer survivors: Recognising and acknowledging the symptoms. *WebmedCentral Oncology,* 2011; **2** : WMC002062
8. Green BL, Rowland JH, Krupnick JL, Epstein SA, Stockton P, Stern NM, Spertus IL, Steakley C. Prevalence of posttraumatic stress disorder in women with breast cancer. *Psychosomatics,* 1998; **39** : 102-111. DOI: 10.1016/S0033-3182(98)71356-8
9. Mundy EA, Blanchard EB, Cirenza E, Gargiulo J, Maloy B, Blanchard CG. Posttraumatic stress disorder in breast cancer patients following autologous bone marrow transplantation or conventional cancer treatments. *Behaviour Research & Therapy,* 2000; **38** : 1015–1027. DOI: 10.1016/S0005-7967(99)00144-8
10. American Psychiatric Association. *Diagnostic and statistical manual of mental disorders* (5th ed.). Washington, DC: Author, 2013.
11. Kangas M. DSM-5 trauma and stress-related disorders: implications for screening for cancer-related stress. *Frontiers in Psychiatry, 2013*. DOI: 10.3389/fpsyt.2013.00122
12. Andrykowski MA, Cordova MJ, Studts JL, Miller TW. Posttraumatic Stress Disorder after treatment for breast cancer: prevalence of diagnosis and use of the PTSD Checklist—Civilian Version (PCL-C) as a screening instrument. *Journal of Consulting & Clinical Psychology, 1998*; **66** ; 586–590. DOI: 10.1037/0022-006X.66.3.586
13. Blake DD, Weathers FW, Nagy LM, Kaloupek DG, Gusman FD, Charney DS, Keane TM.. The development of a clinician-administered PTSD scale. *Journal of Traumatic Stress,* (1995); **8** ; 75-90. DOI: 10.1002/jts.2490080106
14. First MB, Spitzer RL, Gibbon M, Williams JBW**.** Structured Clinical Interview for DSM-IV-TR Axis I Disorders, Research Version, Non-patient Edition. (SCID-I/NP) New York: Biometrics Research, New York State Psychiatric Institute, 2002.
15. Blanchard EB, Jones-Alexander J, Bucklet TC, Forneris CA. Psychometric properties of the PTSD Checklist (PCL). *Behavioral Research & Therapy, 1996;* **34** ; 669-673. DOI: 10.1016/0005-7967(96)00033-2

16. Andrykowski MA, Cordova MJ, McGrath PC, Sloan DA, Kenady DE. Stability and change in posttraumatic stress disorder symptoms following breast cancer treatment: a 1-year follow-up. *Psycho-Oncology,* 2000; **9** : 69-78. DOI: 10.1002/(SICI)1099-1611(200001/02)9:1<69::AID-PON439>3.0.CO;2-R
17. Andrykowski MA, Cordova MJ, Studts JL, Miller TW. Posttraumatic Stress Disorder after treatment for breast cancer: prevalence of diagnosis and use of the PTSD Checklist—Civilian Version (PCL-C) as a screening instrument. *Journal of Consulting & Clinical Psychology, 1998*; **66** ; 586–590. DOI: 10.1037/0022-006X.66.3.586
18. Cordova MJ, Studts JL, Hann DM, Jacobsen PB, Andrykowski MA. Symptom structure of PTSD following breast cancer. *Journal of Traumatic Stress,* 2000; **13** : 301-319. DOI: 10.1023/A:1007762812848
19. Smith MY, Redd W, DuHamel K, Vickberg SJ, Ricketts P. Validation of the PTSD Checklist-Civilian Version in survivors of bone marrow transplantation. *Journal of Traumatic Stress,* 1999; **12** : 485–499. DOI: 10.1023/A:1024719104351
20. Shelby RA, Golden-Kreutz DM, Andersen BL**.** Mismatch of posttraumatic stress disorder (PTSD) symptoms and DSM-IV symptom clusters in a cancer sample: exploratory factor analysis of the PTSD Checklist-Civilian Version. *Journal of Traumatic Stress,* 2005; **18** : 347-357. DOI: 10.1002/jts.20033
21. Kornblith AB, Herndon JE, Weiss RB, Zhang C, Zuckerman EL, Rosenberg S, Mertz M, Payne D, Massie MJ, Holland JF, Wingate P, Norton L, Holland J C. Long-term adjustment of survivors of early-stage breast carcinoma, 20 years after adjuvant chemotherapy. *Cancer,* 2003; **98** : 679-689. DOI: 10.1002/cncr.11531
22. Kangas M, Henry JL, Bryant RA. Predictors of posttraumatic stress disorder following cancer. *Health Psychology,* 2005a; 24 : 579-585. DOI: 10.1037/0278-6133.24.6.579
23. Levine EG, Eckhardt J, Targ E. Change in post-traumatic stress symptoms following psychosocial treatment for breast cancer. *Psycho-Oncology,* 2005; **14** : 618-635. DOI: 10.1002/pon.882
24. Morrill EF, Brewer NT, O'Neill SC, Lillie SE, Dees EC, Carey LA, Rimer BK. The interaction of post-traumatic growth and post-traumatic stress symptoms in predicting depressive symptoms and quality of life. *Psycho-Oncology,* 2008; **17** : 948-953. DOI: 10.1002/pon.1313

25. Pitman RK, Lanes DA, Williston SK, Guillaume JL, Metzger LJ, Gehr GM, Orr SP. Psychophysiologic assessment of posttraumatic stress disorder in breast cancer patients. *Psychosomatics,* 2001; **42** : 133-140. DOI: 10.1176/appi.psy.42.2.133
26. Cordova MJ, Andrykowski MA, Kenady DE, McGrath PC, Sloan DA, Redd WH. Frequency and correlates of posttraumatic-stress-disorder-like symptoms after treatment for breast cancer. *Journal of Consulting and Clinical Psychology,* 1995; **63** : 981-986. DOI: 10.1037/0022-006X.63.6.981
27. Jacobsen PB, Widows MR, Hann DM, Andrykowski MA, Kronish LE, Fields KK. Posttraumatic stress disorder symptoms after bone marrow transplantation for breast cancer. *Psychosomatic Medicine,* 1998; **60** : 366-371. DOI: 10.1097/00006842-199805000-00026
28. Cordova MJ, Giese-Davis J, Golant M, Kronenwetter C, Chang V, Spiegel D. Breast cancer as trauma: posttraumatic stress and posttraumatic growth. *J Clin Psychol Med Settings,* 2007; **14** : 308-319. DOI: 10.1007/s10880-007-9083-6
29. Epping-Jordan JE, Compas BE, Osowiecki DM, Oppedisano G, Gerhardt C, Primo K, Krag DN**.** Psychological adjustment in breast cancer: processes of emotional distress. *Health Psychology,* 1999; **18** : 315-326. DOI: 10.1037/0278-6133.18.4.315
30. Green BL, Krupnick JL, Rowland JH, Epstein SA, Stockton P, Spertus I, Stern, N. Trauma history as a predictor of psychologic symptoms in women with breast cancer. *Journal of Clinical Oncology,* 2000; **18** : 1084-1093.
31. Koopman C, Butler LD, Classen C, Giese-Davis J, Morrow GR, Westerndorf J, Banerjee T, Spiegel D. Traumatic stress symptoms among women with recently diagnosed primary breast cancer. *Journal of Traumatic Stress,* 2002; **15** : 277-287. DOI: 10.1023/A:1016295610660
32. Edmondson D, Richardson S, Falzon L, Davidson KW, Ali M. Posttraumatic stress disorder prevalence and risk of recurrence in acute coronary syndrome patients: a meta-analytic review. *PLoS ONE,* 2012; **7** : e38915. DOI: 10.1371/journal.pone.0038915.
33. Boyer BA, Bubel D, Jacobs SR, Knolls ML, Harwell VD, Goscicka M, Keegan A. Posttraumatic stress in women with breast cancer and their daughters. *The American Journal of Family Therapy,* 2002; **30** : 323-338. DOI: 10.1080/01926180290033466

34. Posluszny DM, Edwards RP, Dew MA, Baum A. Perceived threat and PTSD symptoms in women undergoing surgery for gynaecologic cancer or benign conditions. *Psycho-Oncology,* 2011; **20** : 783-787. DOI: 10.1002/pon.1771.
35. Palmer SC, Kagee A, Coyne JC, DeMichele A. Experience of trauma, distress, and posttraumatic stress disorder among breast cancer patients. *Psychosomatic Medicine,* 2004; **66** : 258-264. DOI: 10.1097/01.psy.0000116755.71033.10.
36. Watson CG, Juba MP, Manifold V, Kucala T, Anderson PED. The PTSD Interview: Rationale, description, reliability, and concurrent validity of a DSM-III-based technique. *Journal of Clinical Psychology,* 1991; **47** ; 179-188. DOI: 10.1002/1097-4679(199103)47:2<179::AID-JCLP2270470202>3.0.CO;2-P.
37. Steinberg AM, Brymer MJ, Kim S, Ghosh C, Ostrowski SA, Gulley K, , Briggs, EC, Pynoos, RS. Psychometric properties of the UCLA PTSD Reaction Index: Part 1, *Journal of Traumatic Stress,* 2013; **26** ; 1-9. DOI: 10.1002/jts.21755.
38. Joseph S. Psychometric evaluation of Horowitz's Impact of Event Scale: A review. *Journal of Traumatic Stress*. 2000; **13** : 101–113. DOI: 10.1023/A:1007777032063.
39. Comprehensive Meta-Analysis (Version 2) [Computer software]. Englewood, NJ: Biostat.
40. Moher D, Liberati A, Tetzlaff J, Altman DG, The PRISMA Group. Preferred Reporting Items for Systematic Reviews and Meta-Analyses: The PRISMA Statement. *BMJ*, 2009; **339** ; b2535, DOI: 10.1136/bmj.b2535.
41. Mystakidou K, Parpa E, Tsilika E, Panagiotou I, Roumeliotou A, Galanos A, Gouliamos A. Traumatic experiences of patients with advanced cancer. *Journal of Loss and Trauma,* 2012; **17** : 125-136. DOI: 10.1080/15325024.2011.595296.
42. Goncalves V, Jayson G, Tarrier N. A longitudinal investigation of posttraumatic stress disorder in patients with ovarian cancer. *Journal of Psychosomatic Research,* 2011; **70** : 422-431. DOI: 10.1016/j.jpsychores.2010.09.017.
43. Mehnert A, Koch U. Prevalence of acute and post-traumatic stress disorder and comorbid mental disorders in breast cancer patients during primary cancer care: A prospective study. *Psycho-Oncology,* 2007; **16** : 181-199. DOI: 10.1002/pon.1057.
44. Mehnert A, Koch, U. Psychological comorbidity and health-related quality of life and its association with awareness, utilization, and need for psychosocial support in a cancer register-based sample of long-term breast cancer survivors. *Journal of Psychosomatic Research,* 2008; **64** : 383-391. DOI: 10.1016/j.jpsychores.2007.12.005.

45. Luecken LJ, Dausch B, Gulla V, Hong R, Compas BE. Alterations in morning cortisol with PTSD in women with breast cancer. *Journal of Psychosomatic Research,* 2004; **56** : 13-15. DOI: 10.1016/S0022-3999(03)00561-0.
46. Matsuoka Y, Inagaki M, Sugawara Y, Imoto S, Akechi T, Uchitomi Y. Biomedical and psychosocial determinants of intrusive recollections in breast cancer survivors. *Psychosomatics,* 2005; **46** : 203-211. DOI: 10.1176/appi.psy.46.3.203.
47. Shelby RA, Golden-Kreutz DM, Andersen BL. PTSD diagnoses, subsyndromal symptoms, and co morbidities contributing to impairments for breast cancer survivors. *Journal of Traumatic Stress,* 2008; **21** : 165-172. DOI: 10.1002/jts.20316.
48. Gandubert C, Carriere I, Escot C, Soulier M, Hermes A, Boulet P, Ritchie K, Chaudieu I. Onset and relapse of psychiatric disorders following early breast cancer: a case-control study. *Psycho-Oncology,* 2009; **18** : 1029-1037. DOI: 10.1002/pon.1469.
49. Amir M, Ramati A. Post-traumatic symptoms, emotional distress, and quality of life in long-term survivors of breast cancer: a preliminary research. Anxiety Disorders, 2002; **16** ; 191-206 DOI: 10.1016/S0887-6185(02)00095-6.
50. Kessler RC, Sonneha A, Bromet E, Hughes M, Nelson CB. Posttraumatic stress disorder in the National Co-morbidity Survey. *Archives of General Psychiatry* 1995; **52** : 1048-1060. DOI: 10.1001/archpsyc.1995.03950240066012.
51. Breslau N, Kessler RC, Chilcoat HD, Schultz LR, Davis GC, Andreski P. Trauma and posttraumatic stress disorder in the community: the 1996 Detroit Area Survey of Trauma. *Archives of General Psychiatry,* 1998; **55** : 626-632. DOI: 10.1001/archpsyc.1995.03950240066012.
52. Guglietti CL, Rosen B, Murphy KJ, Laframboise S, Dodge J, Ferguson S, Katz J, Ritvo P. Prevalence and predictors of posttraumatic stress in women undergoing an ovarian cancer investigation. *Psychological Services,* 2010; **7** : 266-274. DOI: 10.1037/a0020338.
53. Andrews B, Brewin, CR, Stewart L, Philpott R, Hejdenberg J. Comparison of immediate-onset and delayed-onset posttraumatic stress disorder in military veterans. *Journal of Abnormal Psychology*, 2009; **118** ; 767-777. DOI: 10.1037/a0017203.
54. Smid GE, Mooren TT, van der Mast RC, Gersons BP, Kleber RJ. Delayed posttraumatic stress disorder: systematic review, meta-analysis, and metaregression

analysis of prospective studies. *J Clin Psychiatry.* 2009; **70** ; 1572Y1582. DOI: 10.4088/JCP.08r04484.

55. Davidson JRT, Book SW, Colket JT, Tupler LA. Assessment of a new self-rating scale for post-traumatic stress disorder. *Psychological Medicine: A Journal of Research in Psychiatry and the Allied Sciences,* 1997; **27** ; 153-160.

56. Gavaghan DJ, Moore RA, McQuay HJ. An evaluation of homogeneity tests in meta-analyses in pain using simulations of individual patient data. *Journal of the International Association for the Study of Pain*, 2000; **85** ; 415-424. DOI: /10.1016/S0304-3959(99)00302-4.

57. Marshall RD, Amsel L, Suh EJ. Response to McNally and Breslau (2008). *American Psychologist, 2008*; **63** ; 283–285.

58. King DW, King LA, Fairbank JA, Keane TM, Adams G. Resilience-recovery factors in post-traumatic stress disorder among female and male Vietnam veterans: Hardiness, postwar social support, and additional stressful life events. *Journal of Personality and Social Psychology*, 1998; **74** ; 420-434. DOI: 10.1037/0022-3514.74.2.420.

59. King DW, King LA, Foy DW, Keane TM, Fairbank JA. Posttraumatic stress disorder in a national sample of female and male Vietnam veterans: Risk factors, war-zone stressors, and resilience-recovery variables. *Journal of Abnormal Psychology*, 1999; **108** ; 164-170. DOI: 10.1037/0021-843X.108.1.164.

60. Brewin CR, Lanius RA, Novac A, Schnyder U, Galea S. Reformulating PTSD for DSM-V: Life after criterion A. *Journal of Traumatic Stress*, 2009, **22**, 366-373. DOI: 10.1002/jts.20443.

61. Jacobsen PB, Sadler IJ, Booth-Jones M, Soety E, Weitzner MA, Fields KK. Predictors of post-traumatic stress disorder symptomatology following bone marrow transplantation for cancer. *Journal of Consulting and Clinical Psychology,* 2002; **70** : 235-240. DOI: 10.1037/0022-006X.70.1.235.

62. Andrykowski MA, Cordova MJ. Factors associated with PTSD symptoms following treatment for breast cancer: test of the Andersen model. *Journal of Traumatic Stress,* 1998; 11 : 189–203. DOI: 10.1023/A:1024490718043.

63. Lepore SJ, Helgeson VS. Social constraints, intrusive thoughts, and mental health after prostate cancer. *Journal of Social and Clinical Psychology,* 1998; **17** : 89–106. DOI: 10.1521/jscp.1998.17.1.89

64. Manne SL. Intrusive thoughts and psychological distress among cancer patients: the role of spouse avoidance and criticism. *Journal of Consulting and Clinical Psychology,* 1999; **67** : 539–546. DOI: 10.1037//0022-006X.67.4.539.

65. Widows MR, Jacobsen PB, Fields KK. Relation of psychological vulnerability factors to posttraumatic stress disorder symptomatology in bone marrow transplant recipients. *Psychosomatic Medicine,* 2000; **62** : 873–882. DOI: 10.1097/00006842-200011000-00018.

66. Hampton MR, Frombach I. Women's experience of traumatic stress in cancer treatment. *Healthcare for Women International*, 2000; **21** ; 67-76. DOI: 10.1080/073993300245410.

67. Chan MWC, Ho SMY, Tedeschi RG, Leung CWL. The valence of attentional bias and cancer-related rumination in posttraumatic stress and posttraumatic growth among women with breast cancer. *Psycho-Oncology*, 2011; **20** ; 544-552. DOI: 10.1002/pon.1761.

68. Wilkins C, Baker R. Bick D. Thomas P. Emotional processing in childbirth: a predictor of postnatal depression? *British Journal of Midwifery*, 2009; **17** ; 154-159. DOI: http://dx.doi.org/10.12968/bjom.2009.17.3.40077

69. Baker R. *Understanding Trauma: How to Overcome Post-Traumatic Stress*. Lion Hudson: Oxford. 2010.

70. Baker R, Gale L, Abbey G, Thomas S. Emotional processing therapy for post-traumatic stress disorder. *Counselling Psychology Quarterly,* 2013; **26** ; 362-385. DOI: 10.1080/09515070.2013.816840

Appendix C – Courses and Lectures

1. I was approached by the Dorset Association of Counsellors and Psychotherapists (DACAP) to present the first four chapters of my literature review as part of a continual professional development conference for counsellors and psychotherapists. This was presented at the Emotional Processing Workshop, Kingston Maurward, Dorset, UK, in a two lecture format: 1) "Applying emotional processing to clinical problems"; and 2), "Post-traumatic stress disorder: epidemiology, risk factors, and clinical theory" (July 2013).

2. I also delivered seminars and workshops on gaining clinical experience for the Bournemouth University Masters of Science in Foundations of Clinical Psychology (January 2011- January 2013)

3. I have also done marking on a range of undergraduate subjects and delivered seminars on Critical Thinking and Forensic Psychology (Sept- Dec 2013).

4. I attended two training conferences at the Charlie Waller Institute, University of Reading, UK, focused on emotion-centred adjunctive approaches to treating psychopathology. The first conference was called *Self-Compassion and Self-Esteem.* The second conference *was Cognitive-Behavioural and Emotional Processing Therapies for Generalized Anxiety Disorder* (2013).

5. I also attended a teaching course designed to train post-graduates to assess and mark undergraduate assignments, and an advanced statistics module (2011).

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Appendix D – Ethics

This appendix contains the ethics approval letters from the NHS London REC – Dulwich, and the Research and Development (R&D) committee at Royal Bournemouth Hospital, and also the patient information sheets and consent forms used for Study 4.

The Royal Bournemouth and Christchurch Hospitals
NHS Foundation Trust

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Bournemouth
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United Kingdom
BH7 7DW

Tel: 01202 303626
www.rbch.nhs.uk

Mr Gareth Abbey
119a Bournemouth Road
Parkstone
Poole
BH14 9HR

26/09/2012

Dear Mr. Abbey,

Reference:	**A process evaluation of the effect of Emotional Processing Therapy [EPT] on symptom reduction and emotional processing on cancer survivors with Post-Traumatic Stress Disorder [PTSD].**
REC reference:	**12/LO/0236**
CLRN ID:	**n/a**

I am pleased to inform you that this project has now received approvals from all parties and that you now have formal permission to start.

Please see the Terms and Conditions for undertaking research at the Trust at:
http://dorsetresearch.org/docs/drc/TC_for_research_within_DRC.pdf.

Please let me know when you officially start and I would be grateful for a progress report annually.

Good luck with the study,

R. M. Chapman

Dr R. M. Chapman
Head of Research

NHS
Health Research Authority

NRES Committee London - Dulwich
Room 4W/12, 4th Floor
Charing Cross Hospital
Fulham Palace Road
London W6 8RF
Telephone: 020 3311 7227
Facsimile: 020 3311 7280

Mr Gareth Abbey
Post Graduate Researcher
Bournemouth University
119a Bournemouth Road
Parkstone
Poole BH14 9HR

01 March 2012

Dear Mr Abbey

Study title: **A process evaluation of the effect of Emotional Processing Therapy [EPT] on symptom reduction and emotional processing on cancer survivors with Post-Traumatic Stress Disorder [PTSD].**
REC reference: **12/LO/0236**

The Proportionate Review Sub-committee of the NRES Committee London - Dulwich reviewed the above application on 01 February 2012.

Ethical opinion

The following issues were discussed with you.

A. EPT is only available as a research treatment at present and hasn't been evaluated elsewhere. Consequently – for patients declining consent to the research, it was felt more appropriate that they be referred to the local psychology teams where they could get NICE approved treatment.
B. The PTSD clinic has been operating since 2008 and deals with the whole range of trauma. Those patients not fulfilling criteria for PTSD would be referred elsewhere.
C. All patients will be re-assessed just before their treatment commences so direct comparisons can be made between active and control (waiting-list) patients.
D. Recruitment might be widened in future if demand is not great enough – you would submit a substantial amendment if this became necessary.
E. Treatment sessions are to be taped and the tapes will remain as part of the patient's medical records. The transcripts will be linked anonymised (code number linking to questionnaire results).
F. You agreed that patients ought to be warned that they might have to wait for treatment. In addition, if their distress became more acute they would be referred for treatment to the local services (which in Bournemouth are apparently very responsive and easily accessible).

On behalf of the Committee, the sub-committee gave a favourable ethical opinion of the above research on the basis described in the application form, protocol and supporting documentation, subject to the conditions specified below.

Ethical review of research sites

The favourable opinion applies to all NHS sites taking part in the study, subject to management permission being obtained from the NHS/HSC R&D office prior to the start of the study (see "Conditions of the favourable opinion" below).

Conditions of the favourable opinion

The favourable opinion is subject to the following conditions being met prior to the start of the study.

Management permission or approval must be obtained from each host organisation prior to the start of the study at the site concerned.

Management permission ("R&D approval") should be sought from all NHS organisations involved in the study in accordance with NHS research governance arrangements.

Guidance on applying for NHS permission for research is available in the Integrated Research Application System or at http://www.rdforum.nhs.uk.

Where a NHS organisation's role in the study is limited to identifying and referring potential participants to research sites ("participant identification centre"), guidance should be sought from the R&D office on the information it requires to give permission for this activity.

For non-NHS sites, site management permission should be obtained in accordance with the procedures of the relevant host organisation.

Sponsors are not required to notify the Committee of approvals from host organisations.

1. Correct the name of the Committee to the 'Proportionate Review Sub-Committee of the NRES Committee London - Dulwich'.
2. Make the following additions to the participant information sheet .
3. At the end of the first paragraph under Conduct of the study – please add:
    a. 'Your treatment might be delayed until a therapist becomes available. If this happens we will advise you how long this delay might be. If it becomes necessary for you to receive treatment more urgently we will refer you to our colleagues in the local psychology service'.
    b. At the beginning of the section 'What alternatives are there etc' – please add 'This treatment is currently only available as a research intervention'.

It is the responsibility of the sponsor to ensure that all the conditions are complied with before the start of the study or its initiation at a particular site (as applicable).

You should notify the REC in writing once all conditions have been met (except for site approvals from host organisations) and provide copies of any revised documentation with updated version numbers. Confirmation should also be provided to host organisations together with relevant documentation.

Approved documents

The documents reviewed and approved were:

Document	*Version*	*Date*
Investigator CV	Simon Thompson	
Investigator CV	Gareth Abbey	
Investigator CV	David Heathcote	
Participant Consent Form	1	01 November 2011
Participant Information Sheet	4	01 November 2011
Protocol	3	14 December 2011
Questionnaire: All Outcome Measures		
REC application		

Membership of the Proportionate Review Sub-Committee

The members of the Sub-Committee who took part in the review are listed on the attached sheet.

Statement of compliance

The Committee is constituted in accordance with the Governance Arrangements for Research Ethics Committees and complies fully with the Standard Operating Procedures for Research Ethics Committees in the UK.

After ethical review

Reporting requirements

The attached document "After ethical review – guidance for researchers" gives detailed guidance on reporting requirements for studies with a favourable opinion, including:

- Notifying substantial amendments
- Adding new sites and investigators
- Notification of serious breaches of the protocol
- Progress and safety reports
- Notifying the end of the study

The NRES website also provides guidance on these topics, which is updated in the light of changes in reporting requirements or procedures.

Feedback

You are invited to give your view of the service that you have received from the National Research Ethics Service and the application procedure. If you wish to make your views known please use the feedback form available on the website.

Further information is available at National Research Ethics Service website > After Review

12/LO/0236 **Please quote this number on all correspondence**

With the Committee's best wishes for the success of this project

Yours sincerely

pp

Dr Mike Philpot
Chair

Email: atul.patel@imperial.nhs.uk

Enclosures: *List of names and professions of members who took part in the review*

"After ethical review – guidance for researchers"

Copy to: *Professor Mark Hadfield*
Prof. Susan Clarke, Dorset HealthCare University NHS Foundation Trust

Bournemouth University

The Royal Bournemouth and Christchurch Hospitals
NHS Foundation Trust

The Royal Bournemouth Hospital
Castle Lane East
Bournemouth
Dorset
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www.rbch.nhs.uk

STUDY TITLE: A process evaluation of emotional processing therapy to treat cancer survivors with post-traumatic stress disorder

PRINICPAL INVESTIGATOR: Gareth Abbey, BSc (Hons), MSc (Hons), PGCert

SUPERVISORS: Dr Simon Thompson, Dr David Heathcote, Professor Tamas Hickish

PARTICIPANT INFORMATION SHEET

Dear patient,

We would like to invite you to take part in our research study. Before you decide we would like you to understand why the research is being done and what it would involve for you. One of our team will go through the information sheet with you and answer any questions you have. We'd suggest this should take about ten minutes. Feel free to talk to others about the study if you wish. Part 1 tells you the purpose of this study and what will happen to you if you decide to take part. Part 2 gives you more detailed information about how the study is conducted. Please do not hesitate to ask us if anything is unclear.

Part 1:

The purpose of this study

The purpose of our clinic is to provide psychological therapy to individuals who, after having survived cancer treatment and are now disease-free, began to show signs of post-traumatic stress. Post-traumatic stress in cancer survivors remains remarkably less understood than PTSD from other traumas, and so our clinic's mission statement is to not only provide treatment for this clinical group, but also to further understand how to best treat it. Our study is looking at how well a new therapy (emotional processing therapy) is able to treat post-traumatic stress in cancer survivors. We also want to improve our understanding of exactly how effective it is, and why.

Doctors who provide care for people suffering from mental health difficulties often assess the effectiveness of a therapy by how much it relieves a patient's symptoms, and this is often measured by the use of questionnaires asking you how you feel. However, what is done less often is assessing how this effect is caused.

Why have you been chosen?

You have been chosen because you have been recently advised that you are a) a survivor of cancer and its treatment, and b) because you are suffering from symptoms of post-traumatic stress.

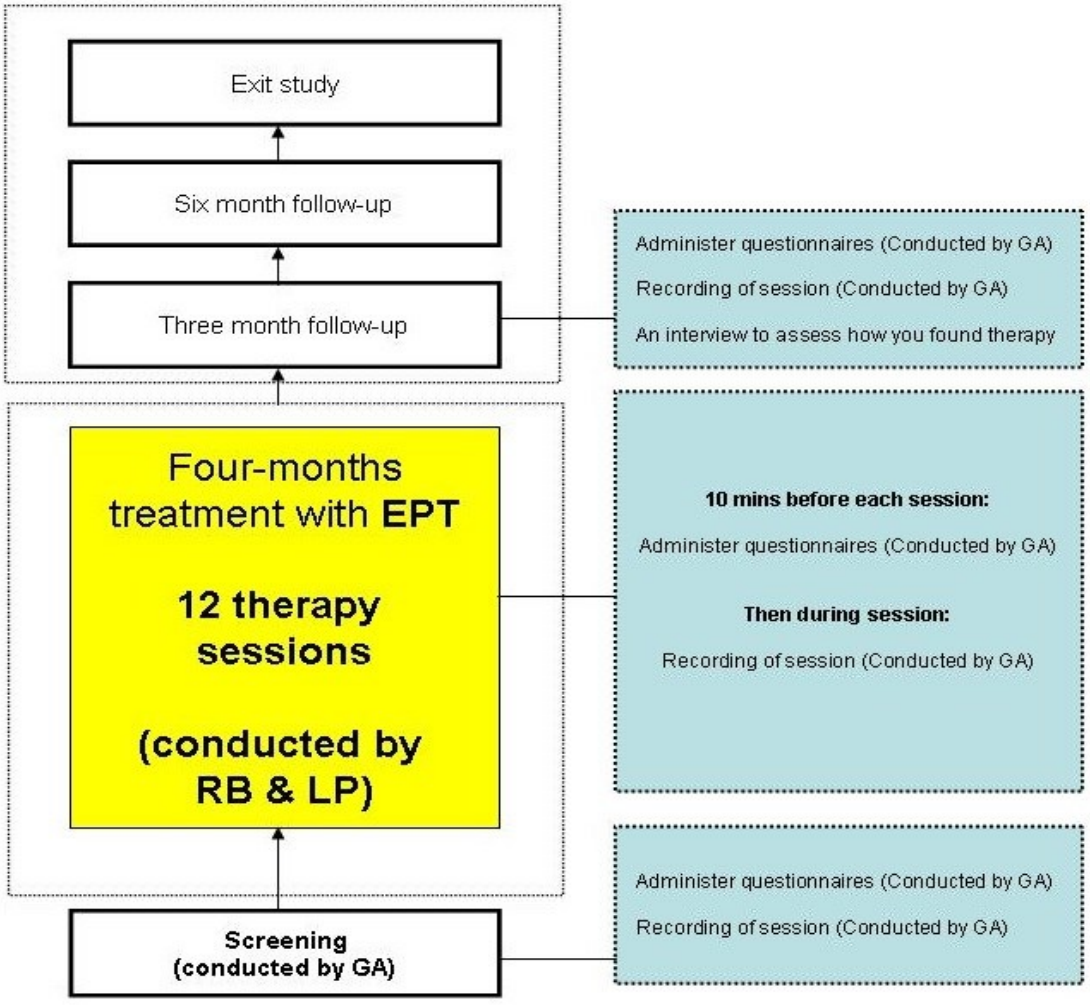
Part 2:

The conduct of this study (see figure below)

In this study, you will receive 12, 50-minute, sessions of emotional processing therapy at one-week intervals, and also one follow-up session at three months post-treatment. The twelve sessions will be spaced out over a period of four months. Before each session, Gareth Abbey (the Chief Investigator) will meet with you for ten minutes to administer some questionnaires. Also, we would like your permission record your sessions using audio and/or video to see if we can discover trends in the therapy that make it effective. An audio recorder will just be placed strategically in the room with you.

Your treatment might be delayed until a therapist becomes available. If this happens we will advise you how long this delay might be. If it becomes necessary for you to receive treatment more urgently we will refer you to our colleagues in the local psychology service.

The assessments are conducted by Professor Roger Baker (Consultant Clinical Psychologist) and the subsequent therapy sessions by Roger Baker and Lin Purandare (Consultant Nurse Oncologist). Follow-up sessions will be conducted by Gareth Abbey.



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Risks and benefits

There are no risks in taking part in this study. No interventions are being performed on you that would a) cause you any harm, or b) cause you to be receiving less care than you would if you refused consent. However, as you are taking part in assessment, therapy, or going through questionnaires, you will be asked sensitive questions, and may experience emotional distress related to your cancer experience or other difficult life experiences. This is very normal, natural, and healthy. There are, however, benefits. 1) Your inclusion would provide valuable information on how to treat post-traumatic stress more effectively, and 2), this therapy would be delivered by clinicians who have a deep clinical understanding and appreciation of your journey through cancer.

What are the alternatives to taking part?

This treatment is currently part of the Trust's normal clinical practice, so if you do not wish to take part in the study, then this will not affect your eligibility to use the service. The only difference will be that you do not undertake a screening with the Chief Investigator (Gareth Abbey), but rather skip straight to the assessment with the Consultant Clinical Psychologist, Prof. Roger Baker. Furthermore, if you refuse consent to enter into the study, whatever information gathered throughout your therapy will not be used for research purposes.

Confidentiality

All information collected about you for this study will be kept fully confidential. All personal details will be removed from any research data collected. A unique ID code will be allocated to you, this will be the only identifier used. Only you, the clinician, and the investigator, will know this ID code. A list linking ID codes to participants will be kept securely in a locked filing cabinet and access will only be granted to authorised members of the research team or regulatory authorities.

Involving your GP

If you agree to participate we will write to your GP to let him/her know that you are taking part in our study.

Your rights

We require your informed consent before we admit you into any study, and this requires us to give you the above facts before you make a decision. However, if you give us your informed consent and you enter the study, you are free to withdraw that consent and exit the study at any time, without any detriment to yourself.

What will happen to the results of this study?

This will be used as part of a PhD dissertation, with a view to be published in the relevant scientific journals. Results may also be presented at science conferences. Your data will not be identifiable. When results are available, a summary of the study can be made available to you by the principal researcher.

Who has organised and funded this research?

The research is organised by Bournemouth University as part of a PhD grant, and sponsored by Royal Bournemouth Hospital.

Who has reviewed this study?

All research in the NHS is reviewed by an independent body, known as a Research Ethics Committee. This is done to protect the interests of all research participants. This study has been reviewed and approved by the London-Dulwich REC.

What if there is a problem? What if I have any questions?

If you are concerned about any aspect of this study, please do not hesitate to speak with the researchers, who will do their best to answer your questions:

Gareth Abbey	(gabbey@bournemouth.ac.uk)
Dr Simon Thompson	(simont@bournemouth.ac.uk)
Dr David Heathcote	(dheathco@bournemouth.ac.uk)
Professor Tamas Hickish	(tamas.hickish@rbch.nhs.uk)

Thank you for taking the time to read this information sheet. If you decide to take part in the study, you will be given a copy of this information sheet and the signed consent form to keep for your own records.

Bournemouth University

The Royal Bournemouth and Christchurch Hospitals
NHS Foundation Trust

The Royal Bournemouth Hospital
Castle Lane East
Bournemouth
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United Kingdom
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Tel: 01202 303626
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STUDY TITLE: A process evaluation of emotional processing therapy to treat cancer survivors with post-traumatic stress disorder

INVESTIGATOR: Gareth Abbey, BSc (Hons), MSc (Hons), PGCert

Study Number: 1 Patient PIN: PE – …….

PARTICIPANT CONSENT FORM

1. I confirm that I have read and understand the information sheet dated 01/11/2011 (version 3.) for the above study. I have had the opportunity to consider the information, ask questions and have had these answered satisfactorily. ☐

2. I understand that participation in this study will involve taking part in therapy as normal, which will also involve filling in questionnaires every session. In addition to therapy as normal, I will also be consenting to the recording of the session either by audio or video. This study will require approximately one 60 minute assessment, 12 60 minute therapy sessions, and two 60 minute follow-up appointments. ☐

3. I am aware that participation in this study is entirely voluntary and that I can withdraw from the study at any time without giving a reason, without my medical care or legal rights being affected. ☐

4. I understand that the information that I provide will be stored securely at the clinic consistent with normal clinical practice, and that the data taken from this information will be stored anonymously on a university database. ☐

5. I understand that relevant sections of my medical notes and data collected during the study, may be looked at by individuals from Bournemouth University, from regulatory authorities or from the NHS Trust, where it is relevant to my taking part in this research. I give permission for these individuals to have access to my records. ☐

6. I agree to my GP being informed of my participation in the study. ☐

7. I agree to take part in the above study. ☐

Name of participant: …… Signed:……Date:……

Name of person taking consent::……Signed:…… Date ……

1 copy for participant, 1 to be filed in medical records, 1 to be filed in research file
Version 1 Date: 01/11/2011

Appendix E – Measures

This appendix contains information regarding the measures used in this study, and where possible, copies for viewing. Many of these measures are not included in this appendix, either due to lack of permission for those that are currently being published, or because of copyright issues. However, all measures are linked with instruction on how they can be obtained.

The Emotional Processing Scale – 25 Item (EPS-25)

The primary author and designer of the EPS-25 (Prof. Roger Baker) has not given permission to disseminate the EPS-25 digitally or in print, and it is only available for research purposes at this stage. Details on the EPS-25 can be found within the relevant citations in this thesis, but also on the relevant website: www.emotionalprocessing.org.

The Trauma History Questionnaire (THQ)

The scale below is available from the Department of Psychiatry at Georgetown University, Wisconsin, USA at the website (http://ctc.georgetown.edu/toolkit).

TRAUMA HISTORY QUESTIONNAIRE

The following is a series of questions about serious or traumatic life events. These types of events actually occur with some regularity, although we would like to believe they are rare, and they affect how people feel about, react to, and/or think about things subsequently. Knowing about the occurrence of such events, and reactions to them, will help us to develop programs for prevention, education, and other services. The questionnaire is divided into questions covering crime experiences, general disaster and trauma questions, and questions about physical and sexual experiences.

For each event, please indicate (circle) whether it happened, and if it did, the number of times and your approximate age when it happened (give your best guess if you are not sure). Also note the nature of your relationship to the person involved, and the specific nature of the event, if appropriate.

Crime-Related Events

			If Yes	
			# of Times	Approx. Age
1.	Has anyone ever tried to take something directly from you by using force or the threat of force, such as a stick-up or mugging?	No Yes	_____	_____
2.	Has anyone ever attempted to rob you or actually robbed you (i.e. stolen your personal belongings)?	No Yes	_____	_____
3.	Has anyone ever attempted to or succeeded in breaking into your home when you weren't there?	No Yes	_____	_____
4.	Has anyone ever tried to or succeeded in breaking into your home while you <u>were</u> there?	No Yes	_____	_____

General Disaster and Trauma

5.	Have you ever had a serious accident at work, in a car or somewhere else? <u>If yes</u>, please specify _____	No Yes	_____	_____

			If Yes # of Times	If Yes Approx. Age
6.	Have you ever experienced a natural disaster such as a tornado, hurricane, flood, major earthquake, etc., where you felt you or your loved ones were in danger of death or injury? If yes, please specify _____	No Yes	_____	_____
7.	Have you ever experienced a "man-made" disaster such as a train crash, building collapse, bank robbery, fire, etc., where you felt you or your loved ones were in danger of death or injury? If yes, please specify	No Yes	_____	_____
8.	Have you ever been exposed to dangerous chemicals or radioactivity that might threaten your health?	No Yes	_____	_____
9.	Have you ever been in any other situation in which you were seriously injured? If yes, please specify _____	No Yes	_____	_____
10.	Have you ever been in any other situation in which you feared you might be killed or seriously injured? If yes, please specify _____	No Yes	_____	_____
11.	Have you ever seen someone seriously injured or killed? If yes, please specify who _____	No Yes	_____	_____

			If Yes	
			# of Times	Approx. Age
12.	Have you ever seen dead bodies (other than at a funeral) or had to handle dead bodies for any reason?	No Yes	_____	_____
	If yes, please specify _____			
13.	Have you ever had a close friend or family member murdered, or killed by a drunk driver?	No Yes	_____	_____
	If yes, please specify relationship (e.g.mother, grandson,etc.)_____ _____			
14.	Have you ever had a spouse, romantic partner, or child die?	No Yes	_____	_____
	If yes, please specify relationship_____			
15.	Have you ever had a serious or life-threatening illness?	No Yes	_____	_____
	If yes, please specify _____			
16.	Have you ever received news of a serious injury, life-threatening illness or unexpected death of someone close to you?			
	If yes, please indicate	No Yes	_____	___
	_____ _____			
17.	Have you ever had to engage in combat while in military service in an official or unofficial war zone?	No Yes	_____	____
	If yes, please indicate where. _____			

Physical and Sexual Experiences

			If Yes	
			Was it repeated?	Approx. how often & what Age(s)
18.	Has anyone ever made you have intercourse, oral or anal sex against your will? If yes, please indicate nature of relationship with person (e.g. stranger, friend, relative, parent, sibling)_____	No Yes	_____	_____
19.	Has anyone ever touched private parts of your body, or made you touch theirs, under force or threat? If yes, please indicate nature of relationship with person (e.g. stranger, friend, relative, parent, sibling) _____	No Yes	_____	_____
20.	Other than incidents mentioned in Questions 18 and 19, have there been any other situations in which another person tried to force you to have unwanted sexual contact?	No Yes	_____	_____
21.	Has anyone, including family members or friends, ever attacked you with a gun, knife or some other weapon?	No Yes	_____	_____
22.	Has anyone, including family members or friends, ever attacked you without a weapon and seriously injured you?	No Yes	_____	_____
23.	Has anyone in your family ever beaten, "spanked" or pushed you hard enough to cause injury?	No Yes	_____	_____

			If Yes	
			Was it repeated?	Approx. how often & what Age(s)
Other Events				
24.	Have you experienced any other extraordinarily stressful situation or event that is not covered above? If yes, please specify. _____	No Yes	_____	_____

The Leahy Emotional Schema Scale (LESS)

The scale used in this thesis (see below) was the 50-item experimental version (Leahy, 2000). However, shorter versions are in the process of psychometric validation. Please see the website for the American Institute for Cognitive Therapy (http://www.cognitivetherapynyc.com/schemas.aspx) to obtain the scale and scoring instructions.

LESS

We are interested in how you deal with your feelings or emotions—for example, how you deal with feelings of anger, sadness, anxiety, or sexual feelings. We all differ in how we deal with these feelings—so there are no right or wrong answers. Please read each sentence carefully and answer each sentence—using the scale below—as to how you deal with your feelings during the past month. Put the number of your response next to the sentence.

Scale: 1=very untrue of me
2=somewhat untrue of me
3=slightly untrue of me
4=slightly true of me
5=somewhat true of me
6=very true of me

1. ____ When I feel down, I try to think about a different way to view things.
2. ____ When I have a feeling that bothers me, I try to think of why it is not important.
3. ____ I often think that I respond with feelings that others would not have.
4. ____ Some feelings are wrong to have.
5. ____ There are things about myself that I just don't understand.
6. ____ I believe that it is important to let myself cry in order to get my feelings "out".
7. ____ If I let myself have some of these feelings, I fear I will lose control.
8. ____ Others understand and accept my feelings.
9. ____ You can't allow yourself to have certain kinds of feelings---like feelings about sex or violence.
10. ____ My feelings don't make sense to me.
11. ____ If other people changed, I would feel a lot better.
12. ____ I think that there are feelings that I have that I am not really aware of.
13. ____ I sometimes fear that if I allowed myself to have a strong feeling, it would not go away.
14. ____ I feel ashamed of my feelings.
15. ____ Things that bother other people don't bother me.
16. ____ No one really cares about my feelings.
17. ____ It is important for me to be reasonable and practical rather than sensitive and open to my feelings.
18. ____ I can't stand it when I have contradictory feelings—like liking and disliking the same person.
19. ____ I am much more sensitive than other people.
20. ____ I try to get rid of an unpleasant feeling immediately.
21. ____ When I feel down, I try to think of the more important things in life---what I value.
22. ____ When I feel down or sad, I question my values.
23. ____ I feel that I can express my feelings openly.
24. ____ I often say to myself, "What's wrong with me?"
25. ____ I think of myself as a shallow person.
26. ____ I want people to believe that I am different from the way I truly feel.
27. ____ I worry that I won't be able to control my feelings.
28. ____ You have to guard against having certain feelings.
29. ____ Strong feelings only last a short period of time.
30. ____ You can't rely on your feelings to tell you what is good for you.
31. ____ I shouldn't have some of the feelings that I have.
32. ____ I often feel "numb" emotionally---like I have no feelings.
33. ____ I think that my feelings are strange or weird.
34. ____ Other people cause me to have unpleasant feelings.
35. ____ When I have conflicting feelings about someone, I get upset or confused.

36. ____ When I have a feeling that bothers me I try to think of something else to think about or to do.
37. ____ When I feel down, I sit by myself and think a lot about how bad I feel.
38. ____ I like being absolutely definite about the way I feel about someone else.
39. ____ Everyone has feelings like mine.
40. ____ I accept my feelings.
41. ____ I think that I have the same feelings that other people have.
42. ____ There are higher values that I aspire to.
43. ____ I think that my feelings now have nothing to do with how I was brought up.
44. ____ I worry that if I have certain feelings I might go crazy.
45. ____ My feelings seem to come out of nowhere.
46. ____ I think it is important to be rational and logical in almost everything.
47. ____ I like being absolutely definite about the way I feel about myself.
48. ____ I focus a lot on my feelings or my physical sensations.
49. ____ I don't want anyone to know about some of my feelings.
50. ____ I don't want to admit to having certain feelings—but I know that I have them.

The Davidson's Trauma Scale

This scale was obtained from:

Mental Health Systems, Inc.
PO Box 950
908 Niagara Falls Blvd.
North Tonawanda, NY 14120-2060
Phone: (800) 456-3003

Website: http://www.mhs.com/product.aspx?gr=cli&id=overview&prod=dts

The Hospital Anxiety and Depression Scale

This scale was obtained from this website: http://www.gl-assessment.co.uk/products/hospital-anxiety-and-depression-scale-0

The Delusions Symptoms States Inventory

This scale is no longer obtainable at this time.