I’m hurt, annoyed and see things: Anger in PTSD and the Role of Visual Imagery

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Date of Submission: 17 July 2015

Produced on archival quality paper
ABSTRACT

This research investigated the role of imagery as a mechanism underlying anger’s relationship to posttraumatic stress disorder (PTSD). Previous research has demonstrated that anger is a strong predictor of PTSD severity and comprehensive reviews have argued for the development of nuanced explanatory models describing the psychological mechanisms underlying this relationship. Research has shown that visual imagery is linked to a range of psychological phenomena and psychopathologies, the clearest case being anxiety disorders. Imagery may also have a strong role in eliciting anger. Exemplifying this is research demonstrating that imagery with angry content has a profound psychophysiological effect. Dysregulated imagery is the hallmark of PTSD and research has shown that anger in PTSD is directly related to intrusion prevalence. PTSD sufferers with high imagery control have fewer intrusions and less anger, compared to those with low imagery control. The current research program investigated these issues in three separate cross-sectional and longitudinal studies. It aimed to explore the potential moderating and mediating roles of imagery and negative thought as twin dimensions of cognition in anger in PTSD, compared to their roles in anxiety and depression. Participants in Studies One and Three comprised treatment seeking past and present police and military personnel with PTSD (n = 123 and 462 respectively). Study Two explored these issues in an analogue population of university students (n = 197). It was predicted that anger would be more strongly associated with PTSD than anxiety and depression and that imagery and thought would be differentially associated with anger compared to anxiety and depression. It was further hypothesised that the magnitude of imagery and thought and their impact on affect would increase in the context of PTSD via direct and interactive effects and that patterns of effect would vary by PTSD population and the nature of the trauma experienced. Results demonstrated that: (a) anger was strongly predictive of PTSD, (b) different facets of imagery were linked to different negative affects, (c) imagery and thought were linked to anger and mediated its relationship to PTSD, (d) thought mediated the effect of imagery in anger in PTSD and (e) the potency of imagery and thought were increased in PTSD and were reflective of the nature of the trauma involved. These findings demonstrated the powerful role played by imagery and thought in the genesis and maintenance of anger in PTSD. They provide a basis for developing a fuller description and theoretical account of the aetiology and maintenance of anger in PTSD and offer new possibilities for improvements in its treatment.
DECLARATION

Statement of Authorship

I, Anthony Francis McHugh, declare that this thesis entitled “I’m hurt, annoyed and see things: Anger in PTSD and the Role of Visual Imagery” is no more than 80,000 words in length, exclusive of tables, figures, appendices, references and footnotes.

It contains no material that has been submitted previously, in whole or part, for the award of any other academic degree or diploma. Except where otherwise indicated, this thesis is my own work.

........................................

Anthony Francis McHugh
ACKNOWLEDGEMENTS

Dedicated to the memory of Bernard, Bon, Gerard and Mark McHugh

The planning and execution of this thesis has been a long and winding road. Its completion would have been impossible without the contributions of a veritable multitude of people. I am grateful to all, but the following deserve special recognition.

Initially, I would like to acknowledge the participants involved in this research. I well remember the origins of the idea that people who are angry and traumatised do not process information in the same way as those not affected by trauma. It was 1995 and I was suddenly thrust by circumstance into delivering, for the first time in my career, anger management groups in the then Heidelberg Repatriation Hospital Veterans Psychiatry Unit PTSD Program. The programs continued and in 2005 the “Repat” initiated a treatment program for others with posttraumatic injuries. Thus I also got to know police injured by their work. Like veterans I was again struck by them as people, their dedication to service and their generosity of involvement and interest in the project. To both groups I salute you.

Enormous thanks goes to my supervision panel of Professors Mark Creamer, Glen Bates, David Forbes and Richard Kanaan. It has been daunting but enlightening to work with you individually and jointly. I have been spoiled by the richness of your knowledge. I thank you all for your group and individual efforts: Mark for your first-rank, talismanic and editorial zeal; Glen for your overwhelming support, wisdom, research genius and vision; David for sharing the dream and providing me much needed practical support, much needed reassurance and guidance; and, Richard for your erudition, humour, specialist knowledge of the mind/brain and support in my moments of “doubt and panic”.

To an extent I have stood on the shoulders of those who have come before me in trying to untangle the riddle of anger in PTSD. Here I pay tribute to Claude Chemtob and Ray Novaco and the aforementioned David Forbes.

I must also thank those whose help I was fortunate enough to acquire along the way in creation to statistical advice, data entry and manuscript layout. Here I specifically thank Clive Berechree, Pam Jury, Dirk Biddle and Meg Walter. I also thank my past and present managers, Les Potter and Louise Roufeil for the crucial, practical support given to me.

To my kith and kin and colleagues past and present, who have shown continued interest in the project, and of whom there are too many to name, I simply say thanks. I
specifically acknowledge my extended family and the “Reser boys”. The greatest thanks, of course, go to my immediate family: Robert (and Demi), Michael and Merryn - you are the pride of my life.

Above all, I thank Lyn Veronica McHugh. It was the most important day in my life, the day I met you. It is a fantastic voyage we are on. I can never adequately repay you for your continued support, encouragement and just being there for me, despite my PhD induced affects.

I have learned much about anger in PTSD through the conduct of this research and I believe I am a better scientist-practitioner for it. There remains much to know about the area, however, for to borrow from Socrates, *The more I learn, the more I learn how little I know.*
GLOSSARY OF TERMS

The following terms are used throughout this manuscript.

<table>
<thead>
<tr>
<th>Acronym</th>
<th>Description</th>
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<tbody>
<tr>
<td>ACPMH</td>
<td>Australian Centre for Posttraumatic Mental Health</td>
</tr>
<tr>
<td>ANSMHWB</td>
<td>Australian National Survey of Mental Health and Well-Being</td>
</tr>
<tr>
<td>CV</td>
<td>Contemporary Veteran</td>
</tr>
<tr>
<td>DSM</td>
<td>Diagnostic and Statistical Manual of Mental Disorders of the American Psychiatric Association</td>
</tr>
<tr>
<td>DSM III</td>
<td>Diagnostic and Statistical Manual of Mental Disorders of the American Psychiatric Association, Third Edition</td>
</tr>
<tr>
<td>DSM III-R</td>
<td>Diagnostic and Statistical Manual of Mental Disorders of the American Psychiatric Association, Third Edition, Revised</td>
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<tr>
<td>DSM IV</td>
<td>Diagnostic and Statistical Manual of Mental Disorders of the American Psychiatric Association, Fourth Edition</td>
</tr>
<tr>
<td>DSM IV-TR</td>
<td>Diagnostic and Statistical Manual of Mental Disorders of the American Psychiatric Association, Fourth Edition, Revised</td>
</tr>
<tr>
<td>DSM 5</td>
<td>Diagnostic and Statistical Manual of Mental Disorders of the American Psychiatric Association, Fifth Edition</td>
</tr>
<tr>
<td>OEI</td>
<td>Operation Enduring Freedom</td>
</tr>
<tr>
<td>OIF</td>
<td>Operation Iraqi Freedom</td>
</tr>
<tr>
<td>FAH</td>
<td>Frustration Aggression Hypothesis</td>
</tr>
<tr>
<td>GAD</td>
<td>Generalised Anxiety Disorder</td>
</tr>
<tr>
<td>IED</td>
<td>Intermittent Explosive Disorder</td>
</tr>
<tr>
<td>INTERFET</td>
<td>International Force East Timor</td>
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<tr>
<td>MDD</td>
<td>Major Depressive Disorder</td>
</tr>
<tr>
<td>NVVRS</td>
<td>National Vietnam Veterans Readjustment Study</td>
</tr>
<tr>
<td>PTSD</td>
<td>Post Traumatic Stress Disorder</td>
</tr>
<tr>
<td>TMN</td>
<td>Trauma Memory Network</td>
</tr>
<tr>
<td>TRMH</td>
<td>Trauma Related Mental Health</td>
</tr>
<tr>
<td>TRMHFD</td>
<td>Trauma Related Mental Health Disorder(s)</td>
</tr>
<tr>
<td>TSRD</td>
<td>Trauma and Stressor-Related Disorder(s)</td>
</tr>
<tr>
<td>UN</td>
<td>United Nations</td>
</tr>
<tr>
<td>USA</td>
<td>United States of America</td>
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<tr>
<td>VV</td>
<td>Vietnam Veteran</td>
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CHAPTER 1: ANGER, POST TRAUMATIC STRESS DISORDER AND ANGER-IN-PTSD: AN OVERVIEW

The fiercest anger of all, the most incurable, is that which rages in the place of dearest love

Medea, Euripides

This research investigates the nature of anger in the context of posttraumatic stress disorder (PTSD). Its overall objective is to illuminate the central importance of dysfunctional anger to PTSD. Accordingly, this thesis proceeds with several key propositions in mind. Initially, it argues that anger and PTSD share an important relationship, such that anger is best understood as a key initiating and driving-factor for a substantial proportion of PTSD presentations. In support of this proposition, it notes the emerging consensus that anger is central to better understanding PTSD and draws on research which records that, as anger intensifies, PTSD becomes more symptomatic and difficult to remediate. It holds the view that it is anger’s relationship with PTSD that is critical, rather than traumatisation per se. That view of anger in PTSD is supported by reviews (see McHugh, Forbes, Bates, Hopwood & Creamer, 2012), meta-analyses (e.g., Olatunji, Ciesielski & Tolin, 2010; Orth & Wieland, 2006) and large population-based studies (e.g., Lasko, Gurvits, Kuhne, Orr & Pitman, 1994) demonstrating that anger does not have the same intrinsic relationship with other trauma-related mental health disorders, like those pertaining to mood and anxiety. Recently, the clinical literature has demonstrated the acuity of the problem of anger in PTSD in military personnel recently returned from combat duties (see Elbogen et al., 2010; Forbes, McHugh & Chemtob, 2013; Morland, Love, Greene & Rosen, 2012), while illustrating that anger is a continuing problem across a range of PTSD-affected populations.

It contends that the relationship of anger and PTSD and variations in that relationship are less-than-adequately explained by the extant PTSD, anger and anger in PTSD literatures. It reviews factors that may account for the manifestation and course of anger in PTSD, anger’s effect on PTSD, the variable presence of anger in the context of PTSD and the overall relationship between the two phenomena. It consequently asserts that improved explanatory theories of anger in PTSD are required.

This assertion is supported by recent critical reflection on the importance of anger’s relationship to PTSD. Over the last 20 years, calls have been continually made for
an increased investigative focus on anger in PTSD and mechanisms underlying it (see Chemtob, Novaco, Hamada, Gross & Smith, 1997; Owens, Chard & Cox, 2008; Morland, Love, Mackintosh, Greene, & Rosen, 2012; Orth & Wieland, 2006; Schutzwohl & Maercker, 2000). Most recently, it has been argued that not only is more research into the nature of anger in PTSD and its treatment required, but research that promotes a more nuanced understanding of the relationship between PTSD and anger and aggression (Hellmuth, Stappenbeck, Hoerster & Jakupcak, 2012).

Pursuant to such suggestions, in a second major objective, this research examines the utility of an explanatory model of anger in PTSD. That model relates to the operation and effect of visual imagery (hereafter typically referred to as imagery) in anger in PTSD, where imagery is understood as the quasi-perceptual, subjectively-influenced, cognitive representation and recollection of perceptual experience in working memory in the absence of the originating stimulus (Kosslyn, 2005a).

Referred to as the hallmark of PTSD (McNally, 2004), imagery has been shown to discriminate PTSD from other trauma-related mental health disorders. PTSD may reasonably be understood as a problem of disordered visual imagery, for although mental imagery can be experienced in any sensory domain (i.e., sight, sound, touch, taste and smell), visual imagery is the most commonly reported form of imagery generally and predominates in PTSD (Ehlers et al., 2002; McNally, 2004).

The intrusive recollections, posttraumatic nightmares and other re-experiencing phenomena of PTSD are often associated with anger. These symptoms are significantly influenced by the arousal symptom cluster (Schell, Marshall & Jaycox, 2004) and, especially, angry rumination (Orth, Cahill, Foa & Maercker, 2008).

Where posttraumatic anger is increased, PTSD is increased. Imagery is never far from such increases and the relationships between PTSD, anger and imagery are arguably far from coincidental. This research in interested in imagery as a) mechanistic device underlying their interconnectedness and b) its capacity for explaining the intensity of anger in PTSD.

Research of anger in PTSD from the vantage point of imagery is timely. There is a wealth of research and evidence showing that imagery and anxiety share a strong connection. There is, similarly, increasing recognition PTSD is associated with emotions other than anxiety (see Resick & Miller, 2009; Rosen, Lilienfeld, Frueh, McHugh & Spitzer, 2010), with some authors estimating that 50% and more of PTSD’s affective experience relates to anger, disgust and shame (Friedman, 2011; Oktedalen et al., 2014;
Power & Fyvie, 2013; Weston, 2014). The relocation in DSM 5 (APA; 2013) of PTSD from the anxiety disorders section [of DSM III (APA; 1980) and IV (APA; 1994)] to the section entitled Trauma and Stressor Related Disorders, is indicative of its ongoing conceptual evolution. There is, accordingly, no conceptual barrier to exploring PTSD as strongly anger related on account of an association with imagery.

1.1 The Need for Research of PTSD, Anger and Anger in PTSD

The Impact of PTSD, anger and anger in PTSD is considerable. Although data about the economic consequences of PTSD is limited (Chan, Air & MacFarlane, 2003), that available emphasises PTSD’s far-reaching fiscal burden. Greenberg and colleagues (1999) concluded that PTSD causes greatest economic impact to society among the then anxiety disorders, where it accounted for one-third of those treated and 40 per cent of total treatment cost (Issakidis, Sanderson, Corry, Andrews & Lapsley, 2004). A mathematical model comparing PTSD and comorbid major depressive disorder costs for US military personnel returned from Iraq and Afghanistan during 2005 estimated the two year per-capita PTSD treatment cost to be between US$5,900-$10,300 and US$12,430-$16,890 for comorbid PTSD-MDD (Tanielian, & Jaycox, 2008). A recent review by Hendricks and colleagues (2012) indicated annual care costs for contemporary US military veterans with PTSD average US$12,473. Economic costs represent only a fraction of the overall burden of PTSD. Research has found PTSD is associated with reduced job status, stability and salaries (Fairbank, Ebert & Zarkin, 1999), and negatively affects health and quality of life, interpersonal and intimate relationships, and aggression and violence (Monson, Taft & Fredman, 2009). Overall, PTSD is characterised by poor physical health and, when untreated, high symptom chronicity, psychiatric and medical comorbidity, and functional impairment (Beckham, Calhoun, Glen & Barefoot, 2002; Elhai, Contractor, Palmieri, Forbes & Richardson, 2011; Ouimette, Cronkite, Henson, Prins & Moos, 2004; Schnurr & Green, 2004; Stander, Thomsen, & Highfill-McRoy, 2014).

A precise reckoning of the economic burden of dysfunctional anger is yet to be undertaken. Its cost is potentially immense, having been judged as equivalent to that applicable to depression (Howells, 1998), which is forecast to become the leading cause of disease burden worldwide by 2030 (WHO, 2011). Consistent with the centuries-old hypothesis that emotions influence physical health (Smith, Glazer, Ruiz & Gallo, 2004), research has powerfully demonstrated that dysfunctional anger is directly associated with morbidity. Most well-known is the fifty-year evidence base of anger’s association with
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cardiovascular disease. Chronically angry individuals have a sevenfold hazard ratio for coronary heart disease (CHD) and 6.4 times the risk of myocardial infarction (Ohira, 2010). Research relating to circulatory disease provides similar evidence of anger’s ill-health effects. For example, it has been reported that those with chronic anger have a six-fold risk of stroke after risk-factor adjustment (e.g., for age, resting blood pressure, smoking, alcohol consumption, body mass index, socio-economic status and diabetes) (Everson et al., 1999). There is considerable evidence of anger’s role in relation to other significant pathologies. It has been implicated in cancer, immune, metabolic, gastrointestinal and respiratory system illnesses (Lemerise & Dodge, 2008; Paulus, Fiedler, Leckband & Quinlan, 2005; Raikkonen, Matthews & Kuller, 2001). It has likewise been shown to be a critical precipitator, exacerbator and perpetuator of chronic pain of physical and psychological origin (e.g., fibromyalgia) (Fernandez & Wasan, 2010; Fishbain et al., 2011; Sayar, Gulec & Topbas, 2004). In this regard, research has identified anger as the strongest predictor of chronic headaches (Nicholson, Gramling, Ong & Buenevar, 2003), anger-suppression as the strongest predictor of pain intensity and pain behaviour (Burns, Johnson, Devine, Mahoney & Pawl, 1998; Quartana, Bounds, Yoon, Goodin & Burns, 2010) and anger-intensity as the strongest predictor of perceived pain, interference and activity level (Kerns, Rosenberg & Jacob, 1994). Dysfunctional anger is associated not only with increased morbidity, but crucially also mortality (Cohen & Pressman, 2006).

Dysfunctional anger is inversely related to quality of life (Horesh et al., 1997). It is strongly associated with psychopathology ranging from everyday dysphoria (Sukhodolsky, Golub & Cromwell, 2001; Thurman, 2005); to self-harm and suicide (Yesavage, 1983; Demirbas, Ilhan & Dogan, 2011). It causes significant impairment in psychological functioning, intellectual performance, reasoning, decision making, problem solving and goal-setting along with anger-related misjudgements, mistakes, and accidents are common (Fessler, 2010, Tripp & Bies, 2010). Dysfunctional anger has a particularly troubling association with aggression and violence and its adverse impact on intimate relationships is well-identified - as evidenced in marital distress, breakdown and domestic violence (Underwood, 2003) and the aggregation of hostility in families (Matthews et al., 1992). In this regard, it is known depressed violent men are more likely to engage in anger toward their partners and more likely to report angry behaviours than non-depressed non-violent men (see Pan, Neidig & O’Leary, 1994). Importantly, while the finding that depression increases interpersonal aggression is common, it has been proposed that anger mediates this relationship (Murphy & Eckhardt, 2005). Dysfunctional anger is socially
judged as abnormal, undesirable and repellent, even in the absence of aggression and violence and when chronic is associated with lack of social support (Scherwitz, Perkins, Chesney & Hughes, 1991). It attracts censure - interestingly at lesser thresholds, for women than men - and is associated with personal, interpersonal, and social loss, as well as rejection (Fischer & Evers, 2010) and the lifestyle of chronically angry people is one of isolation, alienation and association with other angry individuals (Biordi & Nicholson, 2011).

A distinct body of evidence about the burden of anger in PTSD does not exist. It may, however, be inferred from anger’s involvement in the severity and chronicity of PTSD. Anger is considered a key factor in PTSD’s development and moderator of PTSD severity (Andrews, Brewin, Rose & Kirk, 2000; Frueh, Henning, Pellegrin & Chobot, 1997; McHugh et al., 2012; Orth & Maercker, 2009), with upper-end estimates suggesting 40 per cent of PTSD score-variance can be attributable to anger (Novaco & Chemtob, 2002). Anger is an underlying contributor to the aggressiveness that characterise PTSD (Riggs, Dancu, Gershuny, Greenberg & Foa, 1992; Taft, Vogt, Marshall, Panuzio and Niles, 2007) and has a profound effect on the personal and family relationships of PTSD sufferers (see Hinton, Rasmussen, Nou, Pollack & Good, 2009; Worthen et al., 2014). Exemplifying this is the recent literature illustrating the social aggression of contemporary veterans with anger-affected PTSD presentations (see Chapter Two) and research indicating anger is the most often reported presenting problem of treatment seeking combat veterans and their family members (e.g., Biddle, Elliott, Creamer, Forbes, & Devilly, 2002). High PTSD symptom thresholds may not be required for impactful dysfunctional anger. Reminiscent of the Kessler and colleagues’ (1995) findings that sub-threshold PTSD is twice as prevalent as PTSD, and associated with high morbidity and mortality, a study of anger and aggression in a non-clinical sample by Jakupcak and Tull (2005) found those who reported PTSD symptoms (but did not attain diagnosis) had higher levels of anger than those who reported no such symptoms, thereby suggesting the impact of PTSD does not rely on a full hand of symptoms where anger is involved.

There is a general association of anger with poor treatment outcomes across psychiatric disorders (e.g., Burns et al., 1998; Haaga, 1999; Rao, Broome & Simpson, 2004). This applies to PTSD, where higher anger is associated with diminished treatment response (Andrews et al., 2000; Forbes, Creamer, Hawthorne, Allen & McHugh, 2003), and early treatment termination (Stevenson & Chemtob, 2000). This may explain why PTSD, especially when chronic, is difficult to treat (Bradley, Greene, Russ, Dutra &
Successful anger treatment is associated with significant decreases in PTSD symptomatology, severe PTSD included (see Chemtob et al., 1997; Morland et al., 2012), and research consistently shows treatment of anger reduces both anger and intrusion levels in PTSD (Novaco & Chemtob, 1998). In the absence of effective treatment, this anger-PTSD relationship is enduring and in the last two decades, regular recommendations have been made by the field for additional treatment of anger in the presence of PTSD. These began with calls by Pitman (Pitman et al., 1991) and have been successively reiterated, most recently by Elbogen and colleagues (2010) and Elbogen, Johnson and Beckham (2011), Forbes, McHugh and Chemtob (2013), Morland and others (2012; 2014), Rona and colleagues (2015) and Worthen and associates (2014).

1.2 The Explanatory Value of Researching Anger in PTSD from the Perspective of Imagery

As observed, imagery discriminates PTSD from other trauma-related mental health disorders and may reasonably be understood as a problem of disordered visual imagery, where visual imagery is predominant. Furthermore, intrusive recollections, posttraumatic nightmares and the other re-experiencing phenomena of PTSD are often associated with anger and perhaps amplified by and causally-linked to it.

Notwithstanding this, there is surprisingly little PTSD research of this imagery-anger association. This parallels the general imagery-emotion research situation, where imagery is long regarded as a strong contributor to anxiety, yet as observed across chapters four to seven, the impact of imagery on anger is yet to be sufficiently explored.

This research is interested in imagery as a mechanistic device underlying their interconnectedness and its capacity for explaining the intensity of anger in PTSD. Research of anger in PTSD from this vantage point is timely. This is because, there is increasing recognition that PTSD is associated with emotions other than anxiety (see Resick & Miller, 2009; Rosen, Frueh, Lilienfeld, McHugh & Spitzer, 2010), with some authors estimating that 50% and more of PTSD’s affective experience relates to anger, disgust and shame (Friedman, 2010; Oktedalen et al., 2014; Power & Fyvie, 2013; Weston, 2014). Such research and the relocation in DSM 5 (APA; 2013) of PTSD from the anxiety disorders section [of DSM III (APA; 1980) and IV (APA; 1994)] to the section entitled Trauma and Stressor Related Disorders, is indicative of the disorders ongoing conceptual evolution.
There is, accordingly, no impediment to exploring PTSD as strongly anger related on account of an association with imagery. To reiterate, imagery for the purpose of this research is understood as: the quasi-perceptual, subjectively-influenced, cognitive representation and recollection of perceptual experience in working memory in the absence of the originating stimulus (see Kosslyn, 2005a). As detailed in greater depth in Chapter Six, adoption of this understanding emphasises that it is the characteristics of mental imagery invoked by a stimulus, not the stimulus itself, which are the key determinant of the response to that image. In this research, the characteristics investigated are comprised of imagery’s frequency (considered a proxy measure for control), absorption and vividness and anger-related content. As emphasised in chapters four and six, these first three imagery features have been documented for their importance in research of emotion and are well-discriminated from the visual imagery pertaining to perception. This delineation and the multi-study nature of the stepped, multi-study nature of this research permits the contrasting measurement of the impact of basic facets of imagery on anger and anger in PTSD and the development of conceptual argument around the autobiographical, meaning-related and ruminative nature of imagery in PTSD and anger in PTSD.

Accordingly, the latter third of Chapter Four and thereafter Chapter Five make the case for imagery’s involvement in anger and anger in PTSD, while Chapter Six articulates a variety of means by which this may occur and Chapter Seven, in drawing this literature into the rationale for this research, presents a model that proposes increases in anger will lead to increases in imagery (a measured by imagery frequency, vividness absorption and anger) and that imagery will lead to increased PTSD symptomatology.

1.3 Research Structure

This thesis was designed to test key propositions about anger, PTSD and imagery via three inter-related studies. The first study sought to establish the connection of anger to PTSD. For this purpose, it examined affect (anxiety, depression and anger) and PTSD data obtained from PTSD affected, treatment-seeking Vietnam veterans of the Australian Defence Forces (ADF). The data from this study sought to establish the relationship of anger to PTSD, including its variability and course. It confirmed the role of anger as a predictor of PTSD in this group.

Building on the results of study one, the second study subsequently explored the relationship between specific visual imagery characteristics identified in the pertinent literature as implicated in the generation and maintenance of negative affect (anger,
anger and depression) - that is, frequency, absorption and vividness. Because of the research project’s interest in anger, a measure of angry imagery content was also included along with a measure of automatic negative thoughts. Given this study was interested in illuminating normative psychological, rather than psychopathological processes, this investigative approach was applied to a non-clinical population. These data allowed for exploration of direct and mediatrical effects of visual imagery as a mechanistic device and thought on negative affect and, in particular, anger. This approach was perceived as capable of demonstrating the association of difference elements of imagery to different affects, while emphasising the mediating role of thought in imagery’s connection to negative affect and was shown to be successful in this objective.

Following on from the findings of these initial two studies, the third and final study sought to assess the relationship of visual imagery and thought to anger in PTSD in a clinical population. For the reasons articulated, the preferred approach was again to investigate the impact of imagery by recourse to the imagery characterises described (frequency, absorption, vividness and angry content) in this study, rather than the inclusion of measures specifically aimed at tapping into visual intrusions or other clinical characteristics of imagery pertinent to PTSD. Via this approach, this study demonstrated the importance of anger-associated imagery and thought to PTSD.

1.4 Thesis Structure

Beyond this introductory chapter, the succeeding chapters provide a context for, and description of, the studies conducted in this research. The first three of these chapters (chapters two to four) sequentially define PTSD, anger, and anger in PTSD, and describe their, phenomenology, epidemiology, trajectory, risk factors and impact(s). Each chapter also reviews available explanatory theories for the construct in focus and the utility of those theories for accounting for anger in PTSD.

A detailed description of the evidence supporting the key role of imagery in anger in PTSD is provided in Chapter five. Factors that affect imagery and how they might operate at a mechanistic level in anger in PTSD are then considered (Chapter Six).

The next chapter outlines the research rationale, with the three specific studies and their results described in chapters eight to ten. Key findings are presented and discussed for their theoretical and practical importance across these chapters.

The manuscript concludes with a detailed integration of the overall research project and its implications for clinical practice and future research (Chapter Eleven).
CHAPTER 2: THE NATURE OF PTSD

Am I awake or dreaming? It doesn’t matter anymore. When I close my eyes I dream of death and war. When I open my eyes I see death and war.

Mass Casualties: A Young Medic’s True Story of Death, Deception and Dishonour in Iraq (Michael Anthony)

Chapter one provided a contextual overview of this research and its goal of exploring the relationship between dysfunctional anger and PTSD and the role of visual imagery as a mechanistic device underlying this relationship. This chapter defines PTSD and describes its phenomenology, conceptualisation, epidemiology and impact, along with the key theoretical explanatory models. At its conclusion, it notes the relative absence of theoretical and empirical attention given to anger and the consequences of this.

2.1 The Formal Definition of PTSD and its Phenomenology

2.1.1 The diagnosis of PTSD. The formal diagnostic criteria for PTSD were first described in the third edition of the Diagnostic and Statistical Manual of Mental Disorders [DSM III; American Psychiatric Association (APA), 1980]. Its 5th edition (DSM 5; APA, 2013) revised PTSD’s diagnostic criteria for the fourth time. These criteria are outlined in Table 2.1 and fully enumerated in Appendix A.

Inspection of them emphasises the underlying centrality of visual imagery to the disorder’s hallmark re-experiencing (cluster B) criteria - as reflected in the disorder’s unbidden intrusive memories and nightmares and stimulus-cued recollections. It similarly underscores the importance of imagery to the disorder’s avoidance (of trauma-related stimuli) (cluster C) criteria. In this respect, it is reasonable to assume that the aversive nature of painful imagery drives - at least in part - the efforts to avoid the distressing memories (Criterion C1). Thus, PTSD sufferers can be afflicted by intrusive, aversive imagery and preoccupied by attempts (conscious and otherwise) to escape it.
Table 2.1

Summary of PTSD Criteria in DSM 5

<table>
<thead>
<tr>
<th>Criterion Set</th>
<th>Content(s)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Traumatic Stressor</td>
<td>Personal exposure to (threatened) death, serious injury or sexual violation</td>
</tr>
<tr>
<td>Re-experiencing of the trauma</td>
<td>Recurrent, involuntary and intrusive memories and nightmares, dissociative reactions, intense or prolonged distress or physiological reactivity on exposure to traumatic reminders</td>
</tr>
<tr>
<td>Effortful avoidance of trauma-associated stimuli</td>
<td>Avoidance of trauma-related thoughts, feelings or sensations, trauma-related external reminders or interpersonal interactions</td>
</tr>
<tr>
<td>Negative alterations in cognition or mood post-trauma</td>
<td>Inability to recall key trauma features, (distorted) negative beliefs and expectations about oneself or the world, (distorted) blame of self or others, negative trauma-related emotions, markedly diminished interest in pre-trauma activities, feeling alienated from others or constricted affect</td>
</tr>
<tr>
<td>Altered post-trauma arousal and reactivity</td>
<td>Irritable, aggressive, self-destructive or reckless behaviour, hyper-vigilance, exaggerated startle-response, problems in concentration or sleep disturbance</td>
</tr>
</tbody>
</table>

As currently defined in DSM 5, PTSD is a high-impact psychological consequence of exposure to a potentially traumatic event (PTE). For diagnosis, an individual must have experienced a significant traumatic stressor and meet criteria for at least one symptom from each of the re-experiencing and active avoidance clusters and at least two symptoms from each of the negative cognition/mood and arousal/reactivity clusters.

According to DSM 5, diagnosis cannot occur unless symptoms have been present for one month and cause clinically significant distress or impairment in social, occupational or other important areas of functioning. Impairment must not be caused by medication, substance use or other illness. The disorder is considered acute when symptom duration is less than three months, chronic if beyond three months and
delayed where onset occurs six months or more post-event. A dissociative subtype for clinical presentations with high levels of depersonalisation or derealisation has also been introduced in DSM 5.

Although the DSM conceptualisation of PTSD is the most widely used, the diagnosis also appears in the International Classification of Disease (ICD). The ICD’s 10th edition PTSD criteria were adapted from DSM III (APA, 1980) and, since then, the two systems have defined PTSD with minor divergence (see Table 2.2). The first attempt to reconcile the various aspects of the syndrome occurred in DSM III (Benedek & Wynn, 2011). Conceptualisation of the disorder, however, remains fluid and continues to evolve as a result of phenomenological, theoretical and empirical enquiry. This evolution involves lively debate. The range of issues debated has been well-synthesised (see Friedman, 2011; McNally, 2004 and Rosen et al., 2010) and critiqued (e.g., in the 2007 special edition of the Journal of Anxiety Disorders, entitled Saving PTSD from Itself in DSM-V).

Table 2.2

PTSD Diagnostic Criteria to 2013: A system comparison

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Stressor</td>
<td>A</td>
<td>A</td>
</tr>
<tr>
<td>Re-experiencing</td>
<td>B1-5 (1)</td>
<td>B (1)</td>
</tr>
<tr>
<td>Avoidance</td>
<td>C1-2 (1)</td>
<td>C (1)</td>
</tr>
<tr>
<td>Cognition and Mood Alteration</td>
<td>D1-7 (2)</td>
<td>D1 (Amnesia) (1)</td>
</tr>
<tr>
<td>Arousal</td>
<td>E1-6 (2)</td>
<td>D2.(a-e) (2)</td>
</tr>
<tr>
<td>Onset</td>
<td>&gt; 1 month</td>
<td>&lt; 6 months</td>
</tr>
<tr>
<td>Functional Impairment</td>
<td>✓</td>
<td>✓</td>
</tr>
</tbody>
</table>

Various issues have been identified regarding the overall structure of the disorder and each of its constituent criteria sets. Even PTSD’s hallmark reliving criteria have been critiqued, with the fidelity of intrusive images and the allegedly 20th century-bound incidence of the intrusive flashback (McNally, 2003; 2004) being questioned. The most intense debate, however, has surrounded the nature and role of the stressor criterion (criterion A) and, more particularly, the criticality of traumatic...
events to diagnosis and the relationship of trauma-dose to response intensity (McNally, 2004). As a disorder of reactivity, PTSD requires a specific, identifiable precipitant for its onset (Friedman, 2011; Shalev, 2009). Despite conjecture that diagnosis might be improved by eliminating criterion A altogether (see Friedman, Resick & Keane, 2014; O’Donnell, Creamer & Cooper, 2010), the stressor continues to be understood as an essential feature of PTSD by most experts (Friedman, 2011).

There have, however, been significant adjustments in thinking about criterion A. Indicative of the then prevailing understanding, DSM III awarded causal priority in PTSD to traumatic events, rather than personal traits (McNally, 2001). This reversed the prominence given to individual vulnerability and exclusion of event significance in PTSD’s causation in early psychotraumatology (e.g., Freud’s theorising - see McNally, 2003; 2004). From DSM III-R (APA, 1987), the interplay between the stressor and psychological characteristics of the individual has been reiteratively rebalanced. This has involved a shift in concentration from the pathogenic properties of events to vulnerability factors which render some individuals more susceptible to developing PTSD (McNally, 2001).

Significant alterations included removing the DSM III sub-criterion A1 specified need for the event to be “outside the range of usual human experience” and DSM III’s survival-guilt component. These changes shifted emphasis from stressor-severity to the individual’s reaction and implied vulnerability to it. Accordingly, threat perception became a critical ingredient and the stressor-criterion more inclusive (McNally, 2004), thereby allowing for events like non-fatal traffic accidents, medical procedures and even harrowing birth experiences (Ayers, Joseph, McKenzie-McHarg, Slade & Wijma, 2008; Susan, Harris, Sawyer, Parfitt & Ford, 2009) and to be considered traumatic. An unintended consequence of this inclusiveness was the criterion “bracket creep” of that considered traumatogenic (McNally, 2004; Rosen & Lilienfeld, 2008).

In an attempt to limit the inherent risk of medicalising natural human emotional responses to adversity, DSM 5 modified the definition of the stressor event. Accordingly, to meet diagnosis, the event must be personally experienced or where impersonally experienced, involve violence human perpetration or significant accidents, not natural causes and a close relative or friend. These changes have been aimed at repositioning emphasis away from everyday occurrences toward extraordinary, directly experienced events.
Arresting diagnostic bracket-creep in this manner was meant to take account of the trauma dose-response relationship dilemma; that is, the understanding that, while extreme stressors are likely to produce more PTSD symptoms than lesser stressors, reactivity is not inevitable. In noting this quandary’s existence, McNally (2004) cites many possible explanations for it. His most compelling explanation is the requirement that events be subjectively appraised as traumatic. He refers to this as the central paradox of PTSD. In a telling illustration of it, he ponders why public executions in the 19th century were not experienced as traumatic. The same question could, of course, be asked of other trauma-as public event scenarios from earlier eras, such as the French revolution’s guillotine executions and ancient Rome’s gladiatorial contests. McNally proposes that, when overstimulation occurs, vulnerability factors are likely to be involved. The inverse of this is, of course, that individuals, who, by inclination, perceive less threat, react less and are less likely to be symptomatic of PTSD.

The next DSM 5 amendment to PTSD’s stressor criterion was the elimination of the second DSM IV sub-criterion (A2), which required that an individual respond with fear, helplessness or horror. This alteration recognised many trauma-exposed individuals do not typically respond with a fear response and that, as previously observed, dominant emotions in PTSD commonly include non-anxiety based feelings, like disgust, guilt, shame and anger. A prominent example of this being the vocational groups routinely exposed to trauma as part of occupational duties (like police and military personnel) (see Mayhew, 2001; Monson, Resick & Rizvi, 2014; Violanti, 1999).

A major overall in the structure of DSM 5 was also undertaken via the splitting of the DSM III to DSM IV-TR avoidance criterion (C) into DSM 5’s effortful avoidance and negative alterations in cognition/mood criteria (criteria C & D). The resultant four symptom cluster structure is thought to reflect the true nature of PTSD as confirmed by most of a series of confirmatory factor analyses (see Friedman, 2011; Friedman et al., 2014).

2.1.2 The phenomenology of PTSD. The central focus of PTSD is a preoccupation with the past. Sufferers experience this preoccupation as distressing recollections in the form of both unbidden intrusive multi-sensory intrusions and self-initiated, but poorly-controlled thought and mental imagery and high-sensitivity to cues and reminders of the traumata involved that are disruptive of current functioning.
In an attempt to gain psychological control over such phenomena, PTSD sufferers avoid possible event reminders. Avoidance is typically intermittent and temporary, however, and distressing reliving phenomena inevitably re-intrudes. Less commonly, the individual may experience dissociation. This may occur in either or both of two dissociative subtypes (Shauer & Elbert, 2010), where the individual either dissociates into (relives) the traumatic event memory as if it were happening again (resulting in high impact distress and arousal) or dissociates away from it (into mental nothingness) from event memories and reminders (resulting in emotional numbing and disengagement). The apotheosis of dissociation is the complete loss of person, place and time. While dissociation at this intensity is relatively rare, regardless of direction, high intensity peri and post-traumatic dissociation, as identified in the discussion of the risk-factors of PTSD in sub-section 2.3.4, have great prognostic value in terms of the course of the disorder.

Another consequence of this reliving-avoidance cycle identified by PTSD sufferers is alteration to mood and cognition in the form of a generalised impoverishment of affect, trauma-distorted cognitions about the self, others and the world and consequent constriction of behaviour. Not uncommon in extreme intensities, this can involve a pervasive sense of detachment from others, a profound loss of enjoyment of day-to-day activities and an inability to plan for the future.

The final cluster of experiences reported by PTSD-affected individuals is a contrary-response-set to avoidance. It comprises symptoms of hyper-arousal., whereby sufferers experience negative emotions, like guilt, shame, disgust and, above all, anger. This hyperarousal may also be experienced as a state of alert, whereby the individual is primed to new threats of danger at an intensity that engenders over-reaction to innocuous stimuli and interferes with the ability to attend to, concentrate on or accomplish current-day tasks.

**2.2 Historical and Cultural Conceptualisations of PTSD**

Powerful illustrations of what is now known as PTSD exist as far back as the ancient Greek and Roman mythological accounts of the Iliad, Odyssey and Aeneid (see Friedman, 2011 and Trimble, 1985). PTSD is not an artifact of western history (Lewis-Fernandez, Hinton & Marquez, 2014) and long-standing phenomenological descriptions can be found in diverse cultures, including those of Asian, Latino, Middle Eastern and European Cultures and societies (Ben Ezra, 2001; Lewis-Fernandez,
Hinton & Marquez, 2014; Trimble, 1985). These descriptions continue to have contemporary descriptive and explanatory value. For example, Shay (1995) has deployed the Achilles allegory in his psychiatric and literary treatise on the place of anger in US servicemen with combat-related PTSD, starting with veterans of Vietnam (Achilles in Vietnam; 1995) and then Iraq and Afghanistan (Odysseus in America; 2002).

Archetypal descriptions of key PTSD phenomena exist in well-known historical works of literature. To illustrate, in the 16th century, Shakespeare, who first depicted real-life humans in drama (Bloom, 1999), anticipated PTSD’s symptom clusters by several hundred years. Hence, Lady Percy’s address to her husband Lord Henry Percy (aka Hotspur) in Henry IV not only depicts the preoccupation, nightmarish intrusions, lowering of mood, irritability, hyperarousal and altered behaviour of PTSD, but is a most powerful illustration of Shakespeare’s understanding of the effect of trauma and PTSD on individuals and relationship. Lady Percy pleads of Henry:

O, my good lord, why are you thus alone?
For what offense have I this fortnight been
A banished woman from my Harry’s bed?
Tell me, sweet lord, what is’t that takes from thee
Thy stomach, pleasure and thy golden sleep?
Why dost thou bend thine eyes upon the earth,
And start so often when thou sitt’st alone?
Why has thou lost the fresh blood in thy cheeks,
And given my treasures and rights of thee
To thick-eyed musing and curst melancholy?
In thy faint slumbers I by thee have watched,
And heard thee murmur tales of iron wars,
Speak terms of manage to thy bounding steed,
Cry, “Courage! To the field!” And thou hast talked
Of sallies and retires, of trenches, tents,
Of palisadoes, frontiers, parapets,
Of basilisks, of cannon, culverin,
Of prisoners’ ransom, and of soldiers slain,
And all the currents of a heady fight.
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Thy spirit within thee hath been so at war
And thus hath so bestirred thee in thy sleep,
That beads of sweat have stood upon thy brow,
Like bubbles in a late-disturbèd stream
And in thy face strange motions have appeared,
Such as we see when men restrain their breath
On some great sudden hest. O, what portents are these?

(act 2, scene 3)

Another well known description of PTSD selected phenomenon is his description of Samuel Pepys 17th century described the impact of posttraumatic nightmares. Quoted repeatedly across accounts of PTSD’s phenomenology, Pepys observed in the aftermath of the Great Fire of London, in his historically invaluable personal diary, that It is strange to think how to this very day I cannot sleep a night without great terrors of the fire (see Trimble, 1985).

Contemporary descriptions of PTSD can be traced to the 19th century. Impetus for the investigation and conceptualisation of PTSD was derived from events such as warfare, natural and man-made disasters (British rail accidents were an important example of the latter) and crime. Research reflected the work of renowned clinician/researchers (e.g., Briquet, Charcot and Oppenheim in relation to childhood sexual assault) and, in particular, the work of Janet. He described the psychologically fragmenting and dissociating effect of trauma, whereby the individual becomes separated from person, place and/or time, and was the first to assert dissociation is a psychological defence against overwhelming stress (see Friedman, 2011; Monson, Friedman & Bash, 2014; van der Kolk, Brown & van der Hart, 1989; Weisaeth, 2014)

Warfare is a fertile ground for the development of PTSD (Everly, 1995) and the phenomenological description and formal diagnosis of PTSD owe much to its psychological consequences. Exemplifying this, what is now recognised as combat-related PTSD was anciently depicted in fictional works and scared texts, like Homer’s Iliad (750 BC), the Jewish Torah/Christian Old Testament (500 BC) and the epics of Gilgamesh (400 BC) (see Tomb, 1994). Reliable, factual reporting of such was perhaps first cited in Herodotus’s 490BC report of a soldier’s hysterical blindness on witnessing a comrade’s death in the Battle of Marathon (Rawlinson, 1901 reprinted in 1996).
In the 17th century, the power of preoccupation with the past is exemplified in the use of the term Nostalgia to describe Swiss soldiers characterised by an inability to concentrate or perform military tasks due to fixation on prior combat-trauma and a longing for a past-life (Jones & Wessley, 2005). Two centuries later, Napoleon’s troops were reported to have suffered the same affliction. The phenomenon was reported again during the US Civil War and 1905 Russo-Nippon wars, albeit respectively re-termed as Homesickness and Neurasthenia (Jones & Wessley, 2005).

The first modern diagnostic term for the syndrome derives the 1905 Russo-Nippon war. Initially referred to as the aforementioned Neurasthenia, it was quickly re-termed War-Neurosis in 1907 (Crocq & Crocq, 2000). As warfare became globally impactful and its reportage commonplace, terms occasionally entered common parlance. A striking example is the First World War (WW I) term Shell Shock, a condition synonymous with loss of reason, helplessness, overwhelming fear, inability to sleep (and sometimes walk or talk) and a general loss of self-control (Hochschild, 2011). The Second World War (WW II) saw PTSD labelled Combat/Operational Fatigue (Grinker & Spiegel, 1945) and Kardiner instigated the term Physioneurosis to reflect its physiological and psychological symptoms (Tomb, 1994). Unlike Shell Shock, these terms proved transient.

The multiplicity of US military personnel presenting with persistent psychiatric disturbance and re-adjustment problems post service in the Vietnam conflict provided the impetus for a re-evaluation of the diagnostic category of stress responses (Schlenger, Fairbank, Jordan & Caddell, 1999). DSM II (APA, 1968) contained a diagnosis of “adjustment disorder of adult life” to describe psychological reactions to extreme events, but the assumption was that the disorder would remit once the stressor had passed. That view of the syndrome was rejected for its inadequacy and DSM III for the first time in the psychiatric nomenclature fully recognised the psychologically traumatising effects of war and other life threatening events (see Tomb, 1994) and PTSD entered diagnostic and colloquial language.

Events beyond warfare have, of course, also been an important element of PTSD’s conceptualisation and by the 1970s the disorder was recognised to apply to the general population, not just military personnel. Thus, description, theory and research focused on disasters and emergencies of various kinds, crime (especially sexual assault, domestic violence and other crimes against women and children),
atrocities and accidents (Friedman, 2011). Various terms are testament to this. Some have been opaque - for example, the Railway Spine/Brain (that reflected a belief that the disorder was caused by lesions to the spinal cord resulting from train derailments) and Compensation Neurosis (which connoted the complications of secondary psychological or economic gain beginning with the 1871 Prussian introduction of compensation legislation) (van der Kolk, Weisaeth & van der Hart, 1996). Others focused explicitly on the pathogenic event - for example, Concentration Camp Syndrome (Eitinger & Strom, 1973), (holocaust) Survivor Syndrome (Kijak & Funtowicz, 1982) and Rape-Trauma Syndrome (Burgess & Holstrom, 1974) and required little explanation.

Initially, the disorder was thought to be a reflection of physical stress and physically oriented explanations dominated the nosology to mid-way through WW I. Accordingly, Shell Shock’s symptomatology was thought to be due to inescapable exposure to the close-quarter explosions of the immense artillery involved, resulting in subtle brain damage, via commotion and molecular derangement and neuronal disconnection (see Friedman & Pitman, 2007) resulting in injuries like paralysis, blindness and mutism (Shepherd, 2000). As WW I continued, however, the clearer it became that such conditions were psychogenic in nature (Shephard, 2000). Nevertheless, it took until WW II for psychological explanations to supplant those of a physical nature. It is these explanations that dominate today’s thinking.

Logically, all known cultural expressions of traumatic (dis)stress can neither be known nor effectively recorded in a single time and place. The relevant literature suggests, however, that the core phenomenology of PTSD is universal and timeless, albeit subject to specific cultural manifestations (Lewis-Fernandez, Hinton & Marques, 2014; Friedman, 2011). Core signs and symptoms consistently observed across time and cultures include: pre-occupation with traumatic events, intrusive daytime memories and post-traumatic nightmares, avoidance and dissociation, hyper-arousal and profound and all-encompassing irritability and severely disrupted functioning.

There are cultural critiques of the construct - for example, that it takes no account of important cultural syndromes, some of its symptoms (e.g., flashbacks) are indicative of western culture and it represents the medicalisation of human suffering (see Lewis-Fernandez et al., 2014). Nevertheless, key reviewers have predicted that the syndrome’s core symptoms, along with a range specific cultural and contextual
variations, will be continue to be validated by research across populations and settings (Lewis-Fernandez et al., 2014; Friedman, 2011).

2.3 Epidemiology

2.3.1 Trauma exposure and PTSD prevalence. Exposure to post traumatic events is commonplace. Large scale population surveys reveal the lifetime prevalence of exposure to potentially traumatic events is commonplace at around 65 per cent, with most people reporting experiencing two or more lifetime traumata (see Creamer, Burgess & McFarlane, 2001; Kessler et al., 1995; Norris, 2005). The most frequently reported events involve someone close dying unexpectedly (35 per cent of the general population), unexpectedly seeing a dead body or witnessing someone being badly injured or killed or (27 per cent) or being involved in a life-threatening car accident (13 per cent) (Mills et al., 2011).

In specific sub-populations, however, exposure to potentially traumatic events can be even higher. For instance, the psychiatrically disabled, those in countries with vicious internecine conflict, and past and present personnel of organisations whose defined day-to-day role inevitably involves traumatic exposure (e.g., military personnel and members of emergency service organisations, like police, ambulance and fire-fighting personnel). For those groups, rates have been cited to be in the order 90 per cent, 95 per cent and even 100 per cent (Forbes, McHugh & Chemtob, 2013; Mueser, Rosenberg & Mueser, 2009). Interestingly, it has been noted that in some trauma-exposed military populations, rates can approach zero (Bowman & Yehuda, 2004).

Notwithstanding their commonality, not all trauma-exposed individuals develop trauma-related mental health disorders. Such conditions are, by definition, an abnormality and a failure of the natural post-trauma recovery process (McFarlane, 2000). Their prevalence rates are, accordingly, low.

Exposure to a traumatic event may be associated with the development of a range of trauma-related mental health disorders. Principal among them are (depressive) mood and anxiety disorders (Bryant, 2010). The epitome of trauma-related mental health disorders is, however, PTSD.

Table 2.3 offers a comparative summary of estimated prevalence rates by trauma type. It indicates the highest 12 month prevalence of PTSD is associated with rape, with 58 per cent (65% of men; 45% of women) of victims meeting diagnosis for
PTSD (Creamer et al., 2001). Other gross personal violations - such as torture and barbarous internecine, ethnic conflict - also result in similarly high rates of PTSD incidence. In contrast, the lowest incidence of PTSD relates to potentially traumatic events of non-human origin such as natural disasters (Creamer et al., 2001) at four per cent.

Not surprisingly, populations exposed to high rates of potentially traumatic events tend to experience greater PTSD prevalence rates (Mills et al., 2011; Gates et al., 2012; McFarlane, Hodson & Davies, 2011; O’Toole, Catts, Outram, Peirse & Cockburn, 2009). Accordingly, those occupationally at risk - the most obvious examples being military forces, emergency services personnel and disaster relief workers - have among the highest incidence of PTSD. Illustrative of this, various Australian studies of military and veteran populations have estimated the prevalence of current PTSD at between 8.1 per cent and 11.4 per cent (Grayson, Dobson & Marshall, 1998; O’Toole et al., 1996), with lifetime rates approximately double those figures. This compares to general community prevalence rate of around 4.4 per cent for current and 7.2 per cent for lifetime PTSD (Mills et al., 2011).

Posttraumatic stress disorder prevalence rates have been documented across a wide variety of western and non-western countries. Such rates exhibit considerable between population variability. The most recently published WHO estimate of PTSD prevalence, which was incorporated in its 2004 Mortality and Burden of Disease Estimates for Member States, illustrates this, showing that age standardised disability-adjusted life year rates for PTSD, per 100,000 population varied from 59 for Thailand and 58 for the US to 46 for Mexico and 45 for Brazil.

Prevalence rates for PTSD exhibit considerable specific within-population variability. For example, the previously cited 11 per cent PTSD incidence rate for Australian Vietnam veterans (Grayson et al., 1998; O’Toole et al., 1996) contrasts with the 17 per cent rate of their US counterparts (Kulka et al., 1990). Differences between conflicts and military force involved in conflicts are apparent when comparing recently active military populations from different countries (hereafter termed contemporary veterans and taken to refer to serving military personnel and veterans involved in military operations such as the International Force for East Timor, Operation Enduring Freedom and Operation Iraqi and the Afghanistan intervention). For example, up to 13 per cent of such US contemporary veterans met
criterion for PTSD on return from their tour of duty, compared to six per cent for UK counterparts (Hoge et al., 2004; Hotopf et al., 2006).

Other high-risk populations show similarly prevalence diversity. To illustrate, surveys of Australian police suggest between six and 32 per cent of serving personnel may have the condition (Davidson, Berah & Moss, 2006), with comparative US police PTSD rates spanning 12 to 35 per cent (Mann & Neece, 1990) and Dutch police having been identified as having a seven per cent disorder prevalence, with 34 per cent reporting sub-threshold PTSD (Carlier, Lambert & Gersens, 1997).

Table 2.3

Estimated PTSD Prevalence by population

<table>
<thead>
<tr>
<th>Population</th>
<th>Rates</th>
<th>Rates</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>&lt; 12/12s</td>
<td>Lifetime</td>
</tr>
<tr>
<td>Refugees</td>
<td>&gt;50%</td>
<td>&gt;50%</td>
</tr>
<tr>
<td>Prisoners of war</td>
<td>&gt;50%</td>
<td>&gt;50%</td>
</tr>
<tr>
<td>Sexual assault survivors</td>
<td>28-58%</td>
<td>28-58%</td>
</tr>
<tr>
<td>Survivors of torture</td>
<td>&gt;50%</td>
<td>&gt;50%</td>
</tr>
<tr>
<td>Terrorist attack/atrocity survivors</td>
<td>25%</td>
<td>25%</td>
</tr>
<tr>
<td>Criminal assault survivors</td>
<td>15%</td>
<td>15%</td>
</tr>
<tr>
<td>Combat veterans</td>
<td>2-17%</td>
<td>22-33%</td>
</tr>
<tr>
<td>Emergency Services personnel</td>
<td>6-9%</td>
<td>6-35%</td>
</tr>
<tr>
<td>Accident and disaster survivors</td>
<td>4-6%</td>
<td>4-60%</td>
</tr>
</tbody>
</table>

Sources: Carlier, Lambert & Gersens, 1997; Chapman and colleagues (2012), Creamer and colleagues (2001), Davidson, Berah & Moss, 2006; Galea and colleagues (2005), Grayson, Dobson & Marshall, 1998; Hoge and associates, 2004; Hotopf et al., 2006; Kulka and others, 1990; Mann & Neece, 1990; Mills et and associates, 2011; and O’Toole and colleagues, 1996.

Such variations illustrate two important points. First, to reiterate, even when horrific events are involved or recurrent exposure applies, not all trauma-exposed individuals develop PTSD. This holds even for the most morally challenging of traumata. Second, although men are much more likely to be exposed to trauma (by dint of warfare, occupation or social-aggression), women have consistently been shown to exhibit around double the PTSD rate of men (DSM 5; APA, 2013), with some analyses suggesting women exposed to a given trauma are four times more likely to develop PTSD [International Society for Stress and Trauma Studies (ISSTS),
2.3.2 Onset and course. The course of PTSD is relatively predictable, with a clear point of onset and identifiable symptom decay for many trauma survivors (Shalev, 2006). Two symptomatic trajectories are apparent. First, while a high percentage of people will initially experience some trauma-related mental health disorders symptoms (Rothbaum, Foa, Riggs, Murdoch & Walsh, 1992; Tomb, 1994), the vast majority maintain or quickly regain equilibrium and the tendency is to spontaneously diminishing symptom intensity.

Typically, symptoms decrease most in the first 12 months post-event (ACPMH, 2013), with complete recovery occurring for over 50 per cent of those affected within the first three months post-event (Blanco, 2011; Friedman, 2011) (thereby paralleling the three month DSM chronicity marker). Indicative of this, a study of sexual assault survivors by Rothbaum and colleagues (1992), reported that 94 per cent of rape and 76 per cent of non-sexual assault survivors met diagnostic criteria two weeks post event. Three weeks later, the incidence had reduced to 64 per cent and 42 per cent respectively and to 47 per cent and 22 per cent six weeks later.

Such small-group-study findings have been confirmed by large-scale and national population epidemiology data. Kessler and colleagues (1995) examined the longevity of PTSD symptoms by applying survival analysis to data gathered as part of the US National Comorbidity Study. Their analysis thus showed symptoms decreased most substantially in the first 12 months following the event. Applying with the same approach to the 1996 Detroit Area Trauma Survey, Breslau, Chilcoat & Schultz (1998) showed that 26 per cent of PTSD cases remitted by six months and 40 per cent by 12 months. More recent studies and reviews have produced similar such findings and shown this symptom reduction pattern is not constrained by country-of-origin. Thus the Australian study of remission from PTSD in the general population by Chapman and colleagues (2012) demonstrated this rate of decline continued, with 50 to 60 per cent reductions in symptomatology two to 10 years post event.

The second obvious trajectory involves a minority of PTSD sufferers, who continue to meet criteria in a continuous or fluctuating manner for many months, years or even decades post-event (Blanco, 2011; Friedman, 2011). Evident of this,
almost half the male (and one third the female) Vietnam veterans in the US National Vietnam Veteran Readjustment Study with a lifetime diagnosis of PTSD had a current diagnosis of the disorder at the time of survey, more than a decade post event (Kulka et al., 1990). Similar findings have been established in relation to Australian veterans, in the Australian Vietnam Veteran Homecoming Study (O’Toole et al., 1996).

This trajectory is neither restricted to military nor North American populations. Hence, the aforementioned Kessler and Breslau analyses of the (US) National Comorbidity Study and Detroit Area Trauma Survey, respectively showed symptoms endured for six and two years for women and men in 30 per cent or more of those populations. Similarly, a study of Australian fire-fighters after a major bushfire demonstrated that 56 per cent of those with PTSD following it had it four years later (McFarlane & Papay, 1992), while the Australian National Survey of Mental Health and Well-Being (NSMHWB) of over 10,000 participants demonstrated the median time to remission is 14 years (Mills et al., 2011).

Consistent with this, the average duration of PTSD has been estimated at seven years (Kessler, 2000). As observed above, course is typically subject to symptom fluctuation in waves, sometimes over decades. To illustrate, a 20 year longitudinal study of Israeli Lebanon War combat veterans noted a decline in their symptoms around three years post-event, but a rise in symptoms 14 years later (Solomon & Mikulincer, 2006). Recent studies have suggested that up to 50 per cent of individuals receiving treatment require between three and seven years of treatment (Steinhert, Hofmann, Leichsenring & Kruse, 2015). Such data have led to PTSD being described as a disorder of recovery and a chronic condition for a significant minority (Shalev, 2009).

Due to the elective and post hoc nature of PTSD research, it is difficult to definitely establish the relative impact of the various factors which are associated with PTSD, its course and the time to recovery. One such factor relates to trauma dose. This is exemplified in the previously described fact that populations at risk for exposure to PTEs via occupational risk are not only known to have higher PTSD prevalence rates, but also extended symptom longevity, police being an archetypal illustration of this (Mayhew, 2001).

Another relates to event characteristics. As observed, is thus known that interpersonal trauma is more associated with PTSD than natural-event related trauma. This is especially the case for horrifying, malevolent and morally-questionable human
behaviour, which is significantly associated with distress prolongation (see, for example, Litz et al.; 2009). Not surprisingly, the course of PTSD is prolonged by the absence of effective treatment (Friedman & Pitman, 2007; Friedman, 2011) and the US National Comorbidity Survey data analysis reported above (Kessler et al., 1995) showed that (median) time to that 50 per cent of cases had recovered, was 36 months for those who had sought professional assistance, but 64 months for those who had not (Kessler et al., 1995). There are many reasons for this treatment gap. These include, client scepticism, failure to apply best practice, the perceived stigmatisation associated with treatment seeking and treatment delaying and interfering anger (see Becker, Zayfert & Anderson, 2004; Hodson, McFarlane, Van Hooff & Davies, 2011; Stephenson & Chemtob, 2000).

As a broad generalisation, research shows that, across populations, about 30 per cent of those affected recover completely, 40 per cent continue to have mild PTSD symptoms, 20 per cent moderate symptoms, and 10 per cent show an unremitting course of chronic disorder (see ACPMH, 2013; Kessler et al., 1995).

2.3.3 The comorbidities of PTSD. Lone occurrence of PTSD is exceptional and it typically occurs in the company of other psychiatric disorders (Brady, 1997; Forbes et al., 2013; Marcinko et al., 2006). Data from large epidemiological studies some time ago estimated upwards of 80 per cent of people with chronic PTSD met criteria for another trauma-related mental health condition (Breslau, Davis, Andreski & Petersen, 1991; Kulka et al., 1990; Kessler et al., 1995). Recent national health surveys in the US and Australia, confirm such estimates, with 86 per cent of men and 71 per cent of women cited as meeting criterion for another disorder (ACPMH, 2013). Comorbid disorders in combat veterans with PTSD have been reported to be as high as 90 per cent (Kulka et al., 1990; O’Toole et al., 1996). Notable comorbidities of PTSD commonly include a range of mood and anxiety disorders (Elhai, Contractor, Palmieri, Forbes & Richardson, 2011; Stander, Thomsen & Highfill-McElroy, 2014). Addictive behaviours are a significant problem and comorbid substance abuse is particularly prominent. The Australian National Survey of Mental Health and Well-Being of over 10,000 participants, found about one-quarter of those with PTSD had an alcohol use disorder and one-third of those with opioid use disorder had PTSD (Mills, Teesson, Ross & Peters, 2006). Among abusers of alcohol and other substances, men have been respectively found to be 6.6 and 7.2 times more likely to have co-occurring PTSD than those without a disorder,
the corresponding ratios for women being 4.5 and 12.4 respectively (Creamer et al., 2001). The two disorder sets have thus been described as causally linked, with additional enduring clinical implications (Najavits, Dolan & Fee, 2012; Stewart, Oumette & Brown, 2002). There is also some evidence PTSD is similarly associated with gambling-related disorders (Biddle, Hawthorne, Forbes & Coman, 2005).

Disgust, guilt and shame and other similar non-anxiety based affects are highly likely to be associated with PTSD. Their (non)presence in the face of horrific or morally-challenging events have recently been argued to offer a potential explanation of the fact that not all trauma-exposed individuals develop PTSD in its acute or chronic forms (Blanco, 2011; Litz et al., 2009).

2.3.4 Risk factors. To understand PTSD it is necessary to take into account the initiating event, resources and deficits the person brings to recovery and context of his/her life (Keane, 1998). Psychosocial risk factors for PTSD may accordingly be classified into three over-arching categories: the nature of the traumatic event, the pre-event characteristics of the individual and the post-trauma environment (Vogt, King & King, 2014).

Just as the nature of the event impacts on the course of PTSD, once established, a plethora of trauma-related factors contribute to risk. These include trauma-type (especially malevolent interpersonal trauma, such as childhood sexual assault), degree of (life) threat and peri-traumatically prominent dissociation or hyperarousal (Roy-Byrne et al., 2004) or early distress onset [as provided for in DSM 5’s acute stress disorder (APA, 2013)]. Personal factors include intelligence level, marital status, education level, genetic predisposition, and health (and mental health) conditions, such as anxiety and mood disorder(s) (ACPMH, 2013). Socio-demographic factors identified include socio-economic status, ethnicity, and age at the time of trauma, race and gender (Blanco, 2011; Mills et al., 2011; Norris, 2005; Vogt et al., 2014). Post-trauma factors include re-traumatisation, life stressors, lack of social support and resources and chronic anger (Blanco, 2011; Hobfall, Tracy & Galea, 2006).

Not all such factors are of equal bearing and meta-analyses and comprehensive reviews (e.g., Brewin, Andrews & Valentine, 2000; Ozer, Best, Lipsey & Weiss, 2003; Vogt et al., 2014) have identified lower and higher-order predictors. These are summarised in Table 2.4 which displays pre-event, event and post-event factors in a lower-to-higher order split. It shows by adopting a stress-vulnerability
paradigm (Meehl, 1962) and ordering predictors by reference to their impact a nested set of risk factors helps explain PTSD sensitivity.

Regardless of their inherent category and weight, however, there is no single causal factor for PTSD. Its development, as outlined immediately above, is best conceptualised as the reflection of a complex mix of biological, psychological and social variables. A stress-vulnerability approach that understands PTSD as the result of an interaction of individual predisposition and trauma-related factors under certain conditions (see McNally, 2001; 2004) is instructive in demonstrating the interplay of risk factors. Vulnerability is derived from the concept of disposition and invokes the notion of latent tendencies. Such tendencies operate as stable traits, are related to negative affect and can be activated, consolidated and exacerbated by childhood, adolescent and adult experiences. Such models purport that stress and vulnerability variables are each necessary for the expression of a disorder (Ingram & Luxton, 2005). Separately, their psychopathogenic impact is curtailed and in either’s absence, pathology will remain unexpressed.

The previously noted central paradox of PTSD - which, to recall, proposes an event must be subjectively appraised as traumatic for its impact to be registered - is explicable on the basis of an individual’s proclivity for neurotic interpretation (see Ingram & Luxton, 2005 for a definition). Some possible mechanistic devices by which this may proceed are illustrated in a review of the importance of cognitive vulnerabilities in the development and maintenance of PTSD by Elwood, Hahn, Olatunji and Williams (2009). Invoking a stress-vulnerability model, they identify four appraisal-affecting cognitive risk factors for PTSD. They comprise negative attributional style - or the tendency to attribute negative past and present events to internal, stable and global causes; rumination - that creates the perception of ongoing threat; anxiety sensitivity - via that, in the style of panic disorder, the individual interprets PTSD symptoms as signs of impending death, insanity or social oblivion; and, looming cognitive style - that catastrophically predicts likely future situations as dangerous and spends little time contemplating how to cope with such situations. Such cognitive vulnerabilities resonate in the treatment setting for clinician and client alike.
### Table 2.4

<table>
<thead>
<tr>
<th>Factor</th>
<th>Lower Order Predictors</th>
<th>Higher Order Predictors</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre Event</td>
<td>Socio-demographic factors, including gender (female), age (young and old) and socio-economic status</td>
<td>Prior traumatisation, esp. personal trauma such as childhood sexual assault</td>
</tr>
<tr>
<td></td>
<td>Personal characteristics such as intelligence level, educational and marital status</td>
<td>Health and mental health</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Cognitive risk factors, such as negative cognitive, affective and predictive style</td>
</tr>
<tr>
<td>Event</td>
<td>Event severity</td>
<td>Physical injury</td>
</tr>
<tr>
<td></td>
<td>Degree of life threat</td>
<td>Trauma type (esp. malevolent interpersonal trauma)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Prominent peri-traumatic dissociation and/or hyperarousal and early distress onset (esp. Acute Stress Disorder)</td>
</tr>
<tr>
<td>Post Event</td>
<td>Number of symptoms</td>
<td>Re-occurring traumatisation</td>
</tr>
<tr>
<td></td>
<td>Duration of symptoms</td>
<td>Lack of personal support</td>
</tr>
<tr>
<td></td>
<td>Co-occurring disorders</td>
<td>Anger</td>
</tr>
<tr>
<td></td>
<td>Co-occurring medical conditions</td>
<td>Preponderance of uncontrolled visual imagery</td>
</tr>
</tbody>
</table>

Sources: Blanco, 2011; Brewin, Andrews and Valentine, 2000; Hobfall, Tracy & Galea, 2006; Mills and colleagues, 2011; Norris, 2005; Ozer, Best, Lipsey and Weiss, 2003; Vogt and others, 2014; Roy-Byrne and associates, 2004

Prominent anger is a risk factor for the development of chronic PTSD. Briefly mentioned here, but detailed in chapter four, this is the case in the short term and over time. Illustrating the former, Riggs, Dancu, Gershuny, Greenberg & Foa (1992) found that, for female assault survivors, clinically significant anger one week post-assault was predictive of PTSD at four weeks. Again in a population of female sexual assault survivors, Feeny, Zoellner and Foa (2000) examined the contribution of
anger and dissociation symptoms on PTSD severity. They found anger, but not dissociation, was predictive of PTSD three months post-trauma. Andrews, Brewin, Rose and Kirk (2000) also found anger, particularly when directed at others, predicted PTSD one month post-trauma in a violent crime survivor population. Illustrating the latter, Koenen, Stellman, Stellman and Sommer (2003) reported on a study of 1377 US Vietnam veterans over a 14 year period. They indicated potent anger at perceived negative community homecoming attitudes existed nine years post US involvement (and predicted PTSD 14 years later in 1998).

Detailed in chapters four and five, out-of-control visual imagery leads to high arousal (Kosslyn, 2005a), is inextricably linked to PTSD and exists as an individual difference of risk factors. Although not a vulnerability per se, when present as in over-preponderance or an out-of-control fashion, imagery is a cognitive variable that is intimately involved in the experience of PTSD and intrusive memories and cognition in the form of visual imagery are acknowledged as the cardinal feature of PTSD and so-recognised in the DSM and ICD classificatory systems. The second focus of this research, the importance of imagery is amply documented in Chapters Four, Five and Six.

In summary, traumatic events are a necessary condition for PTSD’s development, vulnerability factors render individuals more liable to the condition and both must be present for its occurrence (McNally, 2004). Once kindled, the condition is liable to be exacerbated by post-trauma risk factors. The twin foci of this research, anger and out of control imagery, as provided for in DSM III to DSM 5 imagery are emphatic risk factors for the development of chronic forms of the disorder.

2.4 Theories of PTSD

The coining of the term PTSD in the psychiatric nomenclature in 1980 initiated considerable theorising about the condition. Before them, there were several important early theories. Each requires comment.

2.4.1 Early psychological theories of trauma. In 1895, Sigmund Freud and Joseph Breuer published Studies in Hysteria. This contained Freud’s theory of traumatic neurosis centred on intra-psychic fantasy and wish fulfilment. Following criticism over the previous decade, this approach was a substantial revision of his early work that had reliably reported the sexual assault of his clients (Masson, 1984; Wilson, 1995). In this revision, he attributed traumatic neuroses to repressed
childhood psychosexual drives, rather than the toxic impact of childhood sexual assault. Although Freud attempted to retract what became known as the “seduction theory of trauma”, this was not accomplished before his death in 1939. Freud’s contributions to psychotraumatology, in his acknowledgement of the impact of trauma on human coping, its profound effect on human psychological function and the distorted perception, appraisal and memory that can accompany trauma, were somewhat marginalised by his insistence on the role of fantasy and wish fulfilment (Masson, 1984). This fantasy focus, with its Greek mythology motifs, distracted the field from study of the actual phenomenology of trauma and the benefits which would have derived from that. Thus although, notion of fantasy was repudiated by the psychotraumatology of WW I, thinking on posttraumatic reactions went underground post WW II (see Wilson, 1995; Weisaeth, 2014) until the 1970s prelude to DSM III’s description of PTSD in 1980.

An important theory in this early phase of better understanding PTSD was Horowitz’s Stress Response Theory (1976, 2001). Fundamentally, an information processing theory, it holds that, in the development of PTSD, there occurs an initial post-event psychological outcry response phase followed by a denial (avoidance) response phase aimed at protecting the individual from the pain of loss. In turn, avoidance is then followed by intrusive trauma recollections that constitute an attempt at achieving some assimilation or resolution of the event. Thereafter, oscillations between these intrusive and avoidant states occur, with intrusions continuing to represent attempts to process the event and avoidance (numbing or active avoidance) reflecting attempts to manage the associated distress or deal with the memory in tolerable doses. The model holds that this phasic oscillation continues until some resolution is achieved. This model continues to be clinically valuable for its recognition of the cyclical nature of PTSD symptoms.

A conditioning model of PTSD was proposed by Keane, Zimering and Caddell (1985). A social-interactionist learning theory, it was derived from conditioning theories of pathological anxiety, such as classic Pavlovian fear conditioning and Mowrer’s two factor model (Friedman, 2011). Consistent with such theories, it posits that unconditioned stimuli (traumatic events) automatically evoke unconditioned emotional (fear) responses. The intensity of this response generates avoidant protective responses. Subsequently, a broad array of conditioned stimuli (cues and triggers of past associations) acquires fear-eliciting properties through
stimulus generalisation and higher-order conditioning. Consequently, processes of habituation and fear-extinction relating to this range of stimuli are impeded by cognitive, emotional and behavioural avoidance. This model has solid face-validity in clinical settings.

Another important early PTSD theory which has been instrumental in informing thinking about why trauma dose does not entirely predict PTSD was developed by Janoff-Bulman (1989; 1992). Considered a social-cognition schema theory (Cahill & Foa, 2014), it focuses on the impact of trauma on the individual’s world-view (Park, Mills & Edmonson, 2012). It includes three central propositions: (i) that exposure to trauma has the potential to “shatter” key pre-event assumptions held by individuals, (ii) these assumptions are inevitably centred on the benevolence of the world, the meaningfulness of life and self-agency and (iii) the likelihood of assumption-shattering varies according to both the polarity and valence of which these beliefs (Liechty, 2002). These assumptions are not considered to be equally important and it has been observed that individuals may see themselves in negative terms in order to preserve a sense of world-meaning and benevolence (Liechty, 2002). The model continues to resonate clinically and has independent support from other perspectives - Foa, Ehlers, Clark, Tolin & Orsillo (1999), for instance, identified three types of trauma-related appraisals related to the development of PTSD irrespective of trauma type: negative cognitions about self and world and self-blame. These appraisal-types bear strong resemblance to the core of Janoff-Bulman’s model.

2.4.2 Limitations of early theories. None of these theories, of course, represented flawless explanations of PTSD. For example, the two stages of Horowitz’s model are inconsistent with the three and four-factor PTSD models of DSM III, IV and 5. Thus while it is, as observed immediately above, a clinically useful concept for treating clinician and client alike, it takes no account of the importance of cluster D (mood alteration) or cluster E (arousal) symptomatology to the disorder.

Again, while the conditioning theory of PTSD is appealingly parsimonious with its two factor structure (Friedman, 2011), it does not fully account for the trauma-related cues that lead to PTSD, compared to the specific phobias about which the two factor-anxiety model was initially generated. Its most severe limitation, however, relates to aforesaid recognition that various emotions other than fear are associated with PTSD (Gilihan, Cahill & Foa, 2014).
Similarly, the social-cognition schema model is contentious in that it: (a) is based on an understanding of PTSD as an anxiety disorder driven by fear and worry, when there is contemporary evidence PTSD is strongly-related to other emotions, including anger, and that it is best conceptualised as a stress condition; (b) holds that world views are shattered, when it is equally likely that (negative) assumptions may be exacerbated; and (c) fails to recognise the more parsimonious possibility that PTSD is simply associated (albeit as a lower-order risk factor) with prior negative experience (and that an accumulation of negative live events is a sufficient explanation for distress, thereby rendering assumption-shattering unnecessary).

2.4.3 Prominent contemporary theories. Due to such shortcomings, and despite their influential contributions, these early models have not retained their prominence. Cognitive, emotion-processing and integrative theories of PTSD are representative of current thinking.

2.4.3.1 Cognitive theories. Currently, cognitive theories of PTSD are dominated by information processing and appraisal theories. The former emphasise the importance of knowledge structures (i.e., networks and schemas) and the distorting effect of (perceived or actual) stress and trauma on responses to trauma interpretation. In contrast, the latter emphasise that it is the individual’s interpretation of events which determine their response(s) to trauma.

Information Processing Theories

There are several notable information processing theories. Creamer, Burgess and Pattison (1992) initially posited a model whereby, in contrast to Stress Response Theory, intrusion was thought to precede avoidance and the latter was critically implicated in the development and maintenance of the disorder. The intensity of distress associated with severe intrusions would, for those who developed PTSD, result in activation of avoidance processes that impede the processing of the traumatic material. Therefore, rather than facilitating a graded integration of this material, intensity of intrusion and subsequent avoidance processes impeded the natural process of recovery.

A second information processing theory, Dual Representation Theory (DRT: Brewin, Dalgleish & Joseph, 1996), is a multi-representational-structure theory that posits that after trauma, two parallel memory storage and retrieval systems operate. Reminiscent of Paivio’s (1971) dual-coding notion of cognition, which proposes that learning may occur via verbal association or visual imagery, the first DRT system, the
**Verbally Accessible Memory (VAM) system**, reflects narrative trauma memories. **VAMs** are thought to be integrated with other autobiographical memories and consciously available for verbal processing and communication, and relate to potential losses and past regrets; for example, in guilt over opportunities forgone or anger at careless risk-taking. They are voluntarily accessed in “telling the story”, but often lack the detail of re-experiencing phenomena.

The alternate, **Situationally Accessible Memory (SAM)** system, hypothetically contains information from lower-level trauma processing. **SAMs** are restricted to memories experienced peri-traumatically or in subsequent moments of intense arousal and most often are coloured by emotions of fear, helplessness and horror, but can include other emotions, like anger and detailed response propositions. They use a non-verbal code, are difficult to communicate to others and do not necessarily become modified by subsequent autobiographical memories. Dual Representation Theory holds that **SAMs** are triggered involuntarily via reminiscent stimuli and contain information from more-extensive trauma processing, including sensory information bypassed in conscious processing. They are reflected in flashbacks with detailed sensory and response information, devoid of higher order elaboration and context and often triggered involuntarily. This hypothetically accounts for the inability for many PTSD sufferers to voluntarily retrieve detailed accounts of the trauma.

**Appraisal Theory**

Various commentators have referred to the importance of appraisals in the development of PTSD. To recall, there is the central paradox of PTSD (McNally, 2004) which emphasises that events must be subjectively appraised as traumatic for reactivity to occur. Additionally, there are also the aforementioned trauma-related, PTSD-influencing appraisals identified by Foa (1999) and also Bonnano’s (2005) proposition that mental toughness in resilience is influenced by views that life is meaningful, learning can derive from not only positive but also negative events and a belief in self agency also points to the key role of appraisals.

Thusfar, however, only one appraisal-based theoretical model of PTSD has been advanced, that of Ehlers and Clark (2000). It argues pathological post-trauma responses arise when individuals process trauma-related information in a way that produces a sense of current threat. It asserts the primary mechanisms by which this process operates are negative appraisals of trauma and their sequelae and, to a lesser extent, the nature of traumatic memory storage.
Additionally, processing emphasises the meaning of the event, organises the information and places it in context. Higher order information processing capacity, in the context of trauma, facilitates the integration of the trauma memory with existing autobiographical memories. Alternately, data-driven processing is connected to associative memory, focused on sensory impressions and leads to strong perceptual priming. This processing is “cue-driven”, unintentional and consequently difficult to voluntarily retrieve. There is an over-abundance of such data-driven memory that competes for resources with the conceptual processing. This competition results in poorly elaborated memories that are not integrated with other autobiographical memories. This is supported by research that identifies reduced autobiographical memory in PTSD and its rate-limiting effect on trauma memory work (Moore & Zoellner, 2007; Wheatley, Hackman & Brewin, 2009).

The model further proposes a second mechanism, involving negative appraisals including danger, violation of standards of self or others or loss, maintains a current sense of threat. It posits the persistence of these negative appraisals maintains the currency of the traumatic memory and its level of perceived immediate threat. Consequent of this main theory, Ehlers and others (Ehlers, 2010; Ehlers, Hackman & Michael, 2004) advanced a Warning Signal Hypothesis that argues PTSD’s intrusions, which overwhelmingly take the form of visual intrusions, are appraised as replays or warning signals of past event-reoccurrence or future danger. Thus, underlying psychological processes oriented toward threat-related information or hostile appraisal can increase the propensity to maintain a current sense of threat and impede the processing of trauma memories. Strategies that operate to maintain symptomatology include thought suppression, distraction, anger and the adoption of other safety behaviours.

Theoretical and empirical research has provided support for the role of warning signals in predicting PTSD and indeed, several of the competing theoretical frameworks acknowledge the role of appraisals. These include the information processing, social-cognition schema, conditioning and emotional processing theories described above. Empirical support for this exists in research such as the study by Mayou, Ehlers & Bryant (2002) on road traffic accident survivors, whom they described as maintaining PTSD via a feedback loop biased toward threat detection and hostile appraisal.
The Warning Signal Hypothesis is similar to and supported by the cognitive vulnerabilities model for the development of PTSD identified by Elwood and colleagues (2009). Euphemistic of notions of neuroticism, their model emphasises the impact of negative attributional style for past and current-events and looming cognitive style for future-events on PTSD. They summarise the available research, indicating negative attributional style appears to be consistently associated with PTSD in victims of interpersonal trauma by way of stable and global causal attributions. With respect to looming cognitive style, that is in effect a danger schema for predicting future threat potential, they report these defensive representations become cross-sectionally enduring for some individuals and relate strongly to non-coping perceptions about trauma and PTSD symptoms. Elwood and colleagues note evidence that negative attributional and looming cognitive style may both have an internal and external locus, albeit less conclusively for the latter.

2.4.3.2 Emotion processing theory. Consistent with the network theory proposed by Lang (1977), Foa, Steketee and Rothbaum (1989) have argued traumatic memory is an elaboration of a fear network composed of interconnected situation and response threat-oriented propositions. This network included sensory information about the traumatic event (e.g., sights, smells & sounds), response (including cognitive, emotional and physiological responses to the trauma) and meaning-based propositions. Encounters with either internal or external stimuli matching the propositions in this network are thought to activate the network. When there is a failure to expand or broaden the information in the network beyond trauma-related cues or include safety cues, PTSD develops.

Further elaborating the model, Foa and Rothbaum (1998) proposed that beliefs impact upon the trauma memory network at four levels. First, pre-trauma views about the self are represented at each extreme as the self as invulnerable and entirely benevolent at one end of the spectrum and the self as incompetent, helpless or bad at the other. Second, polarities in pre-trauma beliefs about the world are reflected by extreme assumptions that it is safe and benevolent or alternatively that it is constantly dangerous and threatening. Third, beliefs and interpretations about one’s reactions to the trauma and symptomatic responses have the potential to influence the development of the disorder. Finally they stressed the importance of beliefs about other’s reactions that have the potential to influence the level of support-seeking-behaviour.
2.4.3.3 **Integrative or multiple representation theories.** To date, there has been only one attempt to assimilate these various contemporary theories into an overall explanatory model of PTSD, the Schematic, Proposition, Analogue, Associative Representation Systems Model of PTSD (SPAARS; Dalgleish, 1999, 2004; Power & Dalgleish, 1999). While attempting to integrate other theories of PTSD (Iverson, Lester & Resick, 2011; Friedman, Resick & Keane, 2014), the model has an information processing description of PTSD at its core. It proposes that PTSD occurs when alterations in information processing are sufficient to change the individual’s representational structures.

As its name suggests, the model provides for four levels/formats of mental representation. Like Dual Representation Theory (see 2.4.3.1), it asserts information at the propositional level is verbally accessible. Evocative of the concept of synaesthesia, information at the analogue level is stored as images across all sensory systems. These representations are associative, such that information encoded in one format can be associated with information in that or another format. At the time of the event, individuals appraise information at the schematic level due to the intense fear involved. Memories, emotional intrusions and cognitive biases associated with intense emotion lead to maladaptive, avoidance-based coping strategies.

The model proposes that re-experiencing an associated emotion is generated via two routes: automatic elicitation via associative reorientations and schema-level appraisal process by which the individual attempts to assimilate new information. Although there is recent research arguing in favour of the importance of the SPAARS-model as an explanation of the reactions to childhood sexual assault (Coyle, Karatzias, Summers & Power, 2014), a strong supporting set of data does not yet exist. Consequently, the model has been described as comprehensive, but lacking in empirical support (Iverson et al., 2011).

2.5 **Current Status of the Predominant Theoretical Explanations of PTSD**

Prominent explanatory theories of PTSD emphasise the role of appraisals, information processing phenomena and emotion-overload, regulation and networking. They also emphasise the importance of locating each such explanatory factor within a multi-level causal model.

These theories are an important advance on early theorising about PTSD and have lead to clear treatment improvements. None are without limitation, however.
Sharing commonalities with classical conditioning theory, the emotion processing model of PTSD is thought to be able to better account for PTSD phenomena by clarifying the associations between stimuli, responses and meanings. It has lead clear treatment improvements in the contribution of prolonged exposure in the treatment of PTSD’s anxiety component (McNally, 2007). Limitations include its failure to provide an explanation for numbing and dissociation and the aforementioned focus on fear as the primary emotion and danger as the primary meaning of PTSD (Gilihan et al., 2014).

While well-rooted in, and sharing the strengths of, other models of PTSD, including emotion processing theory, and being a clinically useful alternative to exposure in the treatment of PTSD where clients are incapable of tolerating exposure, cognitive theory fails to account for why the addition of cognitive therapy to exposure therapy does not improve treatment outcomes. In contrast, the reverse application of exposure has an additive-effect over cognitive therapy (Gilihan et al., 2014).

A major shared problem across these contemporary theories is that, in line with early diagnostic conceptualisations of PTSD as an anxiety disorder, they have overwhelmingly focused on fear as the predominant affect, to the neglect of other emotions. This focus is incongruent with the recognition that critical emotions in PTSD commonly include non-anxiety based feelings like disgust, guilt and shame. The aforementioned relocation of PTSD into the Trauma and Stressor Related Disorders section of the Manual in DSM 5, along with reactive attachment, disinhibited social engagement disorder, acute stress disorder and adjustment disorder, is a recognition of this fact (Monson et al., 2014).

As laid out earlier in this chapter, these affects are commonly a comorbidity of PTSD and noted for their importance in the burden of the disorder reported by sufferers. Recently, the chronicity of PTSD has been argued to be intimately associated with the event perpetrator’s intention, the event’s meaning and its moral status (see Chung & Breslau, 2008; Litz et al., 2009; Rosen & Lilienfeld, 2008). Disgust, guilt and shame and other similar non-anxiety based affects are highly likely to be associated with such characteristics and their (non)presence in the face of horrific or morally-challenging traumata offers a potential explanation of the fact that not all trauma-exposed individuals develop PTSD in its acute or chronic forms.

Principal among the emotions neglected by contemporary and past theories of PTSD is, however, anger, and it, along with (the strongly associated emotions of
disgust and shame) has been estimated to be the predominant emotion for up to 50 per cent of PTSD presentations (Coyle et al., 2014; Friedman, 2011; Power & Fyvie, 2013). As argued across this thesis, anger is a highly significant problem in PTSD which needs to be more fully recognised as an aetiological factor in the disorder, a predictor of its course and the attendant treatment-response.

As demonstrated, none of the current theoretical accounts of PTSD or their predecessors are able to provide an explanation of PTSD without limitation. One of their primary limitations is that it is not clear that they, and the treatments that derive from them, are able to account for anger in PTSD. An example of this relates to the concern expressed by Foa, Riggs, Massie and Yarczower (1995) about the suitability of prolonged exposure treatment to PTSD where anger is prominent and has the effect of limiting engagement with the feared memories driving the condition.

The need to broaden focus beyond anxiety in explanatory models is increasingly recognised in comprehensive reviews (see Friedman, 2011; Friedman et al., 2014) and justified by empirical examinations (Coyle et al., 2014; Power & Fyvie, 2013). Shalev (2009) notes that the US Institute of Medicine has judged scientific evidence for the treatment of PTSD as below the level expected for such a common and disabling disorder. He summarises the situation as one where significant progress is being made in the disorder’s treatment, but limited by an apparent treatment-ceiling-effect and a need for more efficacious application of better psychological theories.

In pursuing this objective, recent writings about PTSD have identified special features, key questions and emerging topics deserving of increased descriptive, theoretical and empirical research. Targets thus identified have included constructs such as complex PTSD (as per DSM 5), dissociative PTSD subtypes, sub-syndromal/prodromal PTSD, memory processes in PTSD, biomarkers of PTSD, factors that determine resilience in the face of trauma-exposure and even a PTSD genotype (Friedman, 2011; Friedman, Resick & Keane, 2014).

Of concern, there has been little mention of anger as a special feature of PTSD. This is, as described in chapter four, matched by a relative dearth of empirical enquiry into the relationship of anger to PTSD. There is a need to redress this absence via new, modified or adapted theories. This current research is a response to that need and the following chapter reviews the nature of anger and proposed explanatory theories for their relevance to this task.
CHAPTER 3: THE NATURE OF ANGER

How much more grievous are the consequences of anger than the causes of it.

Marcus Aurelius

Chapter Two reviewed the nature of PTSD. It demonstrated that, although there have been important advances in the conceptualisation and treatment of PTSD, available theoretical models are unable to provide a full explanation for the development and maintenance PTSD. It both noted a significant proportion of those individuals who demonstrate symptoms, especially the group with a chronic form of the disorder, experience significant problems with anger and the current limited capacity for explaining this. This chapter, as the next logical step in the attempt to bridge this explanatory gap, reviews what is known about anger and dysfunctional anger from definitional, phenomenological and epidemiological perspectives and the adequacy of the extant explanatory theories of anger’s development and maintenance.

3.1 Historical and Cultural Conceptualisations of Anger

Anger is a universal emotion with specific neuroanatomical underpinnings, developmental characteristics, physiological and behavioural tendencies and bodily and facial expressions that are culturally invariant (Mayne & Ambrose, 1999). It is neither intrinsically pathological nor maladaptive (Novaco, 1976; Pascual-Leone & Paivio, 2013) and its utilitarian value has long been recognised and at times valorised in cultural discourse - for example as righteous anger or the wrath of supernatural forces (e.g., story of Achilles). Outside this research’s central focus on dysfunctional anger, a summary of anger’s positive functions is provided at Appendix B.

More commonly, anger has been understood as a human frailty involving great suffering (Fernandez, 2013; Hollenhorst, 1998). Thus, from antiquity, culturally and societally-informing discourses by landmark philosophers, social commentators and literary figures, as well as critical cultural documents like the sacred texts of universal religions, have negatively evaluated anger. Well-described in contemporary psychological texts (see Novaco, 2007; Potegal & Novaco, 2010; Fernandez, 2013), such cardinal texts and commentaries ably demonstrate anger’s impacts (see key illustrations at Appendix C).
Notwithstanding this, Cavell and Malcolm (2007), in the editorial to one of the few comprehensive texts on treatment interventions for anger and aggression, observe that little is known about anger among practitioners, theorists and researchers. There is a paucity of enquiry such that, for every published journal article on anger, there are seven on depression and ten on anxiety (Kassinove & Sukhodolsky, 1995). The publication rates for the three affects were tested twice during this research (April 2010 and May 2015). Those examinations confirm that, while the rate of increase in anger publications is outstripping those of anxiety and depression, because of anger’s low publication base-rate, the gap between the quantum of published anger and anxiety and depression research is, in raw terms, increasing (see Table 3.1). Novaco lamented this state of affairs over 35 years ago, stating “Anger is paradoxically one of the most talked about, but least studied of human emotions” (1976: 1124). It is clear progress is slow.

In summary, the message is unchanging: anger is elemental, but often destructive and objectively unjustified and counter-productive (Thurman, 2005). Despite this, anger remains under-researched among negative affects and under-appreciated among treaters for its impact.

Table 3.1

<table>
<thead>
<tr>
<th>Decade</th>
<th>Anger</th>
<th>Anxiety</th>
<th>Depression</th>
</tr>
</thead>
<tbody>
<tr>
<td>2011-14</td>
<td>335</td>
<td>9,876</td>
<td>13,467</td>
</tr>
<tr>
<td>2001 - 2010</td>
<td>1,011</td>
<td>25,756</td>
<td>35,678</td>
</tr>
<tr>
<td>1991 - 2000</td>
<td>556</td>
<td>12,725</td>
<td>17,270</td>
</tr>
<tr>
<td>1981 - 1990</td>
<td>248</td>
<td>6,590</td>
<td>11,153</td>
</tr>
<tr>
<td>1971 - 1980</td>
<td>113</td>
<td>3,010</td>
<td>3,906</td>
</tr>
<tr>
<td>Total</td>
<td>2,263</td>
<td>57,957</td>
<td>81,474</td>
</tr>
</tbody>
</table>

Note: Anger and posttraumatic stress disorder and its variants (e.g., PTSD) were used and hits were limited to their adult experience. This eliminated articles on measurement, children, adolescents and health states or issues (e.g., cardiovascular disease) or where PTSD or anger were described co-incidently.

3.2 Defining Dysfunctional Anger

3.2.1 The absence of a formal diagnostic construct. Unlike PTSD, which is defined according to clear and largely common diagnostic criteria in two psychiatric classificatory systems, dysfunctional anger does not have its own diagnostic criteria. Rather it
is identified as a symptom of three disorders in DSM 5: borderline personality disorder, major depressive disorder and PTSD. Intermittent explosive disorder is the only stand-alone anger-related DSM-based diagnosis. It is, however, largely a measure of behaviour, rather than a symptomatic description of experience. The alternate major diagnostic system for mental health, the World Health Organisation’s International Classification of Diseases (ICD), is similarly lacking in anger-specific disorders. Again, while its 9th (1999) and 10th (2012) editions refer to anger symptoms in three disorders - impulse-control, unsocialised-aggressive and conduct-disturbance disorders - it likewise only includes intermittent-explosive disorder as a specific form of anger-related psychopathology. This situation stands in contrast to the extent of symptom description and categorisation for anxiety and mood disorders in both systems.

Given severe anger problems are associated with significant impairment and disability, the general absence of explicit anger disorders in established classificatory systems is surprising (Fernandez, 2013). It is not clear what this absence means. For example, the question has been asked as to whether dysfunctional anger is best understood as a symptom of psychiatric disorders or a clinical syndrome (see Fava, Anderson & Rosenbaum, 1990; Morland, Love, McIntosh, Green & Rosen, 2012). Irrespective of how this quandary may be addressed, the precise nature of dysfunctional anger will not be found in such classificatory systems and there is a consequent need to define dysfunction anger by other means.

3.2.2 A working definition of dysfunctional anger. Defining anger that is dysfunctional is not easily accomplished. In the first instance, anger been described as a clinically neglected (DiGiuseppe, Tafrate & Eckhardt, 1994), misunderstood (Tavris, 1989) and complex emotion (Fernandez, 2013). Researchers have observed anger to be a fuzzy concept (Fehr & Russell, 1994) and that it is difficult to maintain sharp distinctions between it, aggression and hostility (Smith et al., 2004).

Among negative emotions, it is definitionally less-consistently agreed upon and subject to an uncommon level of conceptual debate (Mammen, Pilkonis, Kolko & Groff, 2007; Fernandez, 2013). It has consequently been argued this has resulted in incongruent findings in the literature (Eckhardt et al., 2004; Kassinove, 2007), with the constant conflating of it, aggression, hostility, hatred and violence being a hindrance to the effective conduct and interpretation of research (Ekhardt et al., 2004; Mammen et al., 2007). Reflective of this, there is no apparent distinction between anger and dysfunctional anger in the research or clinical literatures and little agreement about what constitutes an anger problem or whether
problematic anger should be viewed as separate from normative anger (Olatunji & Lohr, 2004).

For the purposes of this research, dysfunctional anger may be understood as a low-prevalence problem that emerges at various widely accepted thresholds (see DiGiuseppe & Tafrate, 2001). Summarising the literature, these thresholds relate to anger frequency, intensity, duration, sensitivity, disproportionately or latency and involve significant: (a) impairment in psychological functioning, involving compromised reasoning, decision-making, problem-solving, goal-setting and intellectual-performance, misperceptions, poor judgement, mistakes and accidents, (b) negative impact on quality of life, (c) social censure based on its abnormality, undesirability and repugnance, (d) interpersonal and relationship harm based on an increased association with behavioural aggression, (e) association with psychopathology, (f) need for intensive and prolonged treatment, (g) physiological burden with a high association with morbidity, mortality and compromised health status and (h) economic cost.

In defining and understanding dysfunctional anger, it is important to distinguish it from the related constructs that confound research. There are two important contrasts to make. The first is with aggression. Although anger has a strong action potential for the discharge of associated behavioural tendencies and is implicated in aggression (Cavell & Malcolm, 2007; Mayne & Ambrose, 1999), they are best understood as discrete phenomena. This is eminently demonstrable. First, each is neither necessary nor sufficient for the other’s occurrence and can exist independently of the other (Cavell & Malcolm, 2007; Fernandez, 2013; Novaco, 1986). This is not well acknowledged by clinicians and researchers, but can be found in landmark observations. Averill’s (1983) classic distinction, wherein he emphasised aggression is only one of many possible manifestations of anger, is a prime example of this. Researchers have observed the two are only moderately related, with some estimates suggesting as little as ten per cent of the experience of anger leads to aggressive acts (Averill, 1983).

Similarly, individuals may act aggressively without anger. This is illustrated, by the often-articulated recognition that there are goal-directed or instrumental acts of aggression which include little or no anger component (e.g., Fernandez, 2013; Greenberg & Paivio, 1997; Hubbard, McAuliffe, Rubin & Morrow, 2007; Hubbard, Romano, McAuliffe & Morrow, 2010). Alternately termed incentive-based, proactive and predatory (see Bond & Wingove, 2010; Hubbard et al., 2010; Mammen et al., 2007), this instrumentality can be found in events and undertakings as diverse as those of sport, business, child’s play, the
behaviour of psychiatric patients and crime (Hubbard et al., 2007; 2010). Such instrumentality is noted to be driven by different psychological processes to the other type of aggression - variably termed reactive, impulsive or emotional aggression (Hubbard et al., 2007; Mammen et al., 2007) - which is noted to be associated with heightened physiological responses and “hot-headedness” and a different neurochemistry (Hubbard et al., 2010).

This distinction is possible in part because many of the phenomena associated with anger do not pertain to aggression. Anger is associated with multiple other emotions and cognitions. Aggression, in contrast, is rarely associated with emotions other than anger and its cognitive elements are restricted and narrowly focused, being concerned with intentionally antisocial, acquisitive behaviour (Kassinove, 2007) involving harsh comment or physical contact with objects or people (Digiosepppe, Cannella & Kelter, 2007).

Additionally, the subjective experience of the two phenomena is also readily distinguishable. Aggression is rarely perceived by its perpetrators in negative terms and, as observed immediately above, when it occurs instrumentally, is devoid of emotion (Greenberg & Paivio, 1997). In contrast, anger is more often experienced as dysphoric, uncomfortable and aversive (Kassinove & Sukhodolsky, 1995; Olatunji, Ciesielski & Tolin, 2010) and is typically perceived as provoked (Fernandez, 2013; Novaco, 2007; Potegal, 2010).

Finally, as observed repeatedly across this work, anger can be “intra and interpersonally directed” (Van Kleef, 2010). In contrast, aggression requires an external target (see Berkowitz & Harmon-Jones, 2004; Fernandez, 2013). This is the case for the goal directedness of instrumental aggression identified above or the conceptions of instrumental aggression as proactive and reactive aggression (Hubbard et al., 2007; 2010).

The second such contrast that is important to make is with hostility. As noted, there has been a tendency to interchangeably utilise anger and hostility as descriptors and constructs and contemporary scholarship notes the difficulty of maintaining distinctions between the two constructs. It is possible, nevertheless, to discriminate them. Content-wise, hostility’s central cognitive features are thought to involve cynicism (judging others to be selfishly motivated), mistrust (believing others will be hurtful and intentionally provoking) and denigration (evaluating others as dishonest, ugly and mean) (Eckhardt, Norlander & Deffenbacher, 2004). These are not necessarily the characteristics of anger. In addition, the breadth of anger’s content phenomena exceeds the narrow set associated with hostility, which are inevitably bound up with interpersonally and socially-directed anger infused with a flavour of retribution. As detailed in the next subsection, this can relate to other emotions, injustice, contrariness, diminishment and social-role expectation and norm application failure.
Finally, anger and hostility are also subjectively distinguishable. It is hostility’s attitudinal disposition that effectively discriminates it from anger. It has been described as characterised by a negative valence toward and unfavourable judgement of others and a complex set of feelings which motivate aggression and vindictiveness (Eckhardt et al., 2004). These phenomena need not and often do not characterise anger, even in its high valance forms. Lastly, hostility is a smouldering, enduring state. Anger can be cross-sectional or enduring in experience (Fernandez, 2013).

3.3 The Phenomenology of Anger and Dysfunctional Anger

Anger is experienced as aversive and dysphoric. Dimensional in intensity, it ranges from mild annoyance to fury and rage (Fernandez, 2013; Harmon-Jones et al., 2010). Mild-to-moderate anger is commonplace and is more likely to be elective. Extreme manifestations of anger (dysfunctional anger) are less common, but not unusual may be subjectively experienced as avolitional.

The focus of anger is has said to relate to social injustices and interpersonal wrongs and one of anger’s positive qualities is the impetus or energy it can give to the righting-of-wrongs (see Appendix B). This has been observed in relation to occurrences such as human rights violations (Silove et al., 2009) and gender or class-based discrimination (Fischer & Evers, 2010). Catastrophically, when carried to extremes, anger at social injustices can motivate gross acts of anti-social behaviour, such as terrorism (Giner-Sorolla & Maitner, 2013) those acts in turn fuelling a sense of injustice by the targeted parties and on occasions national senses of injustice and demands for retribution (Beck, 1999; Potegal & Stemmler, 2010a).

Dysfunctional anger is not only related to such justice-based concerns, but is also concerned with multiple other perceptions, appraisals and related mental contents. Indeed, an extensive range of triggers have been cited as salient to anger in comprehensive reviews (see Beck, 1999; Fernandez, 2013; Novaco, 2007, 2013). Thus, those with dysfunctional anger report a multiplicity of factors linked to their anger. Table 3.2 documents the plethora of anger-associated mental phenomena, cognitions included, nominated by angry PTSD treatment seekers and offers six overarching thematic categories for understanding the array of possible triggers. Drawn from individuals engaged in programmatic treatment for PTSD (see Table footnote and chapter eight for a description of these individuals and the site of their treatment), these categories correspond to the broad consensus that anger-triggers may
Anger can be understood in terms of threats to autonomy, authority or reputation, disrespect, norm-violation and injustice (Potegal & Stemmler, 2010a).

Anger can be manifested intra and inter-personally. It is, however, described as a socially constructed (Averill, 1982) and relational emotion that requires the involvement of others and is typically directed towards them (Berkowitz & Harmon-Jones, 2004). This is particularly so with dysfunctional anger, where external attributions of fault can be an overwhelming preoccupation. This can be seen in the prominent blaming themes that riddle anger-affected relationships (Beck, 1999; Dutton, 2010), the righteous anger and consequent revenge fantasies of those who feel victimised in the workplace (Tripp & Bies, 2010) and the targeting by traumatic injury patients of health care providers and compensatory health and legal systems (Fernandez & Wasan, 2010). That externalising function is rarely ascribed to other emotions (e.g., anxiety) or disorders, bar angry presentations of PTSD. Accordingly, dysfunctional anger can be understood as a pre-eminent externalising affect.

Commonly, this externalising effect of dysfunctional anger the perception relates to perceived (actual or otherwise) that others are or have been deliberately provocative. To illustrate, in a study of what generally made people angry, Harris (1993) identified five provokers: insensitive and condescending behaviour, harm of others, dishonest behaviour, insults and incompetence. The list of what is provoking has grown since and other authors have added the further anger-provokers of demandingness, challenging and/or individualistic behaviour, social norm violation, behaviour that is contrary-to-expectation, being ignored, demeaned, disrespected or devalued and not receiving recognition and courtesy (see Novaco, 2007; Potegal, 2010; Potegal & Stemmler, 2010a).

Anger often masks the experience of other negative affect(s). This can be illustrated by reference to its interaction with anxiety and depression. Their experience can be sufficiently distressing for various forms of behavioural avoidance to be (un)intentionally invoked in an effort to palliate them. The misuse of substances, isolation, busyness and dissociation are well-recognised examples of this. What is not as readily understood is the significant function anger can play in the avoidance of such affects. This is specifically accommodated in Greenberg and Paivio’s (1997) primary-secondary emotion taxonomy and Beck’s model of anxiety (see Beck & Emery, 1985), which both emphasise the tendency to replace incapacitating distress with anger’s action-orientation. These theoretical viewpoints have been substantiated in research of various anger presentations that stresses the association between anger and other disabling states and conditions (Paivio & Carriere, 2007).
Alongside this, anger can be experienced as a seductive affect and is considered hedonic in nature (Berkowitz & Harmon-Jones, 2004; Harmon-Jones, Petersen & Harmon-Jones, 2010), more than other negative emotion, open to being experienced positively (Tafrate, Kassinove & Dundin, 2002) and associated with an approach motivation (Harmon-Jones et al., 2010). There occurs a clear disparity between pre-act beliefs about (positive) consequences and post-occurrence recognition of its negative outcomes where there is dysfunctional anger. This can be seen in the risk underestimation, reduced loss aversion and overestimation of control (Harmon-Jones et al., 2010; Litvak, Lerner, Tiedens & Shonk, 2010; Potegal, 2010) associated with anger.

As a result of such processes, individuals with dysfunctional anger report compromised psychological function resulting in cognitive distortions, degraded skill performance, misjudgements, mistakes and accidents (Litvak et al., 2010; Mammen et al., 2007). There are also deviations from rational behaviour, relationship-harming self-interested behaviour and increased risk taking behaviour (Guth, Schmittberger & Schwarze, 1982; Lerner & Keltner, 2001). It can also be seen in selective processing of information to anger-congruent stimuli (DeSteno, Petty, Racker, Wegner & Braverman, 2004), diminished task-focus and loss of awareness, perceptual biases and loss of accuracy and anger-associated decreased depth of information processing (Tiedens & Linton, 2001).

Laboratory-based and naturalistic studies have demonstrated that reasoning, decision making, problem solving, goal-setting and diminished use of objective appraisal may also be compromised (Hamilton & Warburton, 1979; Litvak et al., 2010; Tiedens & Linton, 2001), given certain anger thresholds. This has been demonstrated across diverse tasks showing erroneous attributions of causality in ambiguous situations (Keltner, Ellsworth & Edwards, 1993), the tendency to more punitive judgements about individuals in mock civil law tort cases through anger induction even though the source of anger had nothing to do with the fictional case detail (Goldberg, Lerner & Tetlock, 1999; Lerner, Goldberg & Tetlock, 1988) and the association of anger and prejudice, with researchers showing that angry individuals were slower to assign positive attributes to groups to whom they did not belong (DeSteno, Dasgupta, Bartlett & Cajdric, 2004).
Table 3.2

*Common perceptions, appraisals and emotions in anger*

<table>
<thead>
<tr>
<th>Danger</th>
<th>Emotions/states</th>
<th>Contrariness</th>
<th>Injustice</th>
<th>Diminishment</th>
<th>Expectations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Perceived:</td>
<td></td>
<td></td>
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<td>Unmet/misplaced:</td>
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<td>• risk (physical &amp; psychological) to self/significant others</td>
<td>(dis)stress</td>
<td>disagreement</td>
<td>unfairness</td>
<td>ignored</td>
<td>rights</td>
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<td>• attack or threat to self/significant others</td>
<td>worry, fear</td>
<td>challenging</td>
<td>wrongdoing</td>
<td>diminished</td>
<td>wants</td>
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<td>• other’s failure to see risk or danger to self of significant others</td>
<td>insecurity</td>
<td>individualism</td>
<td>moral</td>
<td>disrespected</td>
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<td>• vulnerability (physical &amp; psychological)</td>
<td>tension</td>
<td>“non-team” behaviour</td>
<td>transgressions</td>
<td>disrespected</td>
<td>needs</td>
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<td>• recklessness</td>
<td>jealousy, envy</td>
<td>defiance</td>
<td>malfeasance</td>
<td>devalued</td>
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<td>• impulsivity</td>
<td>hatred</td>
<td>disloyalty</td>
<td>plotting</td>
<td>shamed</td>
<td>standards</td>
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<td>• carelessness</td>
<td>annoyance</td>
<td>“passivity”</td>
<td>culpability</td>
<td>humiliation</td>
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<td>• mistakes</td>
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<td>• other’s (pre)caution failure</td>
<td>vengefulness</td>
<td>ambivalence</td>
<td>discrimination</td>
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<td>• bullying</td>
<td>guilt and shame</td>
<td>disengagement</td>
<td>revenge</td>
<td>derogation</td>
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<td>• intimidation</td>
<td>embarrassment</td>
<td>selfishness</td>
<td>overreaction</td>
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<td>• coercion</td>
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<td>• malevolence</td>
<td>loss, grief and despair</td>
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<td>“sloth”</td>
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<td>depression</td>
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<td>powerlessness</td>
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<td>sadness and sorrow</td>
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<td>remorse, regret</td>
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<td>suspicion, paranoia</td>
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<td>perfectionism</td>
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1 This table’s contents are derived from two decades years of within-session enquiry of participants of comprehensive PTSD group treatment program (GTP) as to what thematically contributes to their anger. That GTP is described in chapter eight.
Anger has distinct, strongly inter-connected, cognitive, behavioural and sensorimotor/autonomic facets (see Cox & Harrison, 2008; Eckhardt et al., 2004). Across cultures, these phenomena include pacing, bodily tension (e.g., fist-clenching, facial expression and aggressive posturing), a range of facial expressions (e.g., grimacing, scowling and glaring) and other phenomena such as body temperature, flushing and hyper-hydrosis (Kovesces, 2010; Stemmler, 2010). Anger’s physiological impact is, the literature suggests, greater than any other emotion. Discussed in greater detail in chapter five, Stemmler (2010) offers an apt summary of anger’s impact on physiology. He notes that a meta-analysis reported by Cacioppo, Berntson, Larsen, Poehlmann and Ito (2000) concluded that negative emotions have a stronger autonomic nervous system impact than positive emotions, but cannot be discriminated. A specific meta-analytic contrast of fear and anger as the top two impacting negative emotions on physiology by Stemmler, however, yielded a number of important differences. Stemmler also cites strong changes in systolic and diastolic blood pressure, heart rate, skin conductance responses and muscle reactivity. He interprets these as alpha-adrenergic activation that creates an increased alertness, vigilance and preparedness to (re)act and which has a functional value for the pursuit of superiority and the avoidance of failure.

To be clear, using these distinctions and the above definitional distillation, this research focuses on the affective dimension of anger, albeit in dysfunctional form, as manifested in the context of PTSD.

3.4 The Scope of the Problem of Dysfunctional Anger

3.4.1 The commonality of anger and dysfunctional anger. It has been observed since the early 20th century that most people experience mild-to-moderate anger from several times per week to several times a day (see Averill, 1983; Kassinove, Sukhodolsky, Eckhardt, & Tsytserav, 1997). Similarly, the contemporary anger literature observes that almost all people get angry at least once-monthly and emphasises the normality of the anger experience (Pinker, 2011).

In contrast, there is no similar consensus about the prevalence of dysfunctional anger. While the definition articulated earlier in this chapter (see page 41) represents a potential means for operationally separating anger from dysfunctional anger, establishing the latter’s prevalence is neither the objective, nor within the scope, of this research. A currently inescapable limit for researchers attempting to investigate the extent of the problem of
dysfunctional anger, this absence requires an estimate to be drawn from clinical research and literature. The following represents such an estimate.

Dysfunctional anger appears substantially less common than its base-level affect. This is not surprising, given emotions are dimensional in nature and normally distributed and extremes of expression are inevitably uncommon. Accordingly, the DSM series and large epidemiological studies, like the US National Comorbidity Survey Replication Study (Kessler, Coccoaro & Maurizio, 2007), place the twelve-month Intermittent Explosive Disorder (which requires repeated anger episodes disproportional to any precipitating event) prevalence rate at four per cent.

An increased incidence of dysfunctional anger is known to be associated with certain demographic characteristics. Youth, maleness, marginalised racial groups and lower-education levels have thus been implicated in problematic anger (Capaldi, Knoble, Short & Kim, 2012; Romanov et al., 1994), while socio-economic status has been shown to be inversely related to anger intensity and duration (Potegal, 2010). Interestingly, research has shown anger is inversely related with age (Capaldi et al., 2012; Potegal, 2010). This is true of trait and episodic anger, with the former declining from middle age (Zimprich, 2012), while for the latter researchers have reported that anger frequency and intensity, but not duration, decline with age (Potegal, 2010; Schieman, 2010). While not commonly acknowledged, men and women have equal potential for experiencing and expressing anger (Fischer & Evers, 2010; Schieman, 2010). There appear to be gender-specific anger expression styles, for example, whereby men are more inclined to physical and verbal antagonism, while women are inclined to cynicism and passive anger and are more verbally-expressive and less physically-expressive (Biaggio, 1989). The literature, nonetheless, understand the incidence of anger is understood as gender non-specific (Fischer & Evers, 2010; 2013). Interestingly, women’s anger is subject to cultural display rules which de-legitimise its public expression (Fischer & Evers, 2010). Such factors obscure the cross-genderness of anger and the equipotentiality of men and women for experiencing and expressing anger (Fischer & Evers, 2010; Schieman, 2010).

Although dysfunctional anger is a low prevalence problem societally, this is not the case in treatment settings. There, along with anxiety and depression, it has been identified as the most commonly encountered problem-emotion (Fernandez, 2002), such that the group is known as the “big three” (Frisch, 2006), the “unholy trinity” and “FAD” of negative affect

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2 Fear-Anger-Depression (those authors’ pun)
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(Zuckerman, 1980). This literature has noted the inter-connectedness of anger to its triumvirate partners.

To summarise, anger is a fundamental emotion which occurs semi-regularly for the vast majority of people without particular negative impact. Humans are not programmed for anger and dysfunctional anger is a low-incidence abnormality that significantly associated with psychopathology and much-deserving of clinical attention.

3.4.2 The trajectory of anger and dysfunctional anger. It has been observed that to understand anger, a critical staring point is its temporal dynamics (Potegal, 2010). Its appreciation is also fundamental to the discrimination of anger from dysfunctional anger.

The typical onset and course of an episode of anger is well-described. With few exceptions - for example, the Japanese experience of anger appears to be briefer and less intense than that of Americans (Schieman, 2010) - the trajectory of anger, albeit subject to cultural display rules, is largely consistent cross-culturally (Kovecses, 2010; Matsumoto, Yoo & Chung, 2010).

The available literature indicates anger’s manifestation is such that most episodes typically vary upwards from five minutes, but last less than 30 minutes. Within an episode, its peak is most often recorded close to episode commencement and anger rises more quickly than it falls (Potegal 2010). Interestingly, anger duration is affected by its directionality, with a lesser duration occurring when anger is directed internally (Wranik & Scherer, 2010). The impact of externally directed anger is substantially increased when familiar individuals are involved compared to strangers and, illustrative of this, domestic episodes of anger are more intense, but briefer, than at the workplace (Potegal, 2010).

Priming or the encoding of information in memory, with a subsequent increased capacity to recall this or related information from cuing, intentionally or otherwise (McNally, 2003), robustly exacerbates angry responses to subsequent trivial events (Mammen et al., 2007; Potegal, 2010) and repetitive anger episodes lead to automaticity and secondary episodes occur within 10 to 20 minutes of the first (Potegal, 2010). Episode duration increases with repetition and intensity, where there is priming via repeated (imagined or real) provocation and anger escalates in a non-linear fashion and significantly outlasts event duration. Thus provoked, anger can escalate even when the provocation remains constant (e.g., a constant low-level annoying sound) and the sequence of escalation is known to proceed via reproaches, insults and threats (Potegal, 2010). Priming, perceived or actual provocation and externally-located causation are associated with rumination. When in the
form of revenge fantasies, rumination can have a half-life of more than a couple of weeks (Tripp & Bies, 2010).

The duration and, concomitantly, rate of anger decline is influenced by several variables. Foremost among them is the inability to control and cease rumination. Other important factors include disruption of anger due to fear, the presence (or not) of distraction possibilities and presence/absence of apology (see Berkowitz, 2010; Potegal, 2010). Accordingly, where there is poor control of rumination and an absence of potentially distracting factors and apology, anger is likely to be prolonged.

3.4.3 The comorbidities of dysfunctional anger. When present with clinical intensity, anger is commonly comorbid with other negative emotions. Considered a “moral emotion” that occurs in response to perceived failure to meet social norms (Digiuseppe & Tafrate, 2001; Hutcherson & Gross, 2011), and concerned with norm enforcement (Fessler, 2010; Wranik & Scherer, 2010), anger has been shown to impact on, and be impacted upon, by guilt and shame. The angriest people often have strong underlying feelings of guilt and shame (Tangney, Wagner, Fletcher & Gramzow, 2001; Tangney, Wagner, Hill-Barlow, Marschall & Gramzow, 1996a). Shame-proneness is thought to be related to anger arousal, resentment, irritability and associated with indirect/non-expression of anger (Hoglund & Nicholas, 1995). These affects often share an affective-content overlap, are recursively interdependent in their dysphoric effects and, given sufficient intensity, present as psychopathology.

Dysfunctional anger can co-occur with a range of mental health and mental state issues. These include specific disorders (e.g., morbid jealousy and psychosis) and temperament and personality pathology, with borderline, antisocial, narcissistic and paranoid personality disorders being cited as exemplars of this relationship (Kassinove & Tafrate, 2002; Lemerise & Dodge, 2008; Novaco, 2007; 2010; 2013). Problematic mental states (e.g., impulsivity, paranoia and irritable obsessionality) (Novaco, 2010) and traumatic stress are likewise strongly associated with dysfunctional anger.

3.4.4 Risk factors for dysfunctional anger. Research describing specific risk factors for dysfunctional anger is limited, not easily located in the clinical and research literature and to some extent must be inferred. Table 3.3 summarises what may be discerned. At the level of individual psychological factors, an angry cognitive set is critical to the development and maintenance of anger. Biases in appraisal of events, attributions of causality and underlying beliefs, dysfunctional schemata and values supporting anger, aggression and violence as acceptable ways of solving conflict or disagreements are likely to
be associated with dysfunctional anger (Bond & Wingrove, 2010; Fessler, 2010; Tripp & Bies, 2010). Somewhat ironically, cognitions associated with disapproval of social norm-violation and enforcement and, especially, morally questionable behaviour by self and others, are also strongly associated with negative appraisals about anger. Ongoing subjective states - for example, rejection or humiliation and shame and anger suppression (Alpert & Spillman, 1997; Martin & Dahlen, 2005; Tangney et al., 1996; 2001) - are also powerful predictors of dysfunctional anger.

Dysfunctional anger is well-established to increase where there is disinhibition due to impulsivity or disinhibition due to mental illness or acquired brain injury (Novaco, 2010) and intoxication due to abuse of alcohol and illicit substances (Najavits, 2002; Norstrom & Paper, 2010; Reilly, Shropshire, Durazzo & Campbell, 2002). Relapse in alcohol abusing men is strongly related to dysfunctional anger (Kelly, Stout, Tonigan, Magill & Pagano, 2010) and alcohol dependence is associated with higher trait anger (Giancola, 2002; Giancola, Josephs, Dewall & Gunn, 2009). In turn, anger in the context of problematic alcohol or drug use increases the risk of severe violence by between 70 and 160 per cent (Pan, Neidig & O’Leary, 1994). While the precise causality of this relationship is yet to be determined, anger has been argued to be a mediator of substance abuse disorders (Barbour, Eckhardt, Davison, & Kassinove, 1998).

Several developmental risk factors are important. The impact of family environment on the development of dysfunctional anger can be profound and it is known anger aggregates in families (Matthews et al., 1992). Dysfunctional anger’s development has been shown to be directly related to angry parenting operating via a range of effects, primary among them modelling and behavioural contingencies (Conger, Neppl, Kim & Scaramella, 2003). Other variables identified as increasing the risk of anger and aggression include experiencing physical, sexual or psychological abuse or witnessing physical or psychological abuse as a child. Abnormally early childhood anger and childhood/teenage violent behaviour (especially cruelty to animals) are also considered a strong predictor of later anger problem (Capaldi et al., 2012; Martin & Dahlen, 2005; Micels et al., 2003).

Demographic risk-factors for dysfunctional anger are inherently difficult to identify, given the lack of anger-disorder status and reluctance of people to self-report it - for example, via large-scale data collection in surveys, such as national census collections - and have consequently not been detailed in depth. The aggression risk factor literature is comparatively well-developed and without resiling from the earlier identified importance of differentiating angry affect from its behavioural correlates, a combination of the extant anger findings and
cautious extrapolation of those from the personal and social aggression and violence literature aptly describes the predictive factors of dysfunctional anger.

Long-standing demographic research into dangerousness, aggression and violence has illustrated that the extremes of age are inversely related to aggression, with adolescence and early adulthood being the times of aggression’s greatest incidence. Of particular note, however, is the strong predictive value of abnormal early age anger (Snyder, Schepferman, Brooker & Stoolmiller, 2007). That literature has also confirmed the role of socio-economic status (SES) in aggression education level and social security status as proxies for SES (see Capaldi et al., 2012; Hastings & Hamberger, 1997; Hubbard et al., 2010; Monagan & Steadman, 1996). The anger literature also highlights this general association, by noting that economic status is inversely related to anger frequency, intensity and duration (Novaco, 2007; Potegal, 2010).

Culture and gender are not risk factors for increased anger (Capaldi et al., 2012; Fischer & Evers, 2010; 2013). There are, however, a small number of important gender differences that are related to the expression of anger. For instance, angry rumination is more common among men than women and associated with the ongoing experience of male anger (Tripp & Bies, 2010). Simultaneously, men are more inclined to physical and verbal antagonism, while women are inclined to cynicism and passive anger and are more verbally expressive and less physical (Biaggio, 1989). Indicative of this, there are gender differences in the trajectory of anger, such that women’s anger episodes are, by self-report, inclined to last longer than men’s (Fischer & Evers, 2010; Tripp & Bies, 2010; Wranik & Scherer, 2010). Cultural factors, although not typically regarded as a specific risk factor for anger, are implicated in dysfunctional anger by dint of their overlap with socio-economic predictors.

Negative life events and dysfunctional anger are positively correlated (Scherwitz et al., 1991) and there is evidence its incidence is sequentially greater where there are stressful and cumulatively stressful events, especially those that are traumatically-stressful. Well known in military personnel, this is documented among various other occupational groups, like police, staff of hospital emergency departments and mental health professionals (Michels, Probst, Godenick & Palesch, 2003; Sugimoto & Oltjenbruns, 2001; Wilson et al., 2001). Researchers have variously noted that stress is positively associated with aspects of anger, such as (dys)control (Germain, Kangas, Taylor & Forbes, 2015), expression and temperament. This association has been shown under experimental and naturalistic research conditions (Taylor et al., 2009). As hypothesised in stress theory (see subsection 3.5.2.5), this relates to both real and perceived stress.
To summarise, anger is a cardinal human emotion that semi-regularly occurs for the vast majority of people without particular negative impact. To recall Pinker’s caution, people have the potential for pro and anti-social states and behaviours. Consequently, dysfunctional anger may be understood as an emotional aberration of low-prevalence. An incisive literature about its commonality, trajectory, comorbidities and risk factors remains to be established. Nevertheless, by referring to a threshold framework involving frequency, intensity, duration, sensitivity and latency and drawing upon pertinent research data, a picture of the nature of dysfunctional anger may be formed. It indicates dysfunctional anger is identifiable by reference to its: (a) blaming externalisation of other, loved ones and family included; (b) high-impact in the face of incongruent trivial triggers; (c) a repetitiveness that facilitates
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priming of angry mood and which occupies a significant amount of waking and sleeping time; (d) non-linear escalation of anger-intensity across episodes, such that there develops an automaticity and blanket-application of angry affect to a broad range of stimuli (e), strong interconnection to other negative affects; (f) increased duration and a leptokurtic anger peak that is maintained, with anger episodes of greater than 30 minutes being a point of demarcation into dysfunctionality; (g) strongly perceptual, interpretive and idiosyncratic character; (h) significant association with rumination, such that preoccupation with “provocations” can last for weeks; and (i) significant expressed emotion and, in the case of men, an increased likelihood of behavioral aggression. It also demonstrates that chronically dysfunctional anger is much more likely to develop where certain risk factors exist. These include maturational experiences (e.g., childhood exposure to anger), personal qualities (e.g., negative temperament or attributional style), the nature of events associated with anger (especially, but not confined to, malfeasance and immoral wrong-doing), cognitions (especially causal appraisals), emotion characteristics (i.e., anger dysregulation) and ongoing stress and, especially, traumatic stress.

3.5 Theoretical Models for Explaining Dysfunctional Anger

As noted, concern with dysfunctional anger dates from antiquity. From the early-to-mid 20th century, psychological theories have sought to explain its aetiology and maintenance. Such perspectives have arisen as part of general theorising about psychological constructs and processes, as well as specific theories about anger. The following depicts those perspectives.

3.5.1 Important early theories. In the early-to-mid 20th century, in the attempt to explain the aetiology of anger, two theories were prominent. One, the Frustration-Aggression Hypothesis (Dollard, Miller, Doob, Mowrer & Sears, 1939), proposed that frustration (i.e., the thwarting of goal-attainment) always leads to some form of anger or aggression. Because of peer critique, which asserted anger was not an inevitable consequence of goal-thwarting only, Miller, Mowrer, Doob, Dollard & Sears (1958) revised the theory to argue frustration may instigate different types of response, one of which is some form of anger.

The other theory of prominence understood anger as instinctively human and reflective of drives and appetites. Articulated most emphatically in anthropological and ethological domains, this understanding is reflected in works such as The Predatory Transition from Ape to Man (Dart, 1953), Demonic Males: Apes and the Origins of Human
Violence (Wrangham & Peterson, 1997) and On Aggression (Lorenz, 1966). This “anger as instinct” model was also provided for by psychological drive theories (see Hull, 1943, 1952 and Spence, 1956, 1960) which proposed that rather than existing as separate, specific urges, anger and aggression are best understood as part of more general instincts, like reproduction, feeding or defence (Scherer, Abeles & Fischer, 1975).

Psychoanalytic theory did not develop a focused, easily testable theory of anger. Nevertheless, the psychodynamic perception is relevant in light of its influence on psychological thinking generally. Freud’s writings stand as an apt summary of this view. He emphasised that humans are instinctively aggressive by nature and that effective management of anger requires cathartic expression that allows individuals, groups and even societies (Pardeck, 1982). Novaco in one of his several scholarly summaries of the history of anger theorising (2007) has paraphrased this view of anger as one in which “outward aggression is an expression of the death instinct in the service of Eros […] and […] any restrictions of aggression directed outwards increase self destruction” (page: 27). The residual influence of this psychoanalytic view of anger may be discerned in the drive-like automaticity and determinism implicit to “anger as frustration” and instinctual models of anger.

Despite their initial impact, celebrated theoretical underpinnings and, in some senses, popular appeal, neither the frustration, aggression nor the ethnological model gained or retained ascendancy. Regardless of the former’s revision, and the inherent utility of understanding that frustration can lead to anger, it was recognised that other psychological factors must be taken into consideration if a fuller understanding of anger causation is to be gained. Consequently, the “anger as frustration” theory lost prominence, although it is still thought of as having some explanatory power.

Similarly, the “anger as instinct” model had initial appeal, but was quickly contested. In contrast to its central proposition, that humans are innately aggressive and anger leads to aggression, there is a considerable contrary scholarship which emphasises that: every vertebrate species uses some form of intra-species violence; all animals have evolved strong inhibitory mechanisms which enable them to suppress aggression when it is in their interest to do so. Further, anger expression is highly susceptible to environmental and social control and reduced by social sanctions (see Clore, Ortony, Dienes & Fujita, 1993). Sympathetic to this view, Grossman (1996), in an analysis of the psychological impact of killing, emphatically argues that, rather than having the nature of natural born killers, humans must be exposed to intense and prolonged training to override the native prohibition on killing, even in warfare. He cites evidence over many wars in different eras demonstrating the
Anger tendency to not fire weapons on the enemy that is neither explained by fear nor the absence of courage. This position was itself taken to an unviable extremity in the United Nations’ Seville Statement (1989) on anger, aggression and violence. To paraphrase that Statement, it concludes it is incorrect to assert humans: have an inherited tendency to aggression and violence; are genetically programmed to be violent; have been selected for aggressive behaviour more than other in their evolution; and, have a “violent brain”.

The debate over whether humans are or aren’t imbued with a primal disposition to anger is to some extent specious. A commonsense third way of better-understanding the situation has been advanced by Pinker (1997; 2011), who asserts that, although the current era is the least violent in human history, and anger and aggression levels have been gradually in decline since Roman times, the human potential for anger and aggression cannot be ignored. There is, he proposes, a constant contest between human Inner Demons (of anger and destruction) and Better Angels (of benevolence and altruism). Novaco’s position (2007, 2013) further finesse this clarification, stating as it does that it is the (dys)regulation of anger that is the issue in anger, not whether it is primal or not.

3.5.2 Prominent contemporary theories and models. Comprehensive texts and reviews (e.g., Cavell & Malcolm, 2007; Potegal, Stemmler & Spielberger, 2010) indicate contemporary anger theories can be ordered by reference to several theoretical perspectives; namely, those emphasising: temperament, disposition and personality factors (i.e., internality/externality/trait models), cognitive processes (i.e., information appraisal and motivational theories), emotions (e.g., regulation deficit models), social interactionist perspectives (i.e., learning theories) and integrative views (e.g., neo-associationist and componential appraisal accounts). Each is described in turn.

3.5.2.1 Dispositional Models. Two dispositional models have been developed to explain anger in PTSD. The first is concerned with the direction-tendency invoked in the experience and expression of anger or, as it is termed, its internality-externality.

At a conceptual level, internality and externality have classically been used to describe emotional experience and more commonly the personality-associated behavioural expression of emotion as reflected in the work of Rotter (1975). Since the 1990s, it has additionally been suggested that internalisation and externalisation, and their links to personality, are a means to better understanding psychiatric disorders and their organisation in classificatory systems (Krueger, McGue & Iacono, 2001). Proponents of this view, using factor analytic studies, have suggested anxiety and mood disorders are examples of
internalising disorders (Watson 2005), while addictive disorders and antisocial disorders are examples of externalising disorders (Krueger & Markon, 2006).

Applied to anger, internalisers are able to be understood as initially more trusting but when frustrated or hurt by someone they are inclined to act out aggressively. Externalisers are distrustful and passively accept the unkind actions of others which re-confirm their already sceptical views of others (Singer, 1995). Dispositional externally directed anger is most associated with the aforesaid health and mental illness outcomes. To illustrate from the physical health literature, Williams, Nieto, Sandford and Tyroler (2001) suggest angry temperament predisposes middle-aged, normotensive persons to a significantly greater risk of myocardial infarct or sudden cardiac death than anger aroused over circumscribed stimuli such as frustration, criticism or unfair treatment. They conclude it is the intense, volatile aspects of anger proneness, not angry reactions, which are the more potent link to CHD. By its nature, an angry temperament can be a more powerful initiator and sustainer of the pathophysiological changes leading to CHD and cardiac death. Consistent with this, but offering an important qualification, is the robust literature implicating the inhibition or suppression of anger as key to such anger-CHD relationships (see Chida & Steptoe, 2009; Williams, 2010).

The second body of work emphasises the impact of trait anger. It is derivative of general trait theory. It emphasises that stable individual differences in emotional arousal exist from the first year of life (Izard, Libero, Putnam & Hayes, 1993). Such theory emphasises and the strong influence of dispositions on information processing, memory storage and retention and the role of schematic representations and high order appraisals in their roles in the interpretation of events.

Applied to anger, the pertinent literature emphasises the contribution of both socialisation and genetics (Schultz, Grodack & Izard, 2010) in noting that anger-proneness is a relatively stable predisposition - measured by reference to the frequency, intensity, and duration of the anger experience (Spielberger, Jacobs, Russell & Crane) - to reacting to stimuli perceived as negative in an angry manner (Spielberger et al., 1983). It notes that individuals with high trait anger experience more quickly, intensely and longer duration and are prone to episodes of rage and fury (Schultz et al., 2010).

3.5.2.2 The social-interactionist perspective. Anger is often explained by social learning models (Novaco, 2007). Originated by Bandura (1973), Social Learning Theory (SLT) proposes that emotion is developed habitually from experience. In a process of vicarious leaning, people (especially children and adolescents) imitate significant others’
behaviour, the expression of anger included. Modelling of angry behaviour is crucial and regulatory systems are thought to operate via contingencies, feedback and cognitions. These contingencies become internalised and habitual. The SLT model is well supported in evidence and links easily to other theories - for example, the approach motivation model of anger discussed in the next subsection.

*Anger Recalibration Theory* (Sell, Tooby & Cosmides, 2009) is a recent specific application of Social Learning Theory to anger. It proposes anger can develop as a habit, partly as a result of efforts to deal with the distress of painful experiences, negative emotions, interpersonal conflict or social pressures. According to it, angry behaviours are more likely to expand where they are followed by some form of immediate gratification or delayed negative consequences.

### 3.5.2.3 Cognitive theories

There are three overarching sets of cognitive theories or models that seek to explain anger. The first emphasises the role of information processing. It proposes anger limits information processing in various ways, leading individuals to revert to processing lessons from past anger-related experiences (Schultz et al., 2010). While anger typically orients the allocation of attentional resources to threatening and angry cues and stimuli, this tends to be greater for those of higher anger. It is driven not so much by specific situation-specific thoughts, but a reversion to existing schemata (Schultz et al., 2010). Even noxious stimuli are subject to information processing (Fernandez & Watson, 2010). To the extent that individuals possess different schemata, information processing is affected differently by the experience of anger.

The second set of cognitive theories of anger is derived from and pertains to appraisal theory. Common to them is the proposition that it is the perception of an event, not the event itself which is the key determinant of affect (Scherer, 2001; Berkowitz & Harmon-Jones, 2004). Most such cognitive models of anger emphasise appraisals are not only necessary, but sufficient causal factors for the experience of anger (Cox & Harrison, 2008).

The role of appraisals have been extensively documented in the genesis and maintenance of anger. They have typically been identified in goal-thwarting, wrong-doing and harm perpetration (see Averill, 1983; Berkowitz, 2010; Berkowitz & Harmon-Jones, 2004). These classic associations significantly underestimate the number of mental contents which may be causally associated with anger. To explain, appraisal theory incorporates primary and secondary appraisal components. The former operates such that, if there is goal relevance, any emotion can result, including anger. If there is goal-outcome incongruence, only negative emotions develop, anger included. Anger occurs if several conditions are met.
First, the event typically requires an external agent and perceived blameworthiness. Second, the event must be meaningful or motivating - anger manifestation being more likely where the focus of attention is significant to the individual. Third, there needs to be intentionality (Berkowitz & Harmon-Jones, 2004, 2010) or significant recklessness (Lemerise & Dodge, 2008).

The third set of models emphasises that there is a motivational element that needs to be appreciated in conceptualising anger. Proceeding from the understanding that emotions involve automated and quasi-involuntary action tendencies, it, as previously observed, emphasises that anger differs from other negative emotions in that it may be and is often associated with an approach motivation (Harmon-Jones et al., 2010). This tendency involves (psychologically and physiologically) engaging with/moving toward the target or source of anger, rather than disengaging/moving away from it. In this sense, it can be either self-initiated and volitional or defensive-protective and reflexive.

### 3.5.2.4 Emotion theory

Emotion theory focuses on the subjective experience of emotions and, in particular, the valence (direction) and strength of experience. It emphasises the evolutionary fiat of biologically-driven, adaptive (survival) programs involving action tendencies in response to eliciting stimuli and, in anger’s particular case, threat, while underscoring a social constructivist understanding of emotion per se (see Gergan, 1985).

There are two principal emotion theories of anger. The first is a Regulatory Deficits Model. It observes that basic anger, like all emotions, is short-lived and its arousal and expression are governed by the interaction of higher-order cognitive perceptual processes and emotional functions (see Schultz et al., 2010). While anger typically orients the allocation of attentional resources to threatening and angry cues and stimuli, this tendency is considered greater for those with higher anger levels and greatest where there is dysfunctional anger. Where this applies, attentional resources are allocated less by specific situation-specific thoughts and more by resort to existing schemata (Schultz et al., 2010).

The second emotion-based model of anger is the primary/secondary/instrumental emotion taxonomy developed by Greenberg and Paivio (1997). It holds that primary emotions are fundamental, direct and initial reactions to events and situations, like fear and shame. Secondary emotions, by definition, are responses to thoughts or feelings rather than the situation; for example, anger in response to hurt, fear or guilt. Pascual-Leone and Paivio (2013) recently reviewed the utility of this model. They noted the crucial distinction between primary states, which are adaptive and accessed for their useful information, and primary states which are maladaptive and need to be transformed. The latter are dysfunctional feelings.
which occur repeatedly and neither change in response to changing circumstance nor provide adaptive directions for solving problems when experienced.

This theory posits that anger can be experienced as preferential to underlying aversive, dysphoric states. Over-control of primary anger for fear of its expression and uncontrolled expression of secondary anger are both potent causes of dysfunction. Addressing each requires different treatment approaches. Primary adaptive emotions need to be accessed for their adaptive information and capacity to organize action, whereas maladaptive emotions need to be accessed and regulated to be transformed. In contrast, secondary emotions need to be reduced by exploring them to access their more primary cognitive or emotional generators (Greenberg & Paivio, 1997).

**3.5.2.5 Integrational models.** There are three major integrational theories that either have relevance to, or have been specifically articulated about, anger. One, Stress Theory, was initially articulated by Selye (1976) in his General Adaptation Syndrome. Stress Theory has been studied across a diverse range - for example, in areas as diverse as coping and anger development in adolescence and criminal behaviour (see Aseltine, Gore & Gordon, 2000). It proposes that an event (stressor) which threatens an individual’s sense of well-being, leads to a three-stage psycho-biological response involving alarm, resistance and exhaustion. Stress need neither be extreme nor trauma-related and can derive from everyday events such as relationships, work and study. Depending on the perceived balance of stress-to-coping resources, individuals will vary in coping effectiveness. Coping may be adaptive or maladaptive.

The second is Berkowitz’s (1990) Neo-Associationist model of anger. Berkowitz asserts that negative affect activates ideas, memories and behavioural reactions associated with anger. Subsequent anger-related thought - involving attributions, appraisals and schematic conceptions - and bodily reactions then interact to further activate this network’s components. While Berkowitz (2010) did not deny the role of appraisals in anger development and maintenance, he emphasised that anger is not only driven by “top-down” cognitive processes, but is also affected by “bottom-up” processes and that there are times when appraisals do not apply. His model stresses that connected feelings, thoughts, memories and motor impulses act in combination in generating the anger response (Berkowitz, 2010; Wranik & Scherer, 2010).

The final integrational model is Novaco’s Contextual Account of anger (1993). He asserts that, to understand anger, clinicians need to appreciate the importance of the context in which the anger occurs. Emphasising the importance of environment, actual threat and
provocations and perceptions thereof, it seeks to move thinking beyond the common representation of anger as due to acute, proximal occurrences. It proposes there are three sets of factors or “determinants” which bear upon the influence of context. The first are situational factors. These include disrespectful treatment, unfairness/injustice, frustration/interruption, annoying traits in others and irritations. The next are distal factors which, he proposes include embeddedness (in issues that can be personal, familial or social), interrelatedness (with other emotions and past experiences) and transformationality (from isolated instances of anger to chronic anger problems and severe acts of aggression). The final set relate to ambient factors which bear upon ongoing environmental circumstances (like urgency, discomfort and impedance). This is an integrational model that is centred on cognitive mediation, but provides for the role behavioural and affective factors.

3.6 Current Status of Theoretical Explanations of Anger

Current prominent explanatory theories of anger emphasise the role of disposition, information and affect processing, appraisals, social-learning processes and the overall interactional effect of cognition, emotion and physiological phenomena on anger. There are also overarching theoretical templates for consolidating the insights of such theories into a more powerful explanatory whole.

These theories and models offer important advances on early theorising and offer a sound basis for understanding and treating dysfunctional anger, such as that found in the context of PTSD. Disposition theories are valuable in their capacity to explain differences in the tendency to externalise or internalise cause in others or self in individuals and across groups and classes (the externalised blaming of men and the internalised blaming behaviour of women being the classical behavioural exemplar of this).

Social Learning Theory propositions emphasising that anger is a learned response modelled by significant others (especially parents) and that this learning, given appropriate contingences, can have a powerful impact across adolescence and adulthood have inherent value. Influenced by both classical and operant conditioning models, such propositions are well supported empirically and have clinical resonance. Its emphasis on contingencies enhances the explanatory value of motivational models of anger, such as approach motivation propositions, and the role of false-positive pre behaviour perceptions of outcomes.

Information processing and appraisal theories likewise explain the distorted perceptions and loss of reality testing which may accompany dysfunctional anger (Berkowitz, 2010; Wranik & Scherer, 2010). They are well supported empirically and offer clear clinical
understandings and paths forward in treatment that can be easily understood and taken up by clients.

Emotion theory, via its regulatory deficits model and primary/secondary/instrumental taxonomy offers a key platform for unravelling the abnormality of anger in their emphasis on dysregulation and avoidance and thereby more effectively treating dysfunctional anger. They also illustrate the strong psychophysiological component of anger that can be reactive and defensively deployed in an out-of-control manner due to the stimulus involved and the fear of dysfunctional anger itself.

These theories have lead to treatments that, like the PTSD theories discussed in chapter two, are reflected in solid client outcomes. These have been consistently described across meta-analyses which endorse both group and individual treatment delivery (DiGiuseppe & Tafrate, 2003). As with PTSD theories, they are, however, neither complete accounts of anger nor without limits theoretically or clinically. For example, disposition theories offer descriptions of tendencies which have strong face validity in explaining dysfunctional anger (e.g., aggression, fear of anger expression and social alienation), but beyond them do not describe the personality factors clinically known to be associated with anger, such as narcissism and rule-bound obsessiality.

Such theories similarly neither coherently detail how the mechanistic devices involved might explain the transition from functional to dysfunctional anger and nor why there are differences in outcome efficacy in the application of well-validated psychological approaches to anger and other negative emotions [see DiGiuseppe, Cannella & Kelter (2007) for a summary of the debate around the applicability of imaginal exposure to anger]. The result is that the although anger treatments are effective - the previously identified metanalyses report grand mean effect sizes of around 0.7 - it remains the case that those treatments remain less-effective when compared to their application to other problem emotions like anxiety and depression (see Norcross & Kobayashi, 1999).

As opposed to the several sets of overlapping guidelines that exist for the treatment of PTSD (see ACPMH, 2013), when faced with a dysfunctionally angry individual in treatment settings, there are no clear guidelines or a consensus view on what represents best practice and what treatments are required for different presentations. Accordingly, continued theoretical development is strongly warranted (Dutton, 2010; Schultz et al., 2010; Van Kleef, 2010).

Particularly pertinent to this research, none of these theories currently account for anger in different populations. A telling example of this relates to why a significant
A proportion of PTSD sufferers experience problematic anger. As emphasised across this research, anger is a highly significant problem in PTSD and explanations of it, and its relationship to PTSD, are keenly sought. Extant anger theories, along with PTSD theories, provide a basis for guiding that theorising, but further work is required.

Foreshadowed in chapter one, and detailed in the latter half of chapter four and across chapters five and six, visual imagery has potential to increase our understanding of anger in PTSD. A fundamental element of cognition, and most-obviously bound to effect information processing, imagery is implicit in many theories and models of anger and PTSD. This has been ignored in anger theories, but is clearly taken up in anger treatments (e.g., Novaco’s imaginal exposure approach to stress inoculation in anger treatment). As cautioned in chapter five, causal explanations cannot be necessarily deduced from outcomes. They are suggestive, however, of the gain that may occur from theory-driven research of visual imagery in anger. The next chapter looks at anger in PTSD and towards its conclusion introduces the potential role of visual imagery.
CHAPTER 4: THE NATURE OF ANGER IN PTSD

If you are distressed by anything external, the pain is not due to the thing itself, but to your estimate of it; and this you have the power to revoke at any moment.

Marcus Aurelius (Meditations)

This chapter describes the nature of anger in PTSD. It builds on the understanding of PTSD and anger developed in the previous two chapters and the explanatory theories described there. Such theories offer a platform for understanding anger in PTSD and significant progress in the conceptualisation and treatment of PTSD and anger has occurred on account of such theories in the contemporary era. Yet these theories are not without limit and the relevant fields have acknowledged the ongoing need for theory enhancement aimed at better-understanding both. This research is predicated on the proposition that anger is a special feature of PTSD deserving increased empirical and theoretical attention. This will now be demonstrated and thereafter the criticality of the relationship of visual imagery to anger in PTSD.

4.1 The Phenomenology of Anger in PTSD

Under the current psychiatric nomenclature, anger exists as a single symptom of the arousal symptom cluster of PTSD. This has been the case from DSM III to DSM 5. Under DSM 5 (2013), the anger-related criterion (criterion E1) of its arousal symptom cluster observes it to be “Irritable behaviour and angry outbursts (with little or no provocation) typically expressed as verbal or physical aggression toward people or objects” (page 272).

There has been limited phenomenological exploration of anger in PTSD. Chemtob and colleagues (1997) in describing a typology of anger regulation deficits in PTSD provided an important early description. Noting cognitive, emotive and behavioural factors involved in anger in PTSD, they identified a “ball of rage” category of individuals with deficits in each of these domains. The only known detailed description of the phenomenology of anger in PTSD, by Forbes, McHugh and Chemtob (2013), albeit one focused on the anger of contemporary combat-veterans with PTSD, observes anger in PTSD to be coloured by a strong, current sense of threat, derived from potent intrusive memories of military/combat-related trauma. Consistent with the earlier, comprehensive review of the literature about anger and PTSD by McHugh and colleagues (2012), it
argued that anger in PTSD, in contradistinction to the existing knowledge, deserves special descriptive, theoretical and investigative attention. What follows in this subsection represents an effort at further articulating the phenomena of anger in PTSD.

### 4.1.1 Characteristics of anger in PTSD derived from PTSD and anger.

Anger in PTSD to some extent reflects its parent disorder. Both typically require that there be a provoking event and their intensity and chronicity are often a reflection of the meaning of that event. Thus, the intention of event perpetrators and moral status of their behaviour(s) are thought to be critical to PTSD (see Chung & Breslau, 2008; Litz et al., 2009; Rosen & Lilienfeld, 2008). This is exemplified in warfare, where morally questionable behaviour can involve perpetrating, witnessing or failing to prevent events that transgress communal and individual psychological or spiritual beliefs, resulting in what has been termed moral injury (Litz et al., 2009). In modern times, this immorality was brought to light during the Vietnam conflict, which was described as an “endless war with no ground gained, only important rage, brutality and a quest of survival” (Stutman & Bliss, 1985). This has been the reality of much military conflict since, as reflected in the experiences of US and Australian soldiers in the International Force for East Timor, Operation Enduring Freedom and Operation Iraqi Freedom (see Forbes, McHugh & Chemtob, 2013).

Beyond war, immorality has long been recognised as a crucial issue in the human response to trauma and, as a causal factor for enduring PTSD. Its impact is also compatible with several of the PTSD theories examined for their utility in Chapter Two; for example, social-cognition-schema and cognitive theories. Similarly, as observed in the previous chapter, anger has long been considered a moral emotion that occurs in response to perceived failure to meet social norms and is concerned with norm enforcement. Either by this route, or through their connection with high-intensity dysphoric emotions of responsibility, like guilt and shame, issues of a moral nature can have a strong connection to dysfunctional anger, and the angriest people can be those who are partner to, or witnessing of morally dubious actions.

In many respects, the experience of anger in the presence of PTSD is that of dysfunctional anger generally. In both, individuals report anger is aversive and dysphoric, dimensional in intensity and provocation-related. There is evidence common psychological processes are involved. First, there is emotion substitution. Provided for in Greenberg and Paivio’s (1997) anger taxonomy (see Chapter Three, page 41), Pascual-Leone (2013) argues that from it’s perspective, anger in PTSD is best construed as a secondary emotion
that masks or deflects PTSD sufferers away from intrusion-activated feelings of fear, to the (pseudo) positivity of angry feelings. This emotion-subjugation by anger in the context of trauma is not limited to anxiety masking. Shakespeare perhaps first articulated this in Macbeth in Malcolm’s advice to Macduff that he best cope with the murderous and traumatic mayhem there such that he “Let grief convert to anger; blunt not the heart, enrage it” (act 4, scene 3). Further is the shared prominence of imagery and, in turn, its linkage to angry rumination.

Another shared feature of PTSD, anger and anger in PTSD is the tendency for key deficits and excesses to reflect dysregulation. As identified in the PTSD and anger literatures respectively reviewed across Chapters Two and Three, these deficits and excesses can become automated and chronic due to out-of-control repetition. This is because, PTSD’s intrusive reliving phenomena can voluntarily and involuntarily interpreted as Warning Signals (see Ehlers, 2010). The prognostic importance of such Warning Signals is not difficult to understand, despite the obvious problem of false positives, and is emphasised in foundational animal and human behaviour theories relating to Signal Theory, like those pertaining to aposematism (Wallace, 1867; Speed & Ruxton, 2005) and Learned Helplessness (Seligman & Groves, 1970; Maier & Seligman, 1976).

Involuntary processes are exemplified in PTSD’s classic unbidden, distressing sensory intrusions and high-provocation dysfunctional anger. As detailed in Chapters Five and Six (see pages 104 and 110), the inability to control imagery leads to high emotional arousal, anger included, and imagery and rumination are inextricably linked to each other in anger in PTSD. Volitional processes in PTSD and anger in PTSD exist as self-initiated rumination, which, as observed in Chapter Six, is where imagery and thought processes become strongly inter-twined.

### 4.1.2 Unique characteristics of anger in PTSD

Anger and anger in PTSD are not identical in form and there are clear, albeit at times subtle, points of differentiation. Anger’s potential for externalising cause is a case-in-point. Anger in PTSD is characteristically focused on those responsible for either the traumatic events or failing to prevent them. It is also preoccupied with those unable or unwilling to understand the nature of the associated loss and hurt and its present and ongoing impact. Associated with this, the personal targets of anger in PTSD relate to specific, identifiable trauma-related groups, rather than a generalised other, as is the case with hostility. These groups range from event perpetrators, through auspicing/employing organisations (e.g., military and police organisations) to community and economic (corporations) and industrial (unions).
entities critiqued by PTSD sufferers as adversaries (like politicians, the news media and worker’s compensation insurers) and often have loosely trauma-defined functional identities.

Importantly, the personal targets to which this anger is directed often remain critically important to the anger-generating individual - for example in personal and marital relationships - and are not construed in derisive or harmful terms, even though there is often palpable significant relationship impact (Healy, Stoeckel & McHugh, 2011). Lamentably, anger and anger in PTSD consequently share the irony that the majority of angry interactions occur in domestic situations. This is long-described in the anger literature (see Averill, 1982, 1983) and supported in recent PTSD research showing anger is one of the most prominent factors in PTSD sufferers seeking treatment (Novaco, 2010), especially where it relates to family (Biddle et al., 2002).

It may also be thematically demarcated from dysfunctional anger. The themes of anger in PTSD are typically focused on the inculcation of values in significant others and the need to protect from the impact(s) of potentially traumatic events. The risks at the core of these themes can be real or symbolic. The status and power of the latter is considerable and well-demonstrated by the potent reminders of past tragedy that young-to-adolescent children may inadvertently represent for military or emergency-service personnel involved in past life-and-death situations. Typically associated with past hurt and loss, such themes can at times be currently and prospectively focused on warning signs for further loss and hurt via event re-occurrence or the connection of present-day issues, cues and triggers to the re-experiencing of past loss. This is consistent with and supported by forementioned Warning Signal Hypothesis. Such warning signs are neither apparent to, nor appreciated by, those without PTSD and the anger of those with PTSD can often relate to others’ failure to recognise or validate their concerns.

The detailed description of the phenomenology of anger in PTSD by Forbes, and colleagues (2013), depicts this. As observed, the authors observe that dysfunctional anger accompanying high symptom PTSD is coloured by a strong, current sense of threat, derived from potent intrusive memories of military/combat-related trauma. This is associated with chronic, intense and prolonged irritable hyperarousal. Contemporaneously, there is peremptory mobilisation of angry and confrontational responses to threat, stress, anxiety and other dysphoric emotions, which results in disproportional responses to everyday events indicative of a coherent networked super-sensitivity to cues and triggers. Along with the mobilisation of these angry responses, there occurs a loss of conscious
awareness of perceptual, emotive and behavioural patterns involving automatic substitution of anger for other emotions. Disagreement and ambiguity are viewed as threatening and trust in others, especially authorities, is diminished generally and particularly in relation to their inability to protect and prevent harm. This results in feelings of being misunderstood and unsupported and an ongoing struggle with issues of self and other’s accountability, responsibility and leadership. This is accompanied by vigilance around values and actions on an everyday basis - for example, orderliness, dependability, punctuality, agreement-keeping and attention to detail. This remains the hitherto most comprehensive description of the phenomenology of PTSD coloured by dysfunctional anger.

4.2 **Historical Conceptualisations of Anger in PTSD**

4.2.1 **Cultural reflection.** General scholarship describing the phenomenology of anger in PTSD has long-acknowledged its importance to the disorder and its associated disability and impairment. From early in recorded history, it was well understood that anger and what is now known as PTSD are intimately related. Evidence of this exists in ancient literary works, such as the dramatisations of Euripides (e.g., Medea), Sophocles (e.g., Antigone) and, especially, Homer and Virgil, whose Achilles and Herakles (Hercules) are the archetypal antiquarian examples of the traumatised warrior with PTSD and rageful anger.

This relationship has been reiterated across time. Important post-antiquarian illustrations include the diverse depictions of the Norse/Celtic story of Beowulf, the murderous rage of Shakespeare’s Hamlet, the behaviour of the Travis Bickle character in the motion picture Taxi Driver and the recent deployment by Shay of the Achilles allegory in his psychiatric and literary treatise on the place of anger in US servicemen with combat-related PTSD, starting with veterans of Vietnam (Achilles in Vietnam; 1995) and then Iraq and Afghanistan (Odysseus in America; 2002). Common to these depictions are intertwined traumatic intrusive visual memories and momentous anger.

4.2.2 **Psychological understandings.** Curiously, despite this lineage, anger in PTSD has been subject to fluctuating interest, diminished recognition, under-representation and neglect in psychological thinking. The construct of anger in PTSD has thusfar not been well developed in psychiatric and psychological thinking about PTSD. Its recognition has consequently followed an undulating trajectory (see figure 4.1) and it is only recently that its central role in PTSD is being re-recognised.
There have been several unexplored modern-era opportunities which, if pursued, might have resulted in an anger-informed view of PTSD. The first occurred in Pierre Janet’s work. In *The Major Symptoms of Hysteria* (1907), he explicitly provided for the role of anger in PTSD. He observed that anger occurs posttraumatically “when the subject is not capable of carrying out well-adapted actions” and that traumatised patients “responded with anger to situations which were trivial for most other people” (Van Der Hart & Horst, 1989; page 7). Despite Janet’s primary focus being on trauma and dissociation, the experience he described is about the avoidance of intolerable trauma-associated emotion and anger, in particular. Interestingly, Janet at times appeared to use anger and hysteria interchangeably and his references to attacks and outbursts may be interpreted as anger writ large. Themes of anger were, however, neither specifically articulated by him nor his contemporaries.

A series of opportunities for recognising the importance of anger to PTSD was then provided by warfare across the 20th century. Despite major military conflicts wherein PTSD-like phenomena were observed from early in the century (e.g., in the horror of WW I’s close-quarter annihilation), it was only toward WW II’s conclusion that observation of US military personnel invigorated interest in what we now term combat-related PTSD and crystallised the centrality of anger reactions to it. Novaco (2007; 2010) provides especially worthwhile summaries of this.

At that time, Grinker and Spiegel (1945) observed high levels of anger were associated with combat-related PTSD. Observing post-mission angry outbursts among airforce personnel, they specified anger and aggression as key elements of the disorder. Sympathetic to this, Kardiner and Spiegel (1947) described the tendency to aggression and violence as one of the most common symptoms of war-related traumatic neuroses. Lindeman's contemporaneous analysis identified furious, interpersonal and socially directed anger as a potent outcome of war. He poignantly described irritability, anger and a loss of warmth in personal relationships as inexplicable and disconcerting to those affected and interpreted by them as signs of approaching insanity (see Novaco & Chemtob, 1998 for a detailed account). Reviewing the psychiatric outcomes of WW II, Kardiner (1959) thereafter comprehensively described the phenomenology of war-caused traumatic neurosis (i.e., combat-related PTSD) by reference to five cardinal features; namely, overall constriction of personality, fixation on the trauma, atypical dream life, persistence of startle response and angry explosive outbursts. These features are reminiscent of the definition of dysfunctional anger offered in Chapter Two.
Nevertheless, recognition of anger’s role posttraumatically faded as part of a
general loss of interest in PTSD itself (see Van der Kolk et al., 1996; Weisaeth, 2014). In
the aftermath of the Vietnam War, US researchers - principal among them Horowitz (1976)
and Figley (1978) - reignited interest in combat-related PTSD among Vietnam veterans.
Even within this burgeoning post-Vietnam interest in PTSD, however, theorising about
anger remained severely limited. For example, the previously described, historically
important Stress Response Theory of PTSD Theory of (Horowitz, 1976; 1979) proposed
there was a knowable course of recovery in PTSD involving oscillating phases of outcry
and then numbing. Although anxious-angry affect is an intrinsic element of the outcry
response-phase and implicit to the subsequent numbing response-phase, anger was not a
central feature of the theory and indicative of this, the imminently developed diagnosis of
PTSD reflected an arousal description, rather than a deeper link to anger in PTSD.

Consistent with this loss of knowledge, classificatory systems over time failed to
afford anger a central place in descriptions of what is now understood as PTSD. At times,
they overlooked it altogether. Thus, neither DSM I’s (APA, 1952) *Gross Stress Reaction*
nor DSM II’s (APA, 1968) *Adjustment Disorder of Adult Life* made any mention of anger.
Even after its incorporation into PTSD’s diagnostic set, largely as a reflection of the
palpable anger of US Vietnam veterans (Novaco & Chemtob, 1998), from DMSM III to
DSM IV, it remained a single criterion of a syndrome classified as an anxiety disorder.

Similarly, although PTSD’s inclusion in DSM III stimulated interest in
conceptualising and investigating it, only a small proportion of research endeavour has, to
date, reviewed the role of anger in PTSD. Thus, two reviews of publications on PTSD
between 1987 and 2001 (Bedard, Greif & Buckley, 2004; Figueira et al., 2007) revealed
that only 189 of the approximately 13,000 articles (i.e., 1.5 %) looked at anger and hostility
in PTSD. Confirmatory searches of the *PsycINFO* and *MEDLINE* databases conducted at
various points during the course of the present research utilising using the terms anger and
PTSD (in its long and short form variants) revealed little change in this anger in PTSD
publication quotient over the last decade (Table 4.1).
Anger-in-PTSD: The Role of Visual Imagery

Chapter 4: The Nature of Anger in PTSD

Figure 4.1 Pivotal Moments in the Recognition of Anger in PTSD

This classificatory and publication absence tends to overshadow a small, but important emerging body of research demonstrating the problem of anger in PTSD. In the general community Power and Fyvie (2013) have argued that less than 50 per cent of individuals seeking treatment from a specialist PTSD treatment service presented with anxiety as the primary emotion and that the majority instead demonstrated primary emotions of sadness, anger or disgust.

Research concerned with combat-related PTSD has noted the particular problems posed by anger in younger combat veterans with PTSD (see, for example, Jakupcak et al., 2007; Morland et al., 2012; Rona et al., 2015; Renshaw & Kiddie, 2012; Shea, Lambert & Reddy, 2013; Worthen et al., 2014). There is concern at increased aggression and interpersonal and social violence (Taft, Vogt, Marshall, Panuzio & Niles, 2007). As a group, they appear to have a disturbingly high risk for domestic violence, aggression and anger, with Reardon and associates (2014) reporting that both they and Vietnam veterans have a well above the community norm rate (24% versus 4%) of Intermittent Explosive Disorder. Meichenbaum (2005) reported a six-fold greater likelihood of them abusing their partners than those without PTSD, with one-third having assaulted their partners in the year prior to enquiry. Evidence suggests such rates of anger are increasing (see Morland et al., 2012; Shin, Rosen, Greenbaum & Jain, 2012) and that PTSD-affected contemporary veterans have the most dysfunctional anger yet encountered (Fontana & Rosenheck, 2008).

Coincidental with, and yet reflective of, this literature, diagnostic thinking has begun to identify PTSD subtypes - for example, dissociative, anhedonic and angry subtypes - as important research targets (see Friedman, 2011; Friedman & Resicke,
2014; Resick & Miller, 2009). Some have even suggested that the hyperarousal subtype of PTSD is the dominant presentation and that it accounts for 70 per cent of cases compared to 30 per cent of the dissociative sub-type (Weston, 2014).

Despite its absolute and comparative infrequency, the momentum of interest reflected in this research interest, if maintained, has potential to lead to new and improved understandings, theories and, ultimately, treatments of anger in PTSD.

4.3 Scope of the Problem of Anger in PTSD

4.3.1 Commonality. Because the phenomenology of anger in PTSD is not yet established in depth, the prevalence of dysfunctional anger in PTSD is difficult to map. Most research of anger in PTSD has to date been conducted on military personnel and combat veterans. Originally about Vietnam veterans, it has consistently showed a strong association between anger and PTSD. Orth and Wieland’s (2006) meta-analytic summary of pertinent research concluded the largest effect sizes (weighted mean effect size $r = .56$) in anger’s association with PTSD existed among Vietnam veterans and that lack of anger control ($r = -.44$), the uncontrolled expression or suppression of angry feelings ($r = .29$), were particularly implicated (Germain et al., 2015). Indicative of this, one of the most comprehensive early studies, the US National Vietnam Veterans Readjustment Study (Kulka et al., 1990), found veterans with a current diagnosis of PTSD had substantially higher scores on anger measures in the preceding 12 months than those without PTSD. Preceding this study, Laufer and colleagues (1985) identified Vietnam veterans with PTSD as angrier than their peers without PTSD, with high correlations between expressions of hostility and the frequency of their PTSD symptoms. Similarly, Boulanger (1986), examining the same dataset, reported those with PTSD were three times as likely to be violent than those without PTSD symptomatology.

Dysfunctional anger has since been reported in veterans of various conflicts, role-types and cultures, including infantry soldiers, support personnel, peacekeepers and peacemakers of Dutch, British, US and Australian military forces (e.g., David et al., 2002; Frueh, Henning, Pellegrin & Chobot, 1997; Hovens et al., 1992; Germain et al., 2015; Johnson et al., 1996; O’Toole et al., 1996; Worthen et al., 2014). As observed, it has been especially noted as problem among contemporary veterans.

Such findings are robust and have been reported across various study types. Early simple investigative studies reported a high prevalence of anger, hostility,
aggression and violence in combat-related PTSD (e.g., Pardeck (1982). Anger management treatment studies at that time noted veterans with recent or remote trauma had persistently higher anger levels (Gerlock, 1994). More ambitious treatment studies subsequently added to this picture. In one such example, Chemtob, Hamada, Roitblat and Muraoka (1994) compared combat exposure matched Vietnam veterans with and without combat-related PTSD and a control group of non-combat veterans with other psychiatric diagnoses. They found those with combat-related PTSD scored substantially higher on multiple measures of anger than either of the contrast groups. Importantly, the three groups did not differ on cognitive or motor impulsivity and these factors, in addition to anxiety, were independent of anger scores. Recent studies, utilising large-scale interview research (Worthen et al., 2014), comparing the efficacy of treatment delivery systems (Mackintosh et al., 2014; Morland, et al., 2014), looking at anger treatment outcome predictors (Morland et al, 2014b) and attempting to understand mechanisms of action in anger management treatment (Mackintosh, Morland, Frueh, Greene & Rosen, 2014) have each illustrated the association of anger and PTSD in contemporary veterans.

Recent research has also shown dysfunctional anger to be problematic across a range of PTSD-affected populations, not just military populations. Anger has thus been identified as a significant PTSD characteristic in: civilian casualties of war (Toren, Wolmer, Weizman, Magal-Vardi & Laor, 2002), victims of crime (Cahill et al., 2003), child and adult sexual assault (Cloitre et al., 2004; Feeny, Zoellner & Foa, 2000; Riggs, Dancu, Greenberg & Foa, 1992; Zoellner, Goodwin & Foa, 2000), survivors of torture and domestic violence (Basoglu, Jaranson, Mollica & Kastrup, 2001; Dunnegan, 1997; Lin, Tazuma & Masuda, 1979), those occupationally at risk for PTSD, like emergency services personnel, train drivers and disaster relief workers (Cothereau, 2004; Evans, Giosan, Patt, Spielman & Difede, 2006; Karlehagen et al., 1993; McDonald, Colotla, Flamer & Karlinsky, 2003; Mearns & Mauch, 1998; Theorell and associates, 1992; Sugimoto, 2001), perpetrators of serious crime (Cauffman, Feldman, Watherman & Steiner, 1998; Rogers, Gray Williams, Kitchiner, 2000) and transport and workplace accidents (Blaszczynski et al., 1998; Chibnall & Duckro, 1994; Ehlers, Mayou & Bryant, 1998; Mayou, Ehlers & Bryant, 2002; Van Minnen et al., 2002).

On the basis of this varied body of research, it is clear dysfunctional anger in PTSD is neither population nor trauma-type specific. In 1994, Lasko and colleagues
provided an apt summary analysis, stating that “increased aggression in war veterans is more appropriately regarded as a property of PTSD, rather than a direct consequence of military combat” (page 373). Twelve years later, in the first meta-analysis undertaken in the area, Orth and Wieland (2006) re-emphasised this, concluding “anger and hostility are substantially related to PTSD among samples who have experienced all possible types of traumatic events, not only in individuals with combat-related PTSD” (page 704). Thus, it is the relationship anger has with PTSD that is critical.

To summarise, dysfunctional anger is a particular problem in PTSD populations and over-represented there compared to dysfunctional anger in the general community. It has a heavy and enduring effect on the trajectory of PTSD, as will next be demonstrated.

4.3.2 The trajectory of anger in PTSD. Stand-alone descriptions of the onset and course of anger in PTSD are difficult to locate in the clinical and research literature. It is possible, nevertheless, to draw inferences based on available information and clinical observation and commonsense about its trajectory.

Logically, the onset of anger in PTSD is trauma-dependent. This is because, in its absence, anger, regardless of intensity, cannot be posttraumatic in origin. In simple terms, anger associated with traumatic events in PTSD must develop posttraumatically. This can be as a primary emotion (see Chapter Three, page 43) or secondary emotion which operates as a mechanistic device for deflecting from primary affects - which, to reiterate, may include anxiety, guilt, disgust, shame and other powerfully experienced primary emotions - and underlying vulnerabilities. Thus a pre-trauma history of anger is not required for the development anger in the context of PTSD and it is possible anger can manifest itself posttraumatically where there was little or no pre-event anger.

From inception, anger in PTSD can be subject to a fluctuating trajectory. This fluctuation is provided for in general theories of emotion which variously emphasise the effect variables such as emotion’s cue-dependent nature (see Clore & Huntsinger, 2009; Izard, 2009), the half-lives applicable to different emotions (Wyer & Srull, 2014) and the effect of priming in the experience of emotion (see Chapter Three, page 48). Similarly, it is also accounted for by research on the psycho-biology of emotion, that emphasises homeostatic mechanisms operate to bring organisms back to balance and away from physiological extremes that cannot be sustained and would
threaten survival in their prolongation (Craig, 2005). Moreover psychometric classificatory systems, stress the diurnal variations which typically apply to emotion (e.g., as per the descriptions of anxiety and mood disorders in DSM 5; APA, 2013). Above all, however, anger in PTSD will fluctuate consistent with reminders, cues (e.g., due to the power of event anniversaries) and triggers associated with traumatic memory and everyday stress.

The trajectory of anger in PTSD is described by PTSD theory and research. The former can be seen in the bi-phasic (outcry and numbing) of Stress Response Theory (Horowitz, 1976; 1979) and the intrusion-avoidance response sequencing of information processing theories (e.g., Creamer et al., 2001) as described in Chapter Two. The latter demonstrates anger in PTSD can be a problem episodically, as seen in the acute anger exacerbations of Intermittent Explosive Disorder or, as it has been euphemistically termed, anger attacks, in PTSD. These have been identified in a variety of populations, including US military personal (Reardon et al., 2014), East Timorese exposed to the atrocities of undeclared-war (Silove et al., 2009) and Cambodian refugees (Hinton, Hsia, Um & Otto, 2003).

Research also shows that anger in PTSD can persist long after the occurrence of a traumatic event (Mueser et al., 2009; Novaco, 2010). This is illustrated by longitudinal research of military populations, sexual assault (Feeny et al., 2000), domestic violence (Chemtob & Carlson, 2004) survivors, political prisoners (Schutzwohl & Maercker, 2000) and refugees (Hinton et al., 2003). Research in such populations similarly illustrates the strong association between anger and enduring PTSD symptoms and that anger causally linked to PTSD symptom persistence. For example, Koenen and colleagues (2003) large-scale study of US Vietnam veterans, indicated that potent anger at perceived negative community homecoming attitudes existed nine years post US involvement and strongly predicted PTSD 14 years later. Similarly, Mayou and colleagues (2002) in a study of road traffic accident survivors reported anger one year post-trauma predicted PTSD two years later, while Andrews, Brewin, Rose and Kirk (2000) again found anger, particularly when directed at others, predicted PTSD in a violent crime survivor population.

### 4.3.3 Risk factors for anger in PTSD

An incisive analysis of the risk factors for anger in PTSD is yet to be struck. In its absence, Table 4.2 highlights probably implicated risk factors. It is drawn from publications which specifically look at anger in PTSD in contemporary veterans (Elbogen et al., 2010; 2011, Morland et
al., 2012; 2014); Rona et al., 2015; Worthen et al., 2014), analysis of anger in PTSD generally (Novaco, 2010) and several articles which emphasise the importance of extreme stressor types, combat exposure, event malevolence and moral wounding in chronic angry PTSD (Litz et al., 2009; Chung & Breslau, 2008; Rosen & Lilienfeld, 2008). Embryonic as it is, this categorisation conforms to the proposition that, just as for PTSD and anger, the risk factors for anger in PTSD are best considered a reflection of the individual’s enduring psychological resources and deficits, his/her developmental life-context and enduring social environment and the nature of precipitating events.

Thus-organising risk factors for anger in PTSD can be justified on several grounds. In the first instance, there is no reason to assume a stress-diathesis paradigm neither applies to nor has explanatory value regarding anger in PTSD. Thus, it is supported by the broad-scale utility of Meehl’s (1962) stress-diathesis paradigm and the wide range of conditions, PTSD included, which can be explained by the interaction of individual vulnerability and external stimuli under given conditions.

Social context and life environment have similarly also been demonstrated to contribute to the risk for anger in PTSD. Under (actual or perceived) conditions of stress is likely to increase the risk for anger. This is predicted by anger-related theories like Anger Calibration Theory (see Chapter Three, page 58). Under conditions of post traumatic stress, this increase in risk of anger in PTSD is stronger again. This is not only underscored by social learning theories, but also PTSD theories which emphasise the impact of maladaptive posttraumatic emotional processing (Foa et al., 1989) and distorted social information processing (Ehlers, 2010).

The warzone and combat-situations are, of course, quintessential stress environments where life and death contingencies motivate highly-charged anger and emotional and information processing can become distorted and predictive of later anger in PTSD (Rona et al., 2015). These are often facilitated by the aforementioned impact of pre-combat military training in mobilising the supposed “strength” of anger to avoid the dysphoric “weakness” of anxiety. This training dehumanises enemy combatants and operationalises the military imperative to negate and eliminate their threat, rendering military personnel more likely to respond to (objective) stress and trauma with anger, thereby precluding or impeding the development of other salient emotions such as anxiety and remorse (see Keane et al., 1985). As observed, the programmatic nature of this training is such that anger in PTSD is difficult to de-
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Operationalise and can become associated with a multitude of seemingly trivial day-to-day occurrences not, which are not directly associated with the originating traumatic experience but are subjectively interpreted as if they were and are associated with extreme levels of distress.

Table 4.2

*Probable Risk Factors for the Development of Anger in PTSD*

<table>
<thead>
<tr>
<th>Factor</th>
<th>Potential Lower Order Predictors</th>
<th>Potential Higher Order Predictors</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre Event</td>
<td>Age (esp. below 40)</td>
<td>High frequency, uncontrolled and vivid imagery</td>
</tr>
<tr>
<td></td>
<td>Childhood trauma/maltreatment</td>
<td>Emotion regulation deficits</td>
</tr>
<tr>
<td></td>
<td>Exposure to anger and aggression</td>
<td>High frequency, uncontrolled and vivid imagery</td>
</tr>
<tr>
<td></td>
<td>Childhood antisocial behaviour</td>
<td>Emotion regulation deficits</td>
</tr>
<tr>
<td>Event</td>
<td>(Heavy) combat exposure</td>
<td>Extreme Stressor types</td>
</tr>
<tr>
<td></td>
<td>Event malevolence</td>
<td>PTSD symptom severity</td>
</tr>
<tr>
<td></td>
<td>PTSD symptom severity of traumatic brain injury</td>
<td>Event malevolence</td>
</tr>
<tr>
<td></td>
<td>PTSD symptom severity of pain conditions</td>
<td>Event malevolence</td>
</tr>
<tr>
<td>Post Event</td>
<td>Financial/social-support circumstances</td>
<td>PTSD symptom severity of traumatic brain injury</td>
</tr>
<tr>
<td></td>
<td>Substance abuse</td>
<td>Traumatic brain injury</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Pain conditions</td>
</tr>
</tbody>
</table>


Like enduring PTSD, anger in PTSD is especially likely to be affected by event type. This is illustrated by Figure 4.2 which shows anger in PTSD will sequentially increase where there is human harm caused recklessly, deliberately and malevolently. There is precedence for this typology; for example, in Terr’s (1991) *Type I* (simple, single event) and *Type II* (complicated, repeated event) trauma distinction. It is also supported in the anger and PTSD literatures where it has been demonstrated interpersonal-events involving culpability and malefeasance typically exacerbate the experience of anger (Lemerise & Dodge, 2008) and PTSD (Litz. et al., 2009). In PTSD, this is especially so where there is malevolence (Chung & Breslau, 2008; Rosen & Lilienfeld, 2008. This is most particularly the case there combat-
related PTSD is present. However, cumulative stress environments of lesser intensity are also inevitably associated with anger in PTSD (Mayhew, 2001). In both military and cumulative stress environments, their impact is likely to be greater where individual vulnerability to anger is high.

<table>
<thead>
<tr>
<th>Cause</th>
<th>Category</th>
<th>Example</th>
</tr>
</thead>
<tbody>
<tr>
<td>Human</td>
<td>Intentional (Acts of Commission)</td>
<td>Terrorism/Atrocities, Warfare, Sabotage/Firesetting</td>
</tr>
<tr>
<td>Accidents/Failures (Acts of Omission)</td>
<td>Mechanical</td>
<td>Human error</td>
</tr>
<tr>
<td>Nature Related</td>
<td>Human-compounded</td>
<td>Failure to maintain, Disease, Famine</td>
</tr>
<tr>
<td>Environment only</td>
<td></td>
<td>Earth, Wind, Fire &amp; Water</td>
</tr>
</tbody>
</table>

*Figure 4.2 A Proposed Typology for Explaining the Association Between Anger in PTSD and Traumatic Event Types*

Additional to the influence of developmental, life-context and enduring social environments and event related characteristics, psychological resources and deficits can create risk for the development of anger in PTSD. Affective and cognitive processes and phenomena are particularly implicated here. Uncontrolled high frequency and cognitive and emotion regulation deficits, particularly those involving responsibility-based attributions - such as guilt-related anger (Kubany, 1998; Kubany & Ralston, 2006) - and aversive emotions related to repulsion, such as disgust (Oktaldeion et al., 2014; Power & Fyvie, 2013), are particularly likely to intensify and prolong anger in PTSD.

Feelings of vulnerability (see Mueser, et al., 2009) and the often-expressed fear of treatment seeking PTSD sufferers that they must not express their anger on account of its potentially highly detrimental consequences to others, also serve to prolong anger in PTSD (Forbes et al., 2002; Healy et al., 2011). The effect of cognition on anger in PTSD is evident from the general tendency to externalise anger.
to others and blame, or at least identify, those who allowed or “caused” the trauma or its aftermath (Mueser et al., 2009; Pitman et al., 1991). It is also apparent in self-directed responsibility attributions, such as guilt-related anger (Kubany, 1998; Kubany & Ralston, 2006), which often occur post-traumatically.

Part of this, and the core interest of this research, information processing in anger-affected PTSD populations is at times heavily and detrimentally influenced by visual imagery. This is supported by a literature emphasising the importance of imagery dyscontrol (e.g., as measured by frequency, vividness and control).

Posttraumatic stress disorder is a problem of memory, albeit not in the manner in which memory deficits are usually construed. This is because it is a disorder of (aversive) memory repetition and retention. Thus, although human memory typically degrades with time and its accumulated distractions, PTSD sufferers report they are afflicted by chronically repetitive distressing memories that are not forgotten.

Although the brain is designed to permit, and facilitate directed memory deletion (Festini, & Reuter-Lorenz, 2013; Golding & MacLeod, 2013), PTSD sufferers do not forget on account of repetitious imagery. This is discussed more in Chapter Six’s articulation of mental imagery processes, where it is emphasised that memory is by its nature a reconstructive exercise that is influenced by cognitive bias. These dysregulated intrusive memories (which are overwhelmingly reported by sufferers as being visual in nature) are a critical mechanistic device that mediates information processing.

While the sub-categorisation of such factors into higher and lower order predictors provided for in Table 4.2 is untested, there is no logical reason why this will not prove to be the case for anger in PTSD. Logically, the greater the number of risk factors present, the greater the likelihood of anger in PTSD. Not all individuals exposed to these risk factors develop dysfunctional anger in PTSD. As with the risk factors for PTSD and anger, no single risk factor alone is likely to result in anger in PTSD. Inevitably, as with PTSD and anger, an appropriately sophisticated stress-diathesis paradigm that takes account of the differential impact of such factors is required to account for the apparent variation. In time it may be possible to develop a valid predictive algorithm of anger in PTSD from its identified risk factors as has recently been shown to be possible with PTSD (Kessler et al., 2014).
4.4 Theories of anger in PTSD

In the attempt to understand anger in PTSD, and the relationship of each to the other, several psychological constructs have been explored. It is important to note, however, that there is no theorising on the matter apparent before the last 20 years and the quantum of research, theoretically-driven or otherwise, is low. The following describes those theories. Given the paucity of research, such descriptions are, necessarily, brief.

4.4.1 Disposition and personality theories. Reflective of general psychological theory of dispositional tendencies (see Chapter Three, page 56) - which, to recall, stressed there are disorders which can be classed as internalising and externalising - Miller, Greif and Smith (2003) sought to establish whether internalising or externalising personality styles affected anger in PTSD. Based on US National Comorbidity Survey data, they demonstrated that externalising style, characterised by high negative emotion and low behavioural constraint, was associated with antisociality and aggression in PTSD (Miller et al., 2003; Miller, Kaloupek, Dillon & Keane, 2004; Miller & Resick, 2007). Forbes, Fletcher, Parslow, Creamer and McHugh (2010) replicated this externalising finding in Australian Vietnam veterans receiving PTSD treatment, having earlier established that externalising style, fear of anger and social alienation predicted poorer recovery following treatment.

The importance of externalisation is consistent with PTSD theory and, in particular, conditioning theory (Keane et al., 1985), as well as the aforementioned outcome of military training, which, as noted, tends to achieve anxiety reduction through a mobilisation of aggressive defences. A propensity for externalisation which, of itself, increases the likelihood of an individual reacting to the trauma with anger, will, consistent with such theorising, be reinforced by the anxiety reducing function of such training.

In light of the enduring nature of anger in PTSD, some researchers have explored the contribution of pre-trauma personality traits in increased dysfunctional anger. For example, Meffert and colleagues (2008), in a large prospective study of US police, confirmed the function of pre-role, trait anger in the development of PTSD symptoms and anger in the first year of active police duty. Consistent with stress-vulnerability models, they found greater trait anger predicted greater PTSD symptoms
at one year and greater PTSD symptoms at one year predicted greater state anger at one year.

Other dispositional/personality-based foci have been researched for their relationship with anger. Novaco has observed the relationship of narcissistic, borderlines and ant-sociality personality disorders and anger (2010), while the connection of anger to paranoid and obsessional personality types and styles is not difficult to make (see Fenigstein & Vanable, 1992; Gudjonsson, Sigurdsson, Brynjólfsdóttir & Hreinsdóttir, 2002; Whiteside & Abramowitz, 2004; 2005).

Directionality dimensions have been identified in general anger research by Fernandez (2008), who advocates for the utility of measuring anger, anger locus, reaction, modality, impulsivity and objective(s) when assessing and treating anger. To date, however, internality/externality of expression and trait-based anger are the only such dimensions explored in research of anger in PTSD.

4.4.2 Cognitive theory. Anger in PTSD has been investigated from a cognitive perspective in a sole study, by Whiting and Bryant (2007). This study investigated the relationship between anger and trauma-related appraisals in a small community sample of trauma-exposed individuals with and without PTSD. It found negative cognitions about the self and world were the strongest correlates of anger. Looking specifically at the role of blaming cognitions in the development of anger in PTSD, they found a strong association between maladaptive appraisals and post-traumatic anger, with catastrophic self and world-appraisals being shown to be a significant predictor of anger.

The role of appraisals in anger in PTSD is supported by the PTSD vulnerability literature. An example of this is Elwood and colleagues (2009) review of cognitive vulnerabilities in PTSD. Although not an empirical study, their comprehensive review identified four such vulnerabilities - rumination, anxiety sensitivity and, most pertinent to the Whiting and Bryant study, negative attributional and looming cognitive styles. Each of these vulnerabilities can exist as stable pre-trauma individual characteristics and, depending on their intensity, act to increase the probability of PTSD-development. The three cognitive appraisals related to trauma identified by Foa, Ehlers, Clark, Tolin and Orsillo (1999) (i.e., negative cognitions about self, negative cognitions about the world and self-blame) are also consistent with this. Ehlers and Clark’s (2000) emphasis on negative appraisals as maintaining factors for symptomatic reactions, similarly supports this view.
4.4.3 **Emotion-focused theories.** Theoretical attention has also been focused on emotion in the attempt to explain anger in PTSD. There are two emotion-focused theories. The first is the Primary-Secondary Emotion Substitution Proposition (see Riggs et al., 1992; Feeny et al., 2000). This model has its antecedents in the idea of anger as a secondary emotion as formulated by Greenberg and Paivio (1997). That theory posits anger can be experienced as preferential to underlying, aversive, dysphoric states. Applied to research of veterans (Feeny et al., 2000) and female crime victims (Riggs et al., 1992), it has been proposed that anger deflects sufferers from intrusion-activated fear to a state less associated with feelings of vulnerability (Paivio & Pascual-Leone, 2010). This is consistent with Foa and Riggs’ (1993) assertion that, in PTSD, anger and dissociation are both processes of disengagement or avoidance of the traumatic memory and fear network. It is also consistent with Forbes and colleagues (2002) research finding that angry veterans with PTSD believe they are misunderstood and maltreated and tend to blame others for their mixed-emotion distress. These processes, are avoidant in nature, and while they may afford temporary relief from anxious distress, inhibit habituation and prevent disconfirmatory or safety-related cues being incorporated into the trauma memory network to modify its associations and interpretations.

The second emotion-focused proposition that attempts to account for the prominence of anger in PTSD is the *Emotional Dysregulation Model of Emotion in PTSD* by Chemtob and colleagues (1997). Initially outlined in the *Cognitive Action Theory of PTSD* (Chemtob et al., 1988), this theory posits that, from an evolutionary perspective, the symptoms of PTSD activate a *Survival Mode* of functioning. Consequent of trauma and PTSD, there is a persistent low-threshold for reactivating *Survival Mode*. This mode is characterised by a narrowing of attention to threat-related cues, hostile appraisal of ambiguous stimuli and mobilisation of pre-emptory threat-related responses including anger and aggression. It acts as an unrecognised, all consuming threat-anger program for aggression (Novaco & Chemtob, 1998). Regulatory deficits, in the form of diminished self-monitoring, hostile appraisal, rapid escalation of arousal and consequent antagonistic or aggressive behaviour maintain the perception of threat, preventing modification of cognitive schema (Novaco & Chemtob, 1998) and sustaining an increased facilitation of reacting to non-survival situations as if they were survival-related. While it is adaptive in combat situations, *Survival Mode* is maladaptive and inappropriate in non-military contexts.
4.4.4 **Integrational/multi-dimensional models.** Each of these theories does not preclude the possibility of disposition-cognition-affect interaction. For example, the *Cognitive Action Theory of PTSD* in some ways could be understood as an appraisal-influenced explanation of anger in PTSD. Only one multidimensional model for explaining anger in PTSD has, however, been tested thus far: Berkowitz’s (1990) Neo-Associationist Memory Networking Model of anger. Described in Chapter Three (see page 60), this model is at its core a learning-based anger theory, but one which holds that aversive stimulation predisposes the individual to negative affect, thereby activating connected feelings, thoughts, memories and motor impulses to the anger response (Wranik & Scherer, 2010). Its application in a study of combat veterans with problematic anger by Taft and colleagues (2007) found negative affect in PTSD is connected through associative networks involving anger-related feelings, thoughts, memories and aggressive inclinations.

4.5 **Status of Current Theories**

The central descriptors emanating from these theoretical models suggest anger in PTSD is the result of an awareness-absent loss of control, pervasive, overarching, distorted and heightened cognitive processes involving angry rumination and, ultimately, a loss of social connection (see Table 4.3). Such theories have thrown much needed light onto the aetiology and nature of anger in PTSD. They also have clear treatment implications. Thus, concepts relating to internalisation/externalisation, cognitive appraisals, affect substitution, survival schema and the networking of anger-instigators and likely responses are each important elements of effective treatment and have strong face-validity for practitioner and client alike (see Forbes, McHugh & Chemtob, 2013).

Nevertheless, they remain little-researched and, as early models of anger in PTSD, appear unlikely in their current form to fully capture why anger is such a problem in PTSD and whether it can be differentiated categorically or dimensionally from other instantiations of anger, like generally dysfunctional anger, other related concepts (e.g., hostility and aggression) and anger co-occurring with other trauma-related mental health disorders (e.g., posttraumatic depression).
### Table 4.3

**Key Characteristics of Anger in PTSD Suggested by Contemporary Theoretical Models**

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Consequence</th>
</tr>
</thead>
</table>
| 1. **A Loss of Volition** | - a level of automaticity, consistent with an absence of control over the response to triggers;  
- diminished self-monitoring  
*As per Chemtob and colleagues (1997 and Novaco & Chemtob (1998)* |
| 2. **Extreme Pervasiveness** | - intensity of feeling that can extend to explosiveness, intense cynicism, hatred or rage;  
- longevity of feeling marked by a smouldering disposition and rumination  
*As per Chemtob and colleagues (1997) and Novaco & Chemtob (1998)* |
| 3. **Overarching Coherence** | - increased memory network linking;  
*As per Chemtob and colleagues, (1997) and Novaco & Chemtob (1998)*  
- tendency for anger to occur secondary to other emotions, especially anxiety, while being superordinate in status  
*As per Berkowitz (1990), Feeny and colleagues (2000), Greenberg & Paivio (1997) and Taft and colleagues, (2007)* |
| 4. **Distorted & Heightened Cognitive Processes** | - loss of the ability to discriminate between significant and insignificant triggers to anger;  
*As per Chemtob and colleagues (1997) and Novaco & Chemtob (1998)*  
- appraisals and causal attributions which go beyond mere externalisation to a ruminative preoccupation with what happened, who allowed it to happen and being wronged or harmed;  
- lack of awareness of a mode of (angry) being that is all consuming  
*As per Berkowitz (1990), Feeny and colleagues (2000) and Taft and colleagues (2007)* |
Their utility is validated by PTSD and anger theories. Prominent PTSD theories emphasise the role of appraisals, information and affect overload, emotion networking and multi-level processes in the condition, while early PTSD theories emphasising the phasic, conditioned and schematic nature of PTSD and together again offer important clinical insights. Theories of anger similarly emphasise the contribution played by disposition, cognitive appraisals, information and affective processing, social-learning/conditioning processes and the overall interactional effect of cognition, emotion and somatovisceral/physiological phenomena.

Doing this does not resolve the observed explanatory gap and the nature of anger’s relationship with PTSD continues to be considered elusive. Evident of this, are the previously noted ongoing and increasing calls for increased theoretical and empirical investigation of anger in PTSD via nuanced-models (see Cahill et al., 2004; Hellmuth et al., 2013; Orth & Weiland, 2006). Such appeals emphasise the importance of building insight into the mechanistic devices which underlie and influence the relationship of anger to PTSD (see Orth & Wieland, 2006).

Important mechanistic targets for review include devices as diverse as traumatic event appraisal - especially around the morality of the trauma-related behaviour of event perpetrators; the contribution of physiological arousal and impact of psychological processes like rumination and anger suppression (Germain et al., 2015). One device, arguably implicit to, if unrecognised in, many and maybe all, existing explanatory models and phenomena and the second of the twin foci of this research, pertains to visual imagery.

### 4.6 Imagery as a Key Mechanism in Anger in PSTD

As frequently observed, imagery, in the form of unbidden, highly distressing, trauma-related autobiographical memories, is the cornerstone of PTSD. The idea that imagery may hold the key to better understanding anger in PTSD and the relationship of the two phenomena and research of it may assist in resolving the limitations identified in current theories is central to this current research.

There is solid, yet to-date unappreciated, evidence to support the position that imagery is intimately associated with anger in PTSD. This evidence is described in Chapter Five. Thereafter, the factors which affect imagery in anger in PTSD are described in Chapter Six.
The remainder of this chapter undertakes three tasks preparatory to those chapters. Initially, it considers why imagery might be associated with anger in PTSD. Flowing from this, it outlines an argument for expanding the hitherto restrictive definition of cognition employed within research into anger in PTSD. Thereafter, it examines how imagery and thought might interact independently, serially, or jointly to influence anger in PTSD. Via the last undertaking, it offers illustrations of how the previously reviewed theories pertinent to anger in PTSD may increase their value by incorporating the role of imagery.

4.6.1 Why imagery might be associated with anger in PTSD. Holmes and Mathews (2010) report on a wide array of imagery-based features capable of explaining anger in PTSD. As detailed in Chapter Five (see page 104) these factors fit within three broad explanatory categories: the direct effect of images on emotional systems in the brain; the tendency for images to have a similar impact to that of real events; and the capacity of images to reactivate past feeling states.

There is also PTSD-related evidence for the association of imagery and anger in PTSD. Exemplifying this is the predominance of PTSD’s arousal symptom cluster as a predictor of overall PTSD severity and its substantial effect on the disorder’s intrusive phenomena (Schell et al., 2004; Shea et al., 2013; Weston, 2014). Amplifying this connection, are further findings indicating the frequency and distress of intrusions are magnified by blaming ruminators (Oktedalen et al., 2014), that ruminative style appears closely linked to PTSD’s re-experiencing symptom cluster (Orth & Wieland, 2006) and that angry rumination has a substantial mediational effect on PTSD (Orth et al., 2008). Of note, ruminating about stressful events can cause autonomic activation similar to that observed in response to the actual incident and may occur and persist long after the event itself has passed (Gerin et al., 2012) and is implicated in coronary heart disease in PTSD via both intrusive imagery (Sack, Cillien & Hopper, 2012) and the rumination classically associated with such illnesses (McClelland, Jones & Gregg, 2009).

Altogether, these explanatory propositions and associated evidence hint at several possibilities. First, there is the likelihood that, while anger has a clear effect on intrusions, this effect is interactive and most-likely reciprocal and neither alone impact as much on the anger-PTSD relationship as they do in combination; for example via the mediating/moderating effect of intrusions.
They also provide for probability that cognitive appraisals will never far from rumination, be they thought or image-based. Evidence exists to support the role of appraisals in maintaining both negative mood states (Szasz, 2009) and intrusive trauma-related memories (Dunmore, Clark & Ehlers, 1999) and the phenomenon of PTSD sufferers angrily going over and over what has intruded and/or ails them is clinically well-known as, previously observed, maintaining of symptomatic reactions.

Alternately, they allow for the possibility that rumination and the form it takes will have a significant singular, direct impact on the anger-PTSD relationship. In this regard, Oktedalen and colleagues (2014) observe that frequency and distress of intrusive memories can be magnified through certain forms of rumination - they cite injustice and blameworthiness. They propose such angry rumination triggers re-experiencing symptoms and increase hyper-arousal and as such, the severity of anger is correlated with the severity of PTSD symptoms. There is also a sense in which rumination may not only be focused on the content of autobiographical memory, but also the repetitiveness of the intrusion process itself. Another possibility, although untested in the research literature, is that, even where the initial affective response to unwanted intrusive imagery is not angry in nature, through a variety of mechanistic psychological devices - including, but not limited to, the aforementioned affect-substitution-effect - it may ultimately lead to angry affect. Thus, as outlined in chapter five, fear of psychological disintegration as represented in the repetitive nature of the (most typically visual) intrusion process may become strongly associated with anger.

In another possibility, when not ruminating, angry PTSD sufferers may also be engaged in, ultimately unsuccessful, efforts at thought (and image) suppression. The effect of the psychological avoidance and suppression of anger is telling and inevitably bound up with the occurrence of rumination and associated imagery and thought. Indicative of this, the capacity of imagery to activate past memories is well described in *Ironic Process Theory/the Zeigarnik Effect* (James & Kendall, 1997; Wegner, 1994; Wenzlaff, 2002). This is the proposition that efforts to suppress mental contents, images included, can paradoxically lead to increased (re)occurrence of that specific content (image). Accordingly, where there is aversive negative-emotion-influenced autobiographical imagery - which is typically experienced with a greater sense of reality (e.g., as measured by vividness), compared to non-emotional or semantic imagery (Holmes & Mathews, 2010) - it often results in unwitting and deliberative attempts to suppress its occurrence, thereby producing counter-intentional
outcomes. As noted in Chapter Three (see page 57), population-based studies have demonstrated the powerful association of anger suppression with cardiovascular disease and chronic heart disease in particular. They show a 57 per cent greater likelihood of chronic heart disease in men who suppress anger and the protective effect of moderately expressed anger (see Williams, 2010).

Finally, they suggest avoidance and numbing - or as they are now known under DSM 5, effortful avoidance of trauma-associated stimuli and negative post-trauma mood alterations - are of least relevance to the relationship of anger and PTSD. This is explained by various factors, among them the lesser relationship they tend to have with imagery.

4.6.2 Rebalancing the notion of cognition in anger in PTSD. There is much that is linguistic in the cognitive processes associated with dysfunctional anger in PTSD. Research of anger in PTSD to date has tended focused on linguistic processes such as cognitive appraisals, cognitive-emotive-physiological-propositional networks and schemata, but paying comparatively scant attention to imagery. Yet not all about anger in PTSD can be explained by a purely lexical model of cognition.

As noted, negative emotional-imagery is typically experienced with a greater sense of reality compared to semantic imagery (Holmes & Mathews, 2010) and importantly, it appears there is a subtle balance at play whereby there is an optimum level of imagery for psychological wellbeing and that an imagery incidence at either extreme of the possible continuum of experience can result in dysfunction. Described in more detail in Chapters Five and Six, imagery avoidance occurs in pathological worry due to excessive (Borkovec & Inz, 1990), while excess imagery is and a preponderance of sensory-imagery encoding of information is associated with persistent and dysfunctional intrusive memories (Bywaters et al., 2004b, Kosslyn, 2005b) and associated with dysfunction (Dadds, Hawes, Schaefer & Vada, 2004).

4.6.3 Relevance of imagery to current theoretical models of anger in PTSD. Rather than create a new, all-embracing explanation for anger in PTSD, this work seeks to optimise the utility of its imagery-based propositions by locating them alongside and building on existing nascent theorising. Consequently, it proceeds from the basis that the emotion of anger and the disorder of PTSD are linked by thought, but also imagery affected on the basis that it is fundamental cognitive device which can inhibit, facilitate or operate independent of thought in that relationship. For example, imagery can influence the impact of anger on PTSD through propositional
memory (via neo-associationist networks), peremptory actions (derived from the automating mental maps of the survivor schema) and emotion substitution (as per the fear-avoidance paradigm). Imagery can be benign (neither positive nor negative) as a mental process. In this sense, imagery is understood as a potentially neutral/content-free mechanistic device that creates its impact via processes related to the aforementioned characteristics like nowness, frequency, absorption, vividness and control.

It argues the benefits of incorporating imagery within existing theories on the basis that an imagery-informed understanding of anger in PTSD may enhance the explanatory value of dispositional models of anger in PTSD. To illustrate, models of PTSD emphasising the association of externalising personality style and anger and aggression (e.g., Forbes et al., 2010) may be able to be refined by examining the noted connection between personality style and anger and imagery (see Chapter Six).

Flowing from this, it provides argument for expanding the hitherto restrictive definition of cognition employed within research into anger in PTSD. That research has focused on linguistic processes such as cognitive appraisals, cognitive-emotive-physiological-propositional networks and paid scant, if any, attention to imagery. Adoption of a theoretical explanation of anger in PTSD which emphasises the critical role of imagery offers many lines of enquiry with the potential to invigorate and crystallise theory and research. As such, it would be consistent with the deeper-level, detailed, localised and practical (“satellite”), theorising advocated in Dalgleish’s (2004) comprehensive analysis of what is required in PTSD research and endorsed by McNally (2004). As it builds on existing descriptions of anger in PTSD, it would also avoid the narrowness-of-focus and alienating-complexity pitfalls Dalgleish warns against.

4.7 Conclusion

Anger is present in a near and possibly substantial majority of PTSD presentations. When dysfunctional, it is burdensome and critically associated with both PTSD chronicity and poor treatment response.

Dispositional, cognitive, affective and somatovisceral/physiological factors independently and jointly affect the development of anger in PTSD. A key characteristic within each factor-domain is that the associated distress and loss-of-functionality that occurs is reflective of disrupted and biased information processing.
Key to this information processing problem is a mechanistic device that arguably contributes significantly to anger in PTSD, but has hitherto been overlooked; that is, the capacity to visually image mental phenomena.

Anger in PTSD is a significant subtype of the disorder deserving of special attention. Advancing a model of anger in PTSD which affords imagery an appropriate place may add to existing and emerging theory and is capable of fitting within an over-arching, multi-representational explanatory model of PTSD. The following two chapters detail evidence that substantiates the role of imagery in anger in PTSD and thereafter the means by which its potency and strength of association with PTSD might vary.
CHAPTER 5: EVIDENCE FOR THE ROLE OF IMAGERY IN THE ASSOCIATION OF ANGER AND PTSD

We are visual creatures. Visual things stay put

Stephen Pinker

The preceding chapters outlined the purposes of this research and described the impact and phenomenology of, and the explanatory theories and models for, PTSD and anger and their relationship. Chapter Four also latterly advanced an argument for applying appropriate focus to visual imagery in anger in PTSD on the basis that it is a mechanistic device that appears causally linked to the disorder’s development and maintenance. Hitherto little-researched in anger in PTSD, the role of imagery can be discerned from sources of evidence pertaining to neuroanatomy, psychopathology, anger and PTSD research. Each will shortly be described, but is necessarily preceded by a definition of visual imagery.

Imagery is known to have a complex structure, involving a potential host of perceptual and memory processes working in concert (Dror & Kosslyn, 1994; Kosslyn et al., 1990). It is manifested across the five human sensory domains via two routes: sensory perception and mental imagery (Pearson, 2007).

Perceptual imagery relates to sensory registration of present stimuli requiring no imagination. Mental imagery is the quasi-perceptual, subjectively-influenced, cognitive representation and recollection of perceptual experience in working memory in the absence of the originating stimulus (Kosslyn, 2005a; Kosslyn, Ganis & Thompson, 2001; Kosslyn et al., 2006). Referred to by a variety of names - for example, visual imagination (Richardson, 2000), pictures in the mind (Libby & Eibach, 2013) and seeing in the mind’s eye (Thomas, 2014) - it is less visually acute than perception, but preserves the perceptible properties of the stimulus and ultimately gives rise to the subjective experience of perception (Kosslyn et al., 2006).

The focus of this research is on mental imagery and, more particularly, visual imagery. There are several justifications for this specific focus. First, although there is overlap between mental and perceptual imagery, they are intrinsically linked and research outcomes in either field are inevitably pertinent to the other (Kosslyn, 2005a), they are dissimilar phenomena and clear points of separation exist between
them. Second, it is mental imagery that is of relevance to the negative affect and stress that are the core foci of this research. Third, the experience of imagery most commonly occurs in the visual domain (Pearson, Deeprose, Wallace-Hadrill, Heyes and Holmes 2013; Kemps & Tiggerman, 2014), an extraordinary example of this being the fact that perceptual cues in alternate sensory domains are often ultimately experienced visually, as recognised in the associative form of the anciently-derived construct of synaesthesia (Cytowic & Eagleman, 2009).

5.1 Neuroanatomical Evidence for the Role of Imagery in Anger in PTSD

5.1.1 Evidence demonstrating the association of imagery and anger.

The connection of imagery to anger can be established by sequentially comparing brain areas associated with anger, imagery and their particular association. The evidence for each is now considered.

There is some evidence that, although overlapping and therefore not wholly distinct, different neural networks are involved in the experience of primary emotions (Lane et al., 1997). The foremost example is that of anxiety and its associated anterior cingulate, orbito-frontal cortex, medial temporal structure, anterior insula and cerebellum activity (Malizia, 2003).

Research has begun to articulate a neurology of anger (Potegal & Stemmler, 2010b). It highlights the prominent involvement of the amygdala, hypothalamus, hippocampus and cortical regions in the brain’s anger response (Dougherty et al., 1999; 2004; Potegal & Stemmler, 2010b; Stein et al., 2007). It also indicates there is activation lateralisation - with relatively greater left cerebral activity (Mitchell & Harrison, 2010; Potegal & Stemmler, 2010b; Shenal & Harrison, 2004) attributable to anger’s offence orientation and approach motivation (Harmon-Jones et al., 2010; Potegal & Stemmler, 2010b).

Research on anger’s neurology further highlights the complexity and functional variability within this system. While it is prudent to emphasise that association should not be taken to construe causation, evidence suggests the amygdala is basic to initial processing of stimuli, while cortical regions are likely to be involved in higher-order (typically response-downscaling) processing and governance. Three studies within the current research literature illustrate this. Furmark and colleagues (2009), in a study of facial recognition in anxiety disorder patients, showed recognition of angry facial expression is specifically associated with the amygdala.
Yet, Vuilleumier (2002), in a functional neuroimaging study, showed that involuntary threat-attention tends to activate the amygdala, while voluntary attention tends to activate the superior temporal and anterior cingulate cortex (ACC). In a further refinement, Dougherty and colleagues (2004) demonstrated anger-eliciting narrative scripts are associated with activation in the left orbitofrontal area and right ACC. Denson, Pederson, Ronquillo and Nandy (2009) expanded on this view. Seeking to review how anger’s cognitive, affective and behavioural components are differentially related to different brain regions, they employed functional Magnetic Resonance Imagery (fMRI) during an anger induction procedure involving insult and instruction to ruminate. They reported activity in the dorsal anterior cingulate cortex was positively related to self-reported feelings of anger and individual differences in general aggression, while activity in the medial prefrontal cortex (PFC) was related to self-reported rumination and individual differences in displaced aggression.

Additional brain areas are likely to be activated following the initial triggering of an anger response. Hence, lesion studies have found that, subsequent to the amygdala’s emotion recognition role, the ventral striatum is involved in signal coding, and general co-ordination of behavioural responses to anger (Calder, Keane, Lawrence & Manes, 2004). Coding and co-ordination can in turn be a prologue to the activation of other brain areas concerned with behavioural ignition. Illustrating this, lesion studies have implicated activation of subcortical areas by the temporal lobe in anger (Iosifesku et al., 2007; Potegal & Stemmler, 2010).

Research also shows anger-related activation patterns can be affected by other factors. For instance, while initial anger appraisal is transmitted to the ventromedial frontal cortex (VMFC) and orbital frontal cortex (OFC), the relationship between the two areas is variable. This is illustrated by Dougherty and colleagues’ (2004) finding of a positive correlation between left amygdala anger activation and the VMFC in individuals with depression and an inverse relationship between such areas in the absence of depression. Again underscoring this complexity, it has been observed the VMFC and OFC act to mediate anger according to possible payoffs and punishments for enactment of anger-related behaviour (Potegal & Stemmler, 2010b).

Consistent with such findings, the literature on the cognitive control of emotions suggests higher cognitive processes are involved in emotion control. The characteristics of such cognitive processes have been elegantly reviewed by Ochsner and Gross (2005). They note that emotion regulation studies have examined either
controlling attention, or cognitively changing the meaning of, emotionally evocative stimuli. They assert that these two forms of emotion regulation depend upon interactions between prefrontal and cingulate control systems and cortical and subcortical emotion-generation systems. They assert there is a fundamental brain architecture involving the PFC, OFC and ACC in specific control processes (e.g., top down processes generated by stimulus appraisals like those involved in aversive interpretations of otherwise innocuous images) and activation in subcortical regions, such as the limbic system and amygdala, in emotional appraisal.

5.1.2 The role of specific brain areas in visual imagery. A large body of evidence has implicated specific brain areas in the manifestation and management of visual imagery. Two areas have been identified. The first comprises the cortical areas associated with the visual system itself. Known as the visual cortex, this consists of the topographically organised primary visual or striate cortex and extrastriate cortical areas (i.e., the Brodman areas 17-19) (Kosslyn et al., 1993) and is responsible for visual perception.

The visual cortex is, however, not directly involved in the production of visual imagery. Rather imagery production secondarily involves a network of spatial subsystems and higher visual areas (Knauff, Kassubek, Mulack & Greenlee, 2000) which are a subset of those involved in visual perception (Ganis, Thompson & Kosslyn, 2004). Thus, imagery cannot occur without prior perception, but does not engage all areas of the visual cortex and rather relies on recall of perceptual information from long-term memory storage when it occurs (Kosslyn, 2005a). Two different areas within the visual cortex are involved in visual imagery - the occipital lobe being associated with the formation of perception and the right hemisphere, near the parietal lobe, being associated with the spatial characteristics of such perceptions (as in the construction of mental maps and object manipulation) (Kosslyn, 2005a).

Abundant, long-standing evidence from brain research via Positron Emission Tomography and fMRI shows the same areas of the brain used for perception are activated by mental imagery (see Miyashita, 1995; Kosslyn, 1996). Ganis and colleagues (2004) further observe this overlap is neither complete nor uniform, being more pronounced in frontal and parietal regions than the temporal and occipital regions. Importantly, they indicate cognitive control processes function comparably in both imagery and perception, but that some sensory processes may be engaged differently in perception and imagery.
Researchers have also long-emphasised the role of a second brain area in the manifestation and management of imagery; that is, the limbic system and, in particular, the amygdala. The latter, and especially the peri-amygdaloid cortex, is activated by imagery-based recollections of trauma (Bystritsky et al., 2001; Shin et al., 2004; Shin et al., 2005). For instance, in a series of emotion recognition tasks, the amygdala has been shown to have a specialised role in recognition of emotional facial expression (e.g., Gobbini & Haxby, 2006). This role has been noted as automatic in quality (Vuilleumier & Sande, 2008) and established for a range of expression(s) - including angry, happy, fearful and surprised faces (Furmark et al., 2009; de Jong, Koster, van Wees & Martens, 2009; Kim et al., 2004; Mogg, Garner & Bradley, 2007).

Brain activation in imagery varies according to the characteristics of the events/tasks concerned (Holmes & Mathews, 2010). To illustrate, in a series of PTSD studies, a range of cortical structures - including the precuneus, superior lingual gyrus, insula, inferior temporal gyrus and fusiform gyrus - were shown to exhibit decreases in activity during script-driven imagery recollections of personally traumatic events (Shin et al., 2004; Shin et al., 2005). The same PTSD research showed activation of the inferior frontal cortex, and in particular the inferotemporal cortex, occurred in situations involving direct exposure to fearful stimuli. Investigation of panic disorder patients, via exposure to fearful stimuli, has shown the same brain circuitry changes and it has been suggested that executive control of imagery occurs via the prefrontal cortex during fearful tasks requiring imaginal representation of objects (Bystritsky et al., 2001). In contrast, grief research comparing the reactions of bereaved women to photographs of the deceased versus strangers showed the cuneus, superior lingual gyrus, insula, dorsal anterior cingulate cortex, inferior temporal gyrus and fusiform gyrus to be stimulated. Interestingly, the same study showed that words associated with the death event activated the precuneus, precentral gyrus, midbrain and vermis (Gundel, O’Connor, Littrell, Fort & Lane, 2003).

Contrasting such bodies of research, indicates the brain areas associated with imagery and anger share considerable overlap. If this were not the case, and the areas, networks and circuits involved in imagery and anger were separate, it could not be argued there is a mechanistic link joining the two. These areas are not isomorphic in operation, and there are obvious dissimilarities. For example, the cortical areas (the occipital and parietal lobes) associated with imagery formation and spatial awareness...
are not the brain areas associated with the experience of anger (or other negative affects).

To paraphrase that above, brain areas typically activated in the experience and processing of mental imagery include: the limbic system and, in particular, the amygdala. The latter and the periamygdaloid cortex are activated by imagery-based recollections of events, while imagery production is mediated by a range of cortical structures - including the precuneus, superior lingual gyrus, insula, inferior temporal gyrus and fusiform gyrus. Where anger is manifested, brain areas typically activated include the amygdala, hypothalamus, hippocampus and elements of the cortical regions. The amygdala processes initial stimuli. Thereafter the ventral striatum codes signals and general co-ordination of behavioural responses to anger, via activation by the temporal lobe. Initial anger appraisal is transmitted to the ACC, VMFC and OFC, and cortical regions are likely to be involved in sensory and behavioural governance with left cerebral activity being involved in response-activation.

Accordingly, the strongest evidence of overlap and possible shared function appears to relate to the limbic system and, in particular, the role of the amygdala. This is particularly pronounced where human faces are implicit to the experience of imagery and/or anger. There is also evidence for the mutual involvement of the paralimbic and periamygdaloid areas, depending on the imagery-trigger type. Co-ordination in these areas has functional value - for example, in the detection and ongoing monitoring of threat. Co-involvement of such brain structures in visual processing of anger in a multi-site-response is likely to prime the anger response in PTSD and thereby underlie the peremptory nature of anger in PTSD.

There are caveats applicable to this necessarily brief review which require explication. First, available scholarship neither claims nor suggests every brain area and circuit involved in the experience of anger is involved in imagery production and vice versa. Notably, the ventral striatum and subcortical regions and the lateral orbitofrontal cortex are not implicated in the development of imagery. Nor does there appear to be any left hemisphere lateralisation involved. In the opposite direction, the operation of the periamygdaloid areas in imagery does not appear central to the experience of anger. Thus, there are bound to be brain areas and circuits of differentiation.

Second, the precise overlap of imagery and anger-implicated brain regions is currently not well-mapped. Anger and anxiety have different (fight and flight) core
themes, which are reflected in differences in neural circuitry (Potegal & Qiu, 2010; Potegal & Stemmler, 2010b). Yet they share circuitry commonalities (Lanius, Frewen, Vermetten & Yehuda, 2010). Anger’s circuitry is, as observed, less well-known and in need of further investigation (Denson, Pedersen, Ronquillo & Nandy, 2009; Denson, 2013). Investigation of the neurology of anger using various imagery-based paradigms will clarify their relationship and how it may differ from other imagery-emotion brain relationships.

Notwithstanding such cautions, the existence of an anger-imagery brain relationship overlap can be justified on various grounds. To illustrate, in a summary of the neuropsychological evidence surrounding imagery and facial expression, Vuilleumier (2002) asserted that the automaticity associated with imagery enables near instantaneous detailed processing of sensory inputs from the environment. This biases attention toward survival-salient stimuli, thereby involving the peremptory connection of imagery to basic negative emotions, like fear and anger. This is supported by Holmes and Mathews’ (2010) comprehensive review of the clinical implications of imagery on emotion. They argued that imagery for emotion precedes language’s development as an early-stage evolutionary response to danger. It is also consistent with the summation of the brain-body-behaviour connection of imagery to anger offered by Mayne and Ambrose (1999) in their selective review of research evidence on the psychological treatment(s) of anger. Drawing on the work of contemporary researchers - such as Berkowitz (1990) and Novaco (1976) - they argue such neural networks are likely to act as part of a complex brain-body interaction. In this interface, anger emerges from: (a) an instinctive assessment of the environment in brain areas programmed for rapid processing of visual information, (b) a match between signals detected and brain networks designed to act upon emotion and, consequently, (c) interaction of physiological processes, emotions and thoughts to modify feelings of anger.

In summary, neural pathways which link the processing of imagery with activation of defensive emotions, like fear and anger, are evolutionarily adaptive in that they enable rapid mobilisation of responses to potential threats, human or otherwise (LeDoux, 1999). When out-of-balance, or as some have proposed, there is functional disconnection or miscommunication (Krans, 2011), this system can become maladaptive in its response-activation to neutral stimuli or reminders. Some
of the ways in which dysfunction can occur are considered in this chapter’s next three-sub sections.

5.2 Psychopathology-related Evidence for the Role of Imagery in Anger in PTSD

Support for the proposition that imagery is involved in anger in PTSD can be derived from evidence gathered in research of psychopathology. That research demonstrates the heavy involvement of intrusive, distressing and repetitive imagery across a range of psychopathologies. That imagery is integral to human perceptual and intellectual processes and psychological functioning is long established and well understood (Singer, 2006). From mid-way through the latter half of the last century, and increasingly so in this one, researchers have noted the critical association of high-preponderance and out-of-control imagery with a plethora of psychiatric conditions and psychological phenomena (see Hackman & Holmes, 2004; Holmes, Brewin & Hennessy, 2004; Holmes & Mathews, 2005; 2010; Kosslyn, 2005b; Pearson et al., 2013). Reflective of this, four psychological journal special issues have reviewed various aspects of mental imagery over the last decade. They are Memory in 2004 - which focused on mental imagery and memory in psychopathology; The Journal of Behaviour Therapy and Experimental Psychiatry in 2007 - which considered imagery in psychopathology and the utility of Imagery Rescripting and Reprocessing Therapy (IRRT) (see page 111); The International Journal of Cognitive Therapy in 2011 - which explored the role of intrusive visual memories in psychopathology; and, The Journal of Experimental Psychopathology in 2012 - which explored the wider context of imagery and psychopathology research, drawing on the research programs of the special editions contributing authors.

The clearest connection of imagery to negative, disordered emotion has been demonstrated in the anxiety disorders, where research has shown it is closely related to specific phobia and social anxiety (Cuthbert et al., 2003). Examples of this include the imagery of feared stimuli central to snake, spider and vomiting phobias (de Jong, Peters & Vanderhallen, 2002; Pratt, Cooper & Hackman, 2004; Price, Veale & Brewin, 2012) and the negative-self-imagery so often central to social anxiety (Clark & Wells, 1995). It is also closely linked to PTSD, such that their relationship is considered axiomatic and, as detailed in the following subsection, imagery lies at the
core of PTSD’s symptomatology, severity-moderation and psycho-physiological activation.

Why imagery is associated with, and has such an effect on negative emotion and psychopathology, is yet to be determined. Many factors have been proposed. As noted, in Chapter Four (see page 91), Holmes and Mathews (2010) argue in favour of three broad explanatory categories emphasising: the direct effect of images on emotional systems in the brain, the tendency for images to have a similar impact to that of real events and the capacity of images to re activate past feeling states. The applicable literature cited by them merits succinct description.

The direct effect of imagery on emotion systems is evident in research showing that negative imagery produces affective and somatic activation (e.g., Nelson & Harvey, 2003). In essence, imagery stimulates, and is stimulated by, associated emotional and physiological arousal. Reactions to emotion-imagery are similar to the actual experience of that emotion and in a reciprocal manner mind-body feedback loops involving arousal further intensify imagery and consolidating its connection to negative affect (Holmes & Mathews, 2010; Lang et al., 1977). Although autobiographical-imagery does not have movie-reel replay quality (Lilienfeld, Lynn, Ruscio & Beyerstein, 2011; McNally, 2001; 2004), individuals perceive imagery as strongly representative of external realities (Singer, 2006) based on long term memory storage of individually salient but non-present perceptual stimuli (Kosslyn, Thompson & Ganis, 2006).

Interestingly, it appears there is an optimum level of imagery for psychological wellbeing. The effect of less-than-optimal imagery levels is described in Borkovec’s well-known observation that imagery avoidance occurs in pathological worry on account of the interfering effect of worry associated with thought (Borkovec & Inz, 1990). The effect of more-than-optimal imagery is evident from the observations that excess imagery can become associated with dysfunction (Dadds, Hawes, Schaefer & Vada, 2004) and a preponderance of sensory-imagery encoding of information is associated with persistent and dysfunctional intrusive memories (Bywaters, Andrade & Turpin, 2004a).

The similar impact of imagery compared to actual experience is well established. It is understood that images can be perceived as real (Singer, 2006) and even “realer than real” (Richardson, 2000) and that imaging an act engages the same motor and sensory programs involved in actually carrying it out (Holmes & Mathews,
The ability to form vivid images is associated with enhanced aversive learning (Bywaters et al., 2004a; Dadds, Bovbjerg, Redd & Cutmore, 1997) and is well-demonstrated in research on specific and social anxiety disorders and PTSD (Cuthbert et al., 2003; McTeague et al., 2010).

The impact of this similar-to-reality effect is also shown in imaginal representations of reality. This is exemplified in the fixated and illusory thinking of Obsessive Compulsive Disorder and the hallucinatory and delusional mental content(s) of psychosis (DSM 5; 2013). Perhaps the most pertinent example, however, occurs in PTSD, where repetition of intrusive affect-laden imagery enhances an individual’s capacity to experience vivid imagery (Bryant & Harvey, 1995).

Beyond these explanatory propositions, the effect of imagery on psychopathology can also reflect the operation of dysfunctional, unconstrained goals discrepant with reality (Conway, Meares & Standart, 2004). This gap-effect has been demonstrated in McNally’s work on the mechanisms of traumatic memory in PTSD (2003). He emphasised the role of priming processes - that is, the encoding of information in memory and subsequent increased capacity to recall this or related information from cuing, intended or otherwise - as important to not only recall, but also the subjective veracity with which such recall occurs, even if mistakenly so.

Finally, as observed in the previous chapter (see pages 92-93), activation of past memories is ironically increased by efforts at image suppression. This is well-provided for in Ironic Process Theory/the Zeigarnik Effect (James & Kendall, 1997; Wegner, 1994; Wenzlaff, 2002) and negative-emotion-influenced imagery, with its great sense of reality, produces counter-intentional outcomes.

In summary, imagery processes and affect-valence appear to act together to increase distress, through a variety of mechanisms, and in the process consolidate their connection to each other. Consequently, the tendency to experience repetitive mental imagery is associated with high levels of negative emotion and an inverse relationship exists between high levels of poorly managed imagery and mental health. Evidence of the existence of image-driven anger in PTSD is now considered.

5.3 Anger-Related Evidence for the Role of Imagery in Anger in PTSD

Imagery’s effect on anger in PTSD can be inferred from a third line of evidence that relates to the effect of imagery on anger and vice versa. Dysfunctional anger has been noted to involve fantasies of harm, retaliation, rumination and imagery
(Thoresen, Harris, Luskin & McCullough, 2000) and this impact is demonstrable at various levels. Initially, imagery has been shown to have the capacity to generate physiological responses indicative of angry mood. This is evident in imagery studies utilising a range of physiological markers, such as heart rate, skin conductance, corrugator and zygomatic facial muscle, bodily temperature perception and blood pressure change (Stemmler, 2010). Investigation of the effect of actual, imaged and recollected instances of anger has found imaged anger can actually generate a greater response (as measured by diastolic blood pressure) than that derived from recollection of actual anger-related events (Foster, Smith & Webster, 1999).

Importantly, while imagery incorporating negative emotion results in strong physiological responses (Bywaters et al., 2004b; Miller, Patrick & Levenston, 2002; Witvliet & Vraa, 1995), it is imagery with angry content that has the most profound effect on physiological responses (Schwartz, Weinberger & Singer, 1981; Sinha, Lovallo & Parsons, 1992). Thus, research has shown imagery elicits angry mood (e.g., Greenberg & Paivio, 1997) and physiological reactivity (Stemmler, 2010), has a particularly powerful effect on physiological responses when angry content is present (Schwartz et al., 1981; Sinha et al., 1992) and, when angry, can generate a greater response (as measured by diastolic blood pressure) than that derived from recollection of actual anger-related events (Foster et al., 1999).

Imagery’s effect on anger is also evident in the treatment of problem anger. First, imagery has routinely been used to elicit past experiences of anger, which then become the target of treatment via a self-instruction training (SIT) approach to treatment of dysfunctional anger (e.g., Novaco, 1997). Induction of anger in this manner indicates that (disordered) imagery has the capacity to stimulate dysfunctional anger. Second, imagery has been utilised in the treatment of dysfunctional anger across a range of populations (e.g., Chemtob, Novaco, Hamada & Gross, 1997; Taylor, Novaco, Gillmer, Robertson & Thorne, 2005; Taylor, Novaco, Gillmer & Thorne, 2002) and treatment approaches - for example, via cognitive behavioural therapy (see Deffenbacher, 2011), emotion focused-therapy (see Greenberg & Paivio, 1999) and SIT.

A change mechanism, of course, does not necessarily reflect aetiology, and causal explanations cannot be deduced from any association between treatment outcome and imagery’s use in treatment. Logically, however, if dysregulated imagery were not intrinsic to maintenance of dysfunctional anger, there would be little
requirement for its use in remedial procedures. Recalling McNally’s (2003) work on PTSD memory mechanisms, it is pertinent to here observe the noted tendency for imagery repetition to enhance imagery capacity.

Sympathetic to this evidence, research has also highlighted the significant impact of imagery on emotion (Beck & Emery, 1985; Holmes & Mathews, 2010; Martin & Williams, 1990). Imaginal processing is considered to be more emotionally coloured than verbal information processing (Holmes & Mathews, 2005). It is also reported to influence the ability to experience emotion (Suler, 1985), have an enduring, significant impact on the expression of emotion and be associated with greater-at-rest affect. Large population sample research has also shown imagery can imbue emotions with intensity consistent with the actual objects of emotion (Kunzendorf, Hartmann, Thomas & Berensen, 1999).

Research has also revealed anger’s impact on imagery. For instance, it has been shown in research of type-A personality individuals that high or increasing levels of anger lead to greater responsivity to imagery (Janisse, Edguer & Dyck, 1986). Similarly, a study of state-trait anger in undergraduate university students showed those with high trait anger had greater reactivity to angry imagery in the absence of enhanced imagery ability (Slomine & Green, 1993). This and the previously cited study by Bywaters and colleagues (2004a), which showed strongly-valenced, high-emotion-arousing tasks (measured via the International Affective Picture System) are associated with increased vivid imagery, suggests angry distress and imagery share an association beyond imagery capacity.

There are also logical possibilities which point to the association of imagery and anger. As often-noted elsewhere, anger more often than not has an externally-directed focus (Spielberger et al., 1995; Wranik & Scherer, 2010). Its external focus directly involves others and their behaviours and instances where this is not so are relatively rare. Interestingly, evidence from studies of visual imaging suggests it is easier to produce imaginal constructs when instructions are given to include others, particularly significant others (e.g., parental figures) (Bent & Wick, 2006; McKelvie, 1994). This inclusion of others further facilitates a reciprocal, image-emotion experience that increases the incidence of imagery. Added to this, the experience of anger, perhaps more than any other emotion, can follow from a multiplicity of possible causes and attributions. As noted, these include causes related to actual or perceived injustice(s), personally being wronged, ignored, disrespected or devalued;
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and threat(s) to oneself, a dependent or significant other. Those suffering from
significant dysfunctional anger often describe multiple reasons for their anger at the
hands of others, each successive angry component operating as unique, compounding
stimulators of imagery’s incidence and impact, thereby strengthening the relationship
of imagery to angry mood. [See Novaco (2007; 2010) or Spielberger et al., (1995) for
a catalogue of anger’s myriad mental contents.]

The characteristics by which imagery may be measured also provide grounds
for asserting the association of imagery to anger. These characteristics include
frequency, nowness, controllability, absorption and vividness (see Hackman, Ehlers,
Speckens & Clark, 2004; Michael, Ehlers, Halligan & Clark, 2005). As noted, these
imagery qualities directly affect emotional systems and reactivate past feeling states
in a manner similar to real events (Holmes & Mathews, 2010).

Detailed in the following subsection on the association of imagery and anger
in PTSD, the effects of imagery prevalence illustrate that frequent, unbidden imagery
can be associated with increased anger on a process basis. Overall, although more
research is required to establish the specific, casual or otherwise, links between
imagery and anger, this link is plausibly deducible, and consistent with the reported
experience of PTSD sufferers.

5.4 Evidence of the Role of Imagery in anger in PTSD

While anger has been shown to be important to PTSD, visual intrusion is
critical to PTSD. Intrusions are described as a core symptom, and a key severity and
physiological-response mediator. When uncontrolled, it is defining of PTSD
(Horowitz, 1983; 2001; Laor et al., 1998) and the strength of the relationship is
considered unique to PTSD, being neither common to other anxiety disorders
(Cuthbert et al., 2003; Olatunji et al., 2010) nor trauma-related mental health disorders
(Brewin & Holmes, 2003).

Investigations and reviews emphasise repetitive, traumatic imagery is a well-
established cause of post-traumatic distress, anger included (Orth & Wieland, 2006;
Schutzwolf & Maercker, 2000). They also stress that affective arousal and
sympathetic nervous system reactivity is a primary driver of PTSD’s intrusive
imagery (Orth & Wieland, 2006; Witvliet, 1997). Thus, the intrusive imagery of
PTSD is both a cause and consequence of posttraumatic anger.
While treatment effects cannot be used to determine causation, the connection of the two phenomena in anger in PTSD can also be discerned from treatment outcome research showing imaginal exposure for PTSD reduces not only intrusions, but also post-traumatic anger (Cahill, Rauch, Hembree & Foa, 2003). Although rare, there have been direct attempts to treat anger in PTSD using imagery. For instance, there is the body of work pertaining to IRRT (see Smucker & Moos, 2005). As a therapeutic technique, IRRT aims to identify, confront and modify (typically visual) “hot” cognitions by manipulating recollections via imagery. It has been described as having successfully reduced symptoms in individuals with problematic PTSD presentations which have not responded to other PTSD treatments and as having better effects on non-fear based problems like anger (Arntz, Tiesema & Kindt, 2007; Grunert, Weis, Smucker & Christianson, 2007).

The precise reasons why the two phenomena may be more strongly linked in the presence of PTSD are yet to be established. However, their link is at least partly a reflection of the effect of the meanings ascribed to intrusions. A potentially powerful determinant of angry distress, it is well established from research on PTSD and other psychopathologies - such as, depression (see Starr & Moulds, 2006) - that where intrusions occur, (negative) meaning(s) given to them typically increase negative mood. Repetitive intrusions thus come to represent a threatening loss of mental control and significant, angry, distress is likely to occur. Horowitz recognised this many years ago in his imagery-based theory of PTSD (1976; 1983; 2001). Essentially an information-processing model of PTSD, his theory asserts image control failure lies at the root of severe posttraumatic symptomatology. It recognises that, although individuals can have a strong desire to resolve differences between pre and post-trauma views of the self, others and the world, this processing task can often be so psychologically painful that it is serially interrupted outside conscious awareness. Over time, this not only results in the oscillation between intrusion and avoidance he identified, but also anger.

The little PTSD research specifically focused on imagery and anger amplifies the importance of control. A study by Laor, Wolmer, Wiener, Weizman and colleagues (1999) suggested image control influences anger presence, such that those with high control of imagery have greater anger control and fewer intrusions compared to PTSD sufferers with low imagery control. In a review of the relationship of reflective thinking and mental imagery and their relationship to PTSD, Kosslyn
(2005b) reached similar conclusions, proposing loss of image control in the presence of emotionally charged high-stress (especially traumatic) events leads to high arousal, thereby creating the potential for imagery to become stuck (i.e., in recursive loops).

The salience of imagery’s content matter provides a second sense in which meanings associated with visual intrusions can have an effect on anger. Traumatic memory content inevitably concerns what happened in the pathogenic event and may be influenced by various factors. Take the case of trauma-type. As previously noted in Chapter Two (see table 2.3), there is clear evidence interpersonal trauma results in more severe PTSD than non-interpersonal trauma, especially where there is culpability and malevolence (Chung & Breslau, 2008; Rosen & Lilienfeld, 2008). As noted in Chapter Four (see figure 4.2), it can also be reasonably predicted that human harm caused recklessly, deliberately or malevolently is in order increasingly likely to be associated with anger. This is highly likely to be the case in externally-directed manifestations of anger. It is also consistent with the dose-response explanation of PTSD (see Schwarz, Kowalski & McNally, 1993; McNally, 2004), and with the general tendency for anger to externalise focus on others and blame, or at least identify, those who allowed or “caused” the trauma or its aftermath (Mueser et al., 2009; Pitman et al., 1991). It can also be evident in self-directed responsibility attributions, such as guilt-related anger (Kubany, 1998; Kubany & Ralston, 2006) that often occur post-traumatically and which, as previously noted, along with disgust and shame, may occupy up to 50per cent of PTSD’s affective experience (Power & Fyvie, 2013).

There is evidence another trauma-related content which may affect anger in PTSD relates to the autobiographical nature of intrusions in PTSD (DSM 5; APA, 2013). Consistent with Holmes and Mathews’ (2010) emphasis on the capacity of images to reactivate past feeling states, irritable aversion is highly probable where such imagery relates to (accurate or erroneous) causal attributions concerning the self or another’s actions pre, peri or post-traumatically, especially where misfortune and harm apply. This is illustrated posttraumatically, where traumatised individuals express angry distress at the misplaced (in)actions of others in the recovery effort, mass-scale post-disaster and emergency situations being a primary example (Jayasinghe, Giosan, Evans, Spielman & Difede, 2008).

Although this intrusion-derived distress can initially or in part be anxious in nature - and to reemphasise, PTSD was until DSM 5 considered an anxiety disorder -
anger is likely to follow and may be only one-step-removed emotionally. This can be argued on several grounds. First, it is consistent with the aforementioned cognitive theory-derived survivor-mode, emotional avoidance and neo-associationist accounts of anger in PTSD. Second, it is predicted by the Warning Signal Model of PTSD’s intrusive memories (Ehlers et al., 2002). This model holds that intrusive memories, through their connection with stimuli in place at the time of the trauma, function as warnings of the potential re-occurrence of the trauma, where those same stimuli are again encountered. Faced with warning-intrusions, survival cognitions ontologically become imperative and the universally observed fight or flight response is invoked - anger and anxiety, being the two well-known emotional endpoints of this chain-of-events. Third, to recall anger, anxiety and depression share status as the big three negative affects (Frisch, 2006). As high-prevalence, dysphoric affects they demonstrably overlap in content and are often clinically comorbid and interdependent. Finally, anger has a role as an energising, secondary or cloaking emotion for dysphoria generally - this latter function being especially likely in the context of PTSD (Feeny et al., 2000). The effect of this is that, even if the initial emotional experience in response to unwanted imagery is not characterised by anger, imagery can indirectly result in angry affect. As observed in Chapter Four (see page 89), this can relate to either or both the content and process-mechanisms of imagery.

The relationship between anger and intrusions in PTSD (Orth & Wieland, 2006), whereby physiological arousal associated with anger leads to intrusions and, in turn, is reciprocally potentiated by such intrusions, has two ironic outcomes, which again strengthen this relationship. The first is that intrusion repetition increases the capacity to experience imagery (see Bryant & Harvey, 1995; Rauch, Foa, Furr & Filip, 2004) and imagery vividness (Laor, Wolmer, Wiener, Sharon et al., 1999). As noted previously, imagery beyond an optimal frequency is likely to result in distress (Dadds et al., 2004). In turn, this distress leads to further intrusions (Kosslyn, 2005b). Given the interaction of intrusions and anger in PTSD, increased image frequency and clarity is an obvious risk for increasing the experience of anger. The second irony is that, just as thought suppression has been shown to result in a rebound effect in PTSD intrusions (e.g., Clark, 2002; Davies & Clark, 1998), attempts to suppress intrusive imagery are likely to lead to perverse and unintended increases in imagery (Holmes, 2003). This effectively forms a psychological double bind for the individual - between being assailed by frequent, out-of-control, vivid, intrusive visual material and
associated loss-of-control of thoughts and feelings and succumbing to the false-refuge of attempting to suppress such material, with its inevitably amplifying consequences. By comparison, depression has perhaps the least strong relationship with imagery. Although research has recently suggested imagery may be associated with lowered mood (Bywaters, Andrade & Turpin, 2004b), dysphoria and depression (Holmes, Lang, Moulds & Steel, 2008; Pearson, Rhodes & McCarron, 2008; Wheately & Hackmann, 2011) and even suicidality in the context of depression (Crane, Shah, Barnhofer & Holmes, 2012), this research is relatively recent and of insignificant magnitude. Thus, the view that depression is a ruminative affect strongly influenced by word-based cognition (see Fresco, Franke, Mennin, Turk & Heimberg, 2002; Segal, Lau & Rock, 1999) continues to prevail.

5.5 Summary

To conclude, although limited attention has been given to the contribution imagery can make to anger in PTSD, there are several interwoven sources of evidence which indicate it can contribute significantly to the relationship of anger to PTSD. Figure 5.1 presents a schematic representation of the hypothetical interaction of these neuropsychological, emotion and disorder-based and imagery influences on anger in PTSD. In accordance with the lines of evidence reviewed, it depicts imagery and anger as intimately connected, especially by threat vigilance and other-involvement. Imagery and anger are shown to potentiate and be potentiated by PTSD symptoms. This is particularly so where responsibility cognitions are prominent. Through intrusion-related distress and practice effect(s), anger and imagery are reiteratively increased. Simultaneously, neuropsychological functioning facilitates anger and imagery and their relationship by differing, yet overlapping neural networks, and via an added, recursive, independent pathway between anger and PTSD symptoms.

This chapter established the evidence to support the idea that imagery is important to anger in PTSD. The next chapter catalogues the variables which affect imagery in anger in PTSD.
Figure 5.1 Prototypical Summary Model of the Relationship between PTSD Symptoms, Neuropsychological Function, Visual Imagery and Anger in PTSD
CHAPTER 6: FACTORS AFFECTING THE MANIFESTATION AND MAINTENANCE OF IMAGERY IN ANGER IN PTSD

The unaided human mind is vulnerable to many fallacies and illusions because of its reliance on its memory for vivid anecdotes

Steven Pinker

As demonstrated across the preceding chapters, PTSD sufferers have an increased likelihood of experiencing dysfunctional anger. Fifty per cent or more can be significantly affected by anger and anger-affected PTSD may be reasonably viewed as a sub-type of the disorder. To some extent, this angry form of PTSD can be explained by the operation of anger-associated intrusive imagery focused on the traumatic event(s) and its consequences for the present and future. Supporting evidence for this proposition was detailed in the latter portion of Chapter Four and in Chapter Five.

The present chapter reviews a number of factors which bear upon imagery’s four functional qualities: generation, inspection, maintenance and manipulation as defined by Kosslyn’s Computational Model of Imagery (Kosslyn et al., 1990; Kosslyn et al., 2005a). They are henceforth referred to as imagery’s manifestation and management. General factors that impact on imagery include demography, individual variability in imagery capacity and the intrinsic nature of imagery. Negatively-valenced emotion, stress and posttraumatic stress moderate the effect of these factors. Each requires description.

6.1 Demographic characteristics that influence imagery

It is well established that the manifestation and management of imagery is influenced by demography (Chambers, 1997; Giambra, 1977; 2000a; 2000b). The following have been demonstrated as influential.

6.1.1 Gender. There is evidence that gender is implicated in the experience of imagery (Campos, Perez-Fabello & Gomez-Juncal, 2004; 2014). Men have been shown to react more strongly to pleasant affective pictures, while women react more strongly to unpleasant affective pictures (Sabatinelli, Flaisch, Bradley, Fitzsimmons & Lang, 2004). Women also experience more vivid images than men.
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(Campos, 2004; Richardson, 1995), and have a superior ability to generate and maintain images (Kosslyn, Margolis, Barrett, Goldknopf & Daly, 1990).

6.1.2 Age. Age has a clear effect on imagery. Children use imagery more than adults (Kosslyn et al., 1990) and imagery in many forms decreases with age (Giambra, 1979, 2000a; 2000b; Kosslyn et al., 1990). It has been hypothesised that the selective loss of neurons due to pathologies occurring across time interferes with image production, resulting in fewer unbidden images, reduced spontaneous shifts of attention to them and decreased absorption in imagery (see Giambra, 2000a; 2000b). Similarly, comparison of younger and older adult males in top-down processing tasks, shows evidence of age-degraded performance on selective - for example imager rotation and activation - but not all imagery processes (Dror & Kosslyn, 1994; 1998).

6.1.3 Culture and ethnicity. Imagery is culturally ubiquitous and yet reflective of culture (Macinnis & Price, 1987; Singer, 2006). To illustrate, transmission of oral history legends and myths via inter-generational recollection are, according to Singer (2006), powerfully reliant on imagery’s content, storage and recall. There is evidence, nevertheless, of racial imagery differences such that peoples from East Asia (e.g., Chinese, Japanese and Koreans) tend to generate more imagery than Westerners (Liang & Kale, 2011) and East Asian students tend to have a more visual cognitive learning style than their Australian counterparts (Fallon, 2004). North American and Australian indigenes are likewise known to have superior visuo-spatial memory to Caucasians (Greenbaum & Greenbaum, 1983; Kearins, 1981; 1986; More, 1987), while Japanese people score higher than Caucasians on tests of visual memory (Flaherty & Connolly, 1996) and Irish and Japanese perform better on mental rotation tasks than Hispanic peoples (Flaherty, 2005).

In summary, imagery’s manifestation is greater among the peoples of East Asia along with indigenous cultures with practical day-to-day connection to the land. It is stronger in women and decreases with age.

6.2 Individual differences in imagery

Imagery’s impact is also determined by several individual psychological factors. These relate to differences in innate capacity, temperament/disposition and cognitive-style.

6.2.1 Innate imagery capacity. D’Argembeau and Van der Linden (2006) reviewed factors associated with the subjective experience of remembering the
past and imagining the future. They concluded that individuals with a higher capacity for visual imagery experience more visual and other sensory details when remembering or imagining past and future events. Capacity may be variously measured.

In 1970, Singer and Antrobus attempted to identify prevalence, content and structural dimensions on which capacity may differ. They identified 29 such dimensions, including imagery frequency, absorption, vividness, temporality (i.e., past, present or future sense), reality (and conversely bizarreness), positivity (and conversely dysphoria - i.e., anxiety, sadness and anger), sexual content and wrongdoing. Beyond these dimensions, MacInnis and Price (1987) and Pearson and others (2013) respectively reviewed the role of imagery in information processing and clinical phenomena. They noted the importance of dimensions related to imagery content and (verbal versus imagery) information processing style.

Research attempting to map these dimensions for their effect on the manifestation and management of imagery has commonly emphasised several aptitudes. One relates to control or the ability to regulate imagery. This has been investigated across a variety of populations and experimental conditions. Often measured by reference to the extent or volume of imagery (e.g., via the number of images evoked and their frequency: Lee & Gretzel, 2007), control has been studied in survivors of missile attacks (Laor et al., 1999), the treatment of post-traumatic nightmares (Krakow et al., 2000) and various anxiety disorder populations (Krans, 2011). Such research demonstrates that uncontrolled or dysregulated imagery is central to psychopathology manifestation and maintenance.

One of the most frequently investigated aspects of imagery is vividness. Indeed, measures of vividness are among the earliest imagery metrics developed. Assessed by reference to the clarity, intensity and distinctiveness of an image (Libby & Eibach, 2013; MacInnis & Price, 1987; Pearson et al., 2013), vividness has been investigated across a variety of populations and conditions, including, incarcerated criminal offenders (Stricklin & Penk, 1980) and PTSD (Cahill et al., 2003; Rauch et al., 2004), treatment seeking depression patients (Torkan et al., 2014), personality and anxiety disorders and phenomena such as optimism in future imagery (Blackwell et al., 2013). Such research demonstrates that vividness increases both the positive and negative impact(s) of imagery.
Absorption, or the degree to which immersion in imagery dominates attention, is another capacity shown to affect imagery’s impact. This, in part, reflects the image’s affect-eliciting properties, content and nowness (i.e., its temporality). Absorption has been investigated in a variety of populations and conditions, particularly anxiety (Hagenaars & Holmes, 2012). Such research has shown absorption correlates highly with the tendency to image, and that individuals vary considerably with respect to the intensity of imagery experience due to differences in absorption levels (Levin & Young, 2002; Lynn & Rhue, 1988).

6.2.2 Dispositional style. Personality phenomena and imagery are interactively related and there are many abilities or traits related to the capacity to image among them absorption, reality-imagination thinness of boundaries and imagery vividness (Radar, Kunnzendorf & Carrabinboi, 1996). General and psychopathological studies of imagery and personality illustrate this connection. Various evidentiary bases for this relationship were detailed in Chapter Five and several mechanistic links between imagery and psychopathology are articulated in subsection 6.4.

Reviews suggest that personality-based types exist (MacInnis & Price, 1987). Factor analyses of Singer and Antrobus’s (1970) Imaginal Process Inventory by Huba and Singer (Huba, Segel & Singer, 1977; Segal, Huba & Singer, 1980) produced a shortened version, the Short Imaginal Process Inventory (SIPI; Huba, Singer, Aneshensel & Antrobus, 1982). Comparison of factor analyses of the SIPI against the McCrae and Costa (1996) Five Factor Model of Personality (5FMoP) by Zhiyan and Singer (1997) showed the three distinct elements of the 5FMoP were positively correlated with facets of imagery. These comprise the associations of: (a) openness to experience with a positive imagery factor, (b) low levels of conscientiousness with a poor attentional control factor and (c) neuroticism with a guilty-dysphoric imagery factor.

There is concordant research linking personality factors to imagery aptitudes. Greater imagery control is, unsurprisingly, associated with greater internal locus of control (Bryan, 1999), while extraversion and introversion are associated with imagery fluency (see Huckabee, 1974). The latter is consistent with the long-standing interpretation that extraverts are verbalisers and introverts are imagers (Riding & Cheema, 1991; Riding & Raynor, 1998).
Imagery vividness has been associated with angry obsessionality as referred to in type-A personality (Dyck, Moser & Janisse, 1987) and individuals with high trait anger have been shown to have greater reactivity to angry imagery in the absence of enhanced imagery ability (Slomine & Green, 1993). It appears, however, that the relationship of personality and imagery vividness may be mediated by distress levels. Stricklin and Penk (1980) found that, among incarcerated female offenders, extroverts reported more vivid imagery than introverts under high-distress, whereas introverts reported more vivid imagery than extroverts under low-distress.

Imagery absorption is another dispositional trait found to correlate highly with the tendency to image and the intensity of the imagery experience (Belicki & Belicki, 1986; Roche & McConkey, 2000). Under conditions of significant stress, absorption is considered to be an imagery-based coping mechanism (see subsection 6.4.4.1).

### 6.2.3 Cognitive style

Individuals have preferred information processing methods. They have been referred to as cognitive style since Allport’s (1937) classic work in the area, and supported by contemporary research. These preferential information processing methods occur via lexical and imagery-based cognition. Each profoundly affects the way people behave and emote. Based on a long history of research on perception, and increasing interest in visual imagery in experimental and clinical research, there is consensus that imagery plays an essential role in information processing. The style of imagery-based information processing is affected by appraisal tendencies, the demands of reflective thinking and the balance between it and thought-based information processing or as it is known verbalising style (Riding & Cheema, 1991; Riding & Raynor, 1998).

#### 6.2.3.1 Appraisal tendencies

Imagery is quasi-perceptual and subjectively determined and, therefore, does not share one-to-one correspondence with external realities. Rather, its operation reflects the nature of memory storage. In turn, storage and retrieval of imagery are inherently influenced by subjective, situation-specific appraisal(s). Pinker (1999) cautions that images are individual constructions of reality that are registered mentally, compared with other related images and then used to construct new images. He states that, in this way, mental images allow individuals to form theories of how the world works without having to directly experience the various possibilities involved.
These image-influenced world theories are essential as human functionality relies on the inherent knowledge-gap filling of assumptions (Kahneman, 2011; Kahneman & Tversky, 1984; Tversky & Kahneman, 1974). A problem, however, exists in that such imaginal constructions are the cumulative result of prior experience and imagery recall and storage fundamentally bias what one perceives (Kosslyn, Thompson, Kim & Alpert, 1995). These biases affect a plethora of real-life, imagery-dependent functions as diverse as eye-witness testimony and threat detection in fear and phobia maintenance. These are elaborated upon in subsection 6.4.3.

### 6.2.3.2 Reflective thinking

Another aspect of cognitive style associated with imagery is the tendency toward reflective thought. Intrinsically associated with negative emotion, distress and negative life events, the impact of reflective thinking on imagery is supported by theoretical and research evidence.

One example of the impact of reflective thinking has on the theoretical models of imagery influenced cognition and emotion described by Lang (Lang, 1979; Lang, Bradley & Cuthbert, 1997) and Ahsen (1982; 1984). These themes emphasise the crucial importance of imagery to psycho-physiological meaning propositions involving cognitive, affective and somato-visceral elements and vice versa. It is also consistent with psychopathology research based on Paivio’s (1971) binary storage model of cognition, including Brewin’s dual representational model of PTSD (see Brewin et al., 1996). As noted in Chapter Two, these models emphasise the role of imagery-based Situational Accessible Memories in lower-level perceptual processing of traumatic scenes. This processing, which is not verbally mediated, is focused on the intense arousal associated with the event and results in unremitting distress. It is also consistent with Kosslyn’s insightful proposition (Kosslyn 2005a; 2005b) that highly emotive images, when formed, allow additional retrieval cues to be entered into long term memory, thereby making it more likely such images will recur.

The association of imagery and reflective thinking is further supported by studies of autobiographical memory, where imagery is considered to play a dominant (Conway, 1990). Illustrative of this, Schonfeld and Ehlers (2006) showed the experimental presentation of a novel picture version of the autobiographic memory test facilitated autobiographical memory recall, albeit of an overgeneralised type, in PTSD sufferers. Greenberg and Knowlton (2014), in a three-part experimental study of the role of visual imagery in autobiographical memory and cognitive style, confirmed that imagery was associated with individuals’ belief they were reliving
their memories. Rubin and Bernstan (2009), in research on the frequency of voluntary and involuntary autobiographical memories across the life span, reported visual imagery was equally implicated in both forms of autobiographical memories.

### 6.2.3.3 Imagery-thought balance

It is long recognised that individuals differ in the capacity to image or, as it has been termed, fluency (Paivio, 1970; Sheehan, 1972). Thus, there are vivid imagers who tend to construct sensory representations of unconscious visual thoughts and imageless thinkers for whom mental activity is consciously experienced but has no sensory or perceptual character (Kunnzendorf, Young, Beecy & Beals, 2000; Thomas, 2014). Early researchers referred to a dichotomy of visualisers and verbalisers (e.g., Richardson, 1969). However, the idea that there are discrete categories of imager is no longer accepted and the capacity to produce and manipulate imagery is understood as normally distributed (Thomas, 2014) and inter-related with verbal processing.

In reality, the same person may evoke thought and imagery-driven cognition independently, consecutively or simultaneously. Thus, as argued above, thought-based appraisals are inevitably linked to imagery. Nothing is purely verbally or visually imagined and the relative balance of imagery and thought in cognition is a reflection of cognitive style, both as an expression of disposition and preference. This is recognised in research on cognitive style which emphasises the connection of these dual aspects. This is epitomised in research and theory around the Wholistic-Analytic and Verbaliser-Imager dimensions articulated by Riding (e.g., see Riding & Cheema, 1991; Riding & Raynor, 1998).

In summary, imagery’s impact varies with innate capacity - those with less control, higher vividness and “newness” of content are likely to experience greater imagery impacts. This is, in part, determined by temperament, disposition and cognitive style. There are also implicit imagery-related factors which influence its impact. These are now considered.

### 6.3 Intrinsic imagery-related factors

The manifestation and management of imagery reflects the qualities of the specific imagery undertaking involved. Three characteristics have a particular impact: motivation, specific task requirements and repetition effects of the imagery task. Although their importance has only recently begun to be documented in mental imagery research (Hagenaars & Holmes, 2012; Holmes & Mathews, 2010; Pearson et
al., 2013), these factors are well described over time in research on perceptual imagery. Before discussing each of these, it is timely to reiterate that, although perception and imagery are different phenomena, imagery is perception dependent and stored and retrieved imagery is the fulcrum on which perception turns and research of either set of processes illuminates the other.

6.3.1 **Motivation.** It is widely accepted that, at any time, the information array confronting an individual is greater than can be fully processed (Parkhurst, Law & Nieber, 2002). Contemporaneously attempting to identify all relevant elements of interest is unworkable and selective attention must occur. In the absence of selective attention, overload and functional inoperability is likely.

To prevent inoperability, attention is drawn to elements of the array via (data-driven) bottom-up and (schema-driven) top-down information processing (Parkhurst et al., 2002; Thomas, 2014). Bottom-up processing represents an initial sweep of the perceptual field and reflects lower cortical processes. Differences in patterns, shapes, colours, novelty, movement and sudden changes of direction are detected and the observable physical properties of a stimulus noted (Thomas, 2014).

Top-down processing, in contrast, guides searching in the stimulus array to critical targets, the appearance of which, on the basis of prior experience, is known in advance (see Wolfe, 1994; 1998; Niebur & Koch, 1996). More so than bottom-up processing, it directs what is included in an image and, therefore, what can be processed. Some time ago Chambers and Reisberg (1985) contended that it is cognition’s upper levels, especially intention, which deserves greater research attention. This proposition has since found general consensus in the perception literature (see Chambers & Reisberg, 1992; Liang & Kale, 2011; Rouw, Kosslyn & Hamel, 1998).

The importance of top-down intention is equally important to mental imagery. This is because top-down processing is driven by motivational relevance or stimulus-salience, which not only directs attention, but enhances elaborative perceptual processing (Sabatinelli et al., 2004). Elaboration reflects, but is not dependent on, perception. As an important illustration of this, colour blindness does not prevent humans from interpreting stimulus salience related to hue (Thomas, 2014) and even those blinded from an early age report experiencing colour imagery (Bridge, Harrold, Holmes, Stokes & Kennard, 2012).
Various theories seek to explain the operation of top-down imagery processes. Three derive from the psychology of sport and performance, where considerable work has occurred. One is Sackett’s (1934) Symbolic Learning Theory. It proposes mental imagery operates as a coding system that provides the opportunity to practice elements of a motor task and acquire a cognitive program or blueprint for its accomplishment via specific movement patterns that facilitate automaticity through cognitive processes (see Janssen & Sheik, 1994; Martin, Moritz & Hall, 1999).

Another is Arousal/Activation Theory (Schmidt, 1982). It proposes imagery rehearsal lowers the sensory threshold required for task performance, selectively focuses attention on what is to be accomplished and creates an optimal level of psychophysiological arousal for task accomplishment. The final is Suinn’s (1984; 1997) Visual Motor Behaviour Rehearsal Model. It posits imagery is imagery is central to the complete (re)integration of experience involving visual, auditory, tactile, emotional and kinaesthetic cues in a holistic process and has demonstrated that physiological responses stem from an athletes use of mental imagery.

These theories and the associated research are powerful illustrations of how imagery invokes a mind-body dualism that is critical to the objectives to which imagery is targeted. Overarching explanations of imagery’s role in pathological process are described in section 6.4.3. Regardless of their different foci, the two theoretical domains are relevant to the other in stressing that imagery is functionally organised and core to psycho-psychophysiological responses in everyday and abnormal stress.

6.3.2 Task requirements. The fluency with which imagery is manifested and managed is also affected by the specific requirements of the task. At one level, this relates to task difficulty. For example, perception research reveals that efficacy in image recognition and description, as measured by time-to-task-completion, corresponds to the distance between foci on the depicted object and the relative size of images in comparative illustrations (Kosslyn, 1973; Kosslyn, 1975; Kosslyn, Ball & Reiser, 1978). Similarly, in tasks involving mental rotation of similar objects, the time required to determine object shape similarity and rotate objects respectively depends on the number of rotations required to bring target objects into comparable alignment (Shepherd & Meltzer, 1971) and the amount of rotation required (Cooper & Shepherd, 1973).
Pearson and colleagues (2013) argue that task characteristics are also important to the mental imagery that can accompany psychopathology and associated treatment approaches. They refer to the mental synthesis required to produce imaginal patterns and create insights into such patterns, arguing that psychological treatment techniques like imagery rescripting make use of such processes. Applied to PTSD treatment, it is equally probable that prolonged exposure therapy and other evidence-based approaches like EMDR are also reflective of such processes (see McNally, 2007; Brewin, Gregory, Lipton & Burgess, 2010).

The strategies employed also affect the fluency with which imagery occurs. For instance, the way in which instructions are communicated strongly influences image generation efficacy, this being true of words, pictures and diagrams (Liang & Kale, 2011; Paivio, 1971). Paivio established that concrete words are more effective in generating imagery than their abstract counterparts (1965) and that words which evoke imagery are better remembered (1969). One strategy particularly likely to evoke imagery is to incorporate people (especially significant others) into instruction sets. This applies to concrete and abstract imagery tasks (Bent & Wick, 2006).

**6.3.3 Repetition effects.** Repetition affects imagery in several ways. As for any cognitive skill set, there is a practice effect that enhances imagery fluency and, in the absence of psychopathology, efficacy (Ahsen, 2001). Consistent with this view, Suler (1985) demonstrated that, given adequate training in the use of imagery, even poor imagers can enhance their abilities. This is supported by Belicki’s (1992) research on nightmares, psychopathology and visual imagery, wherein she found imagery frequency increases imagery intensity (as measured by the frequency and impact of dreaming).

Evidence of the positive effect of rehearsal and practice on imagery is also demonstrated by skill improvement where performance is targeted in guided imagery and visualisation training. This is represented in the Symbolic Learning, Arousal/Activation and Visual Motor Behaviour Rehearsal Models briefly reviewed above, each of which have a cognitive blueprinting, enabling, organising and integrating role (Janssen & Sheik, 1994; Martin et al., 1999; Schmidt, 1982; Suinn, 1984; 1997).

There are, of course, limits to practice effects. Limiting factors include the effect of motivation. Imagery enhancement is more readily achieved using motivating factors that are salient. Imagery fluency also declines in the absence of practice. A
further limit is the negative consequence of high frequency, unintended and involuntary imagery that can occur in negative emotional states.

In summary, the management and manifestation of imagery is affected by a range of imagery-related qualities. Principal among them are the purpose of imagery, its personal relevance and meaning and the effect of practice. As purpose, salience and motivation in imagery increase in importance, the impact of imagery increases. This is particularly noticeable in autobiographical memory. Such processes and their impact inevitably reflect prior experience and prior knowledge fundamentally biases what one perceives and remembers. Thus, it is assumed that humans have inherent individualised mental models that guide imagery of both a perceptual and imaginative kind (Kosslyn, 2005b; Pinker, 1999).

6.3.4 Imagery-thought interplay. Although imagery is sometimes conceptualised and researched as if it were a distinct cognitive process from thought (Moritz et al., 2014), the two phenomena are strongly interlinked. This is provided for in the dual-coding model of cognition originated by Paivio (1971), which has long understood information processing may occur by either verbal association or visual imagery or both. Further, imagery and thought have the potential for expanding or limiting each other’s role.

As noted previously, Kosslyn (2005b) proposed that reflective thinking and imagery are strongly connected and that imagery plays a central role in reflective thinking processes. He emphasises the “normal” development of cognition and meaning are programmed by imagery and most cognitive activity occurs without conscious reflection. This view coalesces with summaries and reviews by key researchers that emphasise the interplay between thought and image-based elements of cognition (e.g., Holmes and Mathews, 2010; Pearson et al., 2013; Singer, 2006). This literature shows that, beyond a point, high prevalence imagery interferes with the capacity to reason (Knauff & Johnson-Laird, 2002), performance on imagery tasks is impeded by verbal coding of stimuli (Brandimonte & Gerbino, 1993) and even the deliberate construction of imagery (whether it be self-initiated or in response to instruction) may “interfere with the underlying processes of unconscious visual thinking” (Kunzendorf et al., 2000; page 981). It appears therefore, that imagery prevalence at either extreme of the possible continuum of imaginal experience has the potential to result in dysfunction.
There are various explanations for this functional confounding. One relates to the interference that occurs in working memory when the brain systems involved are in competition rather than co-operation (Englehard et al., 2011). As noted previously, a preponderance of either compromises the integrity of the other. For example, excessive imagery is associated with the decreased verbalisation of social anxiety, while excessive thinking is exemplified in the reduced visualisation of generalised anxiety disorder (Borkovec & Inz, 2009).

6.4 Imagery amplification in the context of disordered negative emotion, stress and traumatic stress

The manifestation of imagery is distributed on a continuum, ranging from productive to dysfunctional (Holmes & Hackman, 2004; Holmes & Matthews, 2010; Pearson et al., 2013). As discussed, where an individual’s experience of imagery is best located on this continuum is, in part, due to demographic, individual and imagery-associated characteristics. How imagery is manifested also reflects the environmental context in which it occurs.

In the absence of abnormal stress, imagery operates as a functional tool and is clearly often beneficial. Where any or all of high-valence negative emotion, negative life events or stressors are present (especially that which is traumatic in nature), they recursively amplify imagery’s fluency and its role in the elicitation of psychopathology. Although this imagery can be voluntarily elicited, it is its intrusive and avolitional aspects which predominate and are most commonly dysfunctional, distressing to individuals and clinically critical (Kranks, 2011). This imagery-context interaction is well-accounted for by a stress-diathesis perspective which emphasises that, given sufficiently intense stress, underlying individual imagery proclivities will find expression as disordered levels of imagery. For example, as represented in imagery control, vividness and/or absorption. Thus, the influence of demography (gender, age and ethnicity), individual psychological factors (innate ability, dispositional style and cognitive style) and intrinsic imagery-related factors (motivation, task requirements and imagery-thought interplay) on imagery manifestation and management are affected in the following ways.

6.4.1 Negative Emotion. Imagery may precede a feeling. As noted in Chapter Five, research has highlighted the significant impact of imagery on emotion (Beck & Emery, 1985; Holmes & Mathews, 2010; Martin & Williams, 1990) and it
appears that there is a special relationship between imagery and emotional memory. Representative of this, information processing instructions which require individuals to imagine rather than think about tasks elicit stronger emotional responses (Holmes, Mathews, Mackintosh & Dalgleish, 2008). Researchers have thus concluded, imaginal processing is more emotionally coloured than verbal information processing (Hagenaars & Holmes, 2012; Holmes & Mathews, 2005; 2010). Imaginal processing also has an enduring, significant impact on emotion, and can imbue emotions with intensity consistent with the actual objects of emotion (Kunzendorf, Hartmann, Thomas & Berensen, 1999) and can even influence the ability to experience emotion itself (Suler, 1985).

This relationship is bidirectional and it has, importantly, also been shown that emotion causally precedes (Hagenaars & Holmes, 2012). Research shows that viewing aversive, emotional filmed material leads to increased intrusive images about the material, but not intrusive verbal thoughts (Hagenaars, Brewin, van Minnen, Holmes & Hoogduin, 2010). Similarly, highly emotion-arousing tasks are known to increase vivid imagery (Bywaters et al., 2004b). The finding that the best remembered parts of an imaginal memory are its emotional elements has been interpreted as being due to their superior encoding in memory due to imagery (Arntz, de Groot & Kindt, 2005).

6.4.2 Anger. Research has demonstrated anger’s impact on imagery. High levels of anger are associated with greater responsivity to imagery, regardless of imagery ability or repetition (Janisse et al., 1986; Slomine & Green, 1993).

Multiple mechanistic links and potentiators can be involved. These may reasonably be understood to comprise: (1) appraisal-tendencies and their meaning-relatedness - as demonstrated by reference to angry threat perception; (2) anger’s interconnectedness with other emotions; (3) the culpability in conduct of self/others, where the absence of intent is known to be associated with less intense anger; (4) the morality and injustice of behaviour committed by the self or others and (5) the malevolence of that behaviour (see Lemerise & Dodge, 2000; Litz et al., 2009; Wranik & Scherer, 2010).

With each successive anger-imagery link inclusion, anger can become more dysfunctional, distorting of perception and judgement and increasingly resistant to decay. This is ultimately exemplified in angry, imagery-influenced rumination of an over-generalised autobiographical nature, coloured by a loss of control and the
tendency to think repetitively and passively about negative emotions/symptoms of distress, precipitators of negative events and the meaning of that distress and those events.

6.4.3 Stress. Beyond certain levels, stress is inherent to dysfunction. The relationship between the intensity of stressors and reduced performance was classically articulated in the Yerkes-Dodson Law in 1908 (Cohen, 2011). Since then it has been the subject of ongoing research programs, like those of Cohen (see Cohen & Wills, 1985; Cohen & Janicki-Deverts, 2012) and Lazarus (see Lazarus & Folkman, 1984; Lazarus, 2006). Although not accounted for specifically in such research, as noted in Chapter Five, the existence of a bi-directional relationship between imagery and high valence negative emotion and stress is well established in research on imagery’s clinical impact of imagery (e.g., see Holmes & Hackman, 2004; Holmes & Mathews, 2005; 2010).

Although such imagery processes can be self-directed, high demand stress, (e.g., due to insufficient time, task complexity and outcome contingencies), automated top down information processing tends to predominate. It is this form of imagery that is most commonly dysfunctional, distressing to individuals and clinically critical (Krans, 2011). Consequently, there are striking examples of error and dysfunction due to imagery in stressful real life tasks as diverse as retrospective eye-witness identification in legal environments (see Arkowitz & Lilienfeld, 2010; Davis & Loftus, 2012), ill-advised imagery and emotion-affected securities trading (MacGregor, Slovic, Dreman & Berry (2000)), heightened detection biases associated with false positive signals in specific phobias [(e.g., for snakes, spiders, rodents and dogs (de Jong et al., 2002; Haberkamp et al., 2103). In the domain of traumatic stress, McNally’s work around the nature of memory failure after traumatic stress, wherein he emphasises the fallibility of memory due to such factors as memory disorganisation, the memory prolonging effect of event malignancy and increased recall errors in children and adolescents is particularly incisive (McNally, 2005; 2006; Schwarz, Kowalski & McNally, 1993).

There are two notable explanatory theories for how imagery is linked to psychopathology. Lang’s Bioinformational Theory of Emotional Imagery (Lang, 1979; Lang et al., 1999) argues that an image is a functionally organised set of mental propositions. Lang has long suggested that vivid recollection of an emotional event depends on the workings of an associated information network (Cuthbert et al., 2003).
He proposes imagery acts as part of a cognitive propositional network that intimately links physiological and behavioural patterns. In simple terms, there are stimulus and response propositions. The former describes the content of the scenario to be imagined. Response propositions, as implied, describe the individual’s response to that content. Cues can be external or internal. Irrespective, the image acts as a template for overt responding. Modifying it or overt behaviour will result in a change in the other. According to Lang, there is a correlation between image vividness and psychological reactivity under emotionally charged conditions.

Lang (1979) proposed emotion may be experienced by reference to thinking (words, phrases, propositions) or pictorial representation (diagrams, pictures, symbols, images). He asserts that emotion related words are rarely only processed as words because language is connected to areas and networks of the brain related to physiological and behavioural responding. Lang also observed that single words describing emotion-related events, through their connection to imagery, have been repeatedly shown to prompt the same bodily responses as those cued by the events themselves.

The second model that seeks to explain psychopathology by reference to imagery is Ahsen’s Triple Code Model (Ahsen, 1982; 1984). According to him, there are three fundamental elements to an image. The first is that the image must be centrally arousing and cause the individual to interact with the image as if it were representative of the real world. Second, it must include a somatic response element, such that imaging results in somato-visceral changes. Finally, the image must contain a specific meaning, the content of which will vary individual-by-individual in accordance with their developmental experiences and individual characteristics, regardless of the similarity of the imagery instructions involved.

Ahsen and Lang have written extensively on the nature of cognition over the last several decades. Ahsen’s argument differs from Lang’s in that he gives additional emphasis to the role of imagery in cognition. He argues against the idea that physiological responses emerge from propositional networks and, instead, suggests an image can cause a direct physiological response (Suler, 1985). In addition, Ahsen is the only theorist whose model overtly articulates the direct representational function of meaning in imagery.

Regardless of their specific differentiating points, both theories emphasise that idiosyncratic meaning and its correlates of motivation and intention are implicit
to the symbolism, priming, and mental propositions, integrational and somato-visceral elements which lie at the core of imagery’s dysfunctional manifestation. Such meaning may appear irrational to the observer. Regardless, meaning is strongly implicated in the selective attention required to make sense of the extant sensory array and, thereafter, the top-down salience determination that secondarily guides perception. It is, similarly, strongly associated with autobiographical memories, image comparison and synthesis and the resultant world-theories that may arise for the individual.

6.4.4 Posttraumatic Stress. Dysregulated imagery is centrally associated with high impact psychological distress (Bywaters et al., 2004a). Posttraumatic stress disorder is a prime example of this. Various factors influence this connection and require description.

6.4.4.1 Posttraumatic stress and disposition. Personality oriented PTSD research emphasises that the disorder’s expression reflects the operation of identifiable personality dimensions (Holeva & Tarrier, 2001). Research on combat veterans has, accordingly, referred to the existence of subgroups based on personality styles (e.g., Pierkarski, Sherwood & Funari, 1993; Forbes et al., 2010). Personality disorders identified for their association with PTSD among veterans include obsessive compulsive, schizoid, avoidant borderline and anti-social types (Hyer, Davis, Albrecht, Boudewyns & Woods, 1994; Sherwood, Funari & Pierkarski, 1990; Southwick, Yehuda & Giller, 1993).

One plausible explanation for the association between PTSD and personality relates to the role of imagery. Sympathetic to this view, Belicki and Belicki (1986), investigated the possibility that the tendency to readily employ imagery predisposes individuals with PTSD to nightmares. They asserted that imagery is not only a reflection of personality, but may be part of personality style itself. Around the same time, Grigsby (1987), while reflecting on the use of imagery in combat-related PTSD psychotherapy, argued that the capacity to deploy imagery relates to personality and is most likely genetic in origin. PTSD associated phenomena demonstrated to be associated with personality variables include nightmares (Levin & Masling, 1995), intrusive imagery and thought (McCarthy-Jones, Knowles & Rowse (2012), depersonalisation in the context of traumatisation (Levin, Sirot, Simeon, & Guralnick, 2004; Ross, Farley & Schwartz, 2004) and diminished emotion regulation and impulse control (Kraus et al., 2010).
Importantly, there is evidence that anger in PTSD is in some part a reflection of personality. As observed in Chapter Four, Miller and others (see Miller et al., 2004; Miller & Resick, 2007) and Forbes and associates (2010) have highlighted that internality-externality personality dimensions account for anger in Australian Vietnam veterans and their US counterparts, which have been noted to be strikingly similar in their personality dimensions (Elhai, Forbes, Creamer, McHugh & Frueh, 2003).

The directionality of this traumatic-stress-personality-imagery relationship is innately difficult to determine. Causally, it is possible that increasing posttraumatic stress levels facilitate imagery, albeit at intensities underwritten by personality. The opposite explanation that imagery influenced by personality renders individuals more liable to stress reactions and negative clinical implications is also possible. Regardless of actual cause, the evidence suggests latent personality variables find expression in PTSD and imagery may be an important downstream outcome of this manifestation. This possibility is entirely consistent with the stress-diatheses explanatory models described earlier and across Chapters Two to Four.

6.4.4.2 Posttraumatic stress and cognitive style. There is evidence that a cognitive style that is oriented toward imagery is exacerbated in the presence of anger and PTSD. Gerin and colleagues, reporting on the role of imagery in emotional arousal, chronic stress and responses to provocative events in cardiovascular disease patients (see Gerin, Davison, Christenfeld, Goyal & Schwartz, 2006; Gerin et al., 2012), state that acute stressors typically generate a constellation of negative cognitions and emotions that form a persistent mental representation of the originating event. This cardiovascular research has found similar relationships in angry PTSD (Beckham, Calhoun, Glenn & Barefoot, 2002; Beckham et al., 2002; Vrana, Hughes, Dennis, Calhoun & Beckham, 2009). Such persistence in memory and its retrieval is redolent of rumination.

Rumination is an enduring and stable personal characteristic. Once thought to be the province of depression related states, rumination of an angry form is now being given considerable attention (see Denson, Moulds & Grisham, 2012; Potegal, 2010; Siedlecka, Capper & Denson, 2015; Tripp & Bies, 2010). As noted earlier and in prior chapters, a ruminative style of emotion regulation is thought to be closely linked to re-experiencing in PTSD and it is considered a cognitive vulnerability for the development of PTSD (Elwood et al., 2009).
Although traditionally perceived as a consequence of thought, the literature suggests that rumination contributes to ongoing threat perception via cognitive appraisal and visual imagery, especially in PTSD. Angry rumination is, perhaps, the primary example of where imagery and thought meet as super-abundant elements of cognition and emotion. To recall the observation of Beck and Emery 25 years ago, undesirable imagery stimulates verbal cognitions (Beck & Emery, 1985). The combination of both is likely to impact more than either alone. Thus, visual imagery, rumination and anger appear inextricably linked in PTSD.

This is not to say imagery associated with rumination in PTSD is inevitably abnormal, for even intrusive emotional images in PTSD are not beyond the realm of normal imaginal experience (Holmes, 2003). Each can be involuntarily provoked by external stimuli or voluntarily self-initiated and are most typically autobiographical in nature (see Ehlers, 2010; Greenberg & Knowlton, 2014; Rubin & Bernstan, 2009). Unlike rumination, imagery can be content benign. Rumination, in contrast, is inevitably interpretive and biased due to both its thought and imagery-based elements.

Cognitive appraisals are crucial to the development of rumination in PTSD across trauma types. As noted in Chapter Four, Foa and associates (1999) hypothesised three highly impactful types of negative apply: negative cognitions about the self, the world and self-blame. There is also evidence attentional bias toward threat results in associated with fear conditioning via exaggerated detection of anomalies (McTeague et al., 2010). Preoccupation with intrusive imagery is directly implicated in this. Whether it takes the form of facilitated orientation to, or delayed disengagement from, trauma-related stimuli, it is maladaptive in that it renders incomplete processing of corrective information and/or results in an inefficient cognitive processing style, impairing subsequent processes like detailed memory retrieval (Fani et al., 2012). Simultaneously, rumination in PTSD is subject to decreased autobiographical memory specificity (Moore & Zoellner, 2007). Both are capable of interfering with the operation of the trauma memory network (see Wheately, Hackman & Brewin, 2009).

Eventually, rumination can alter the proclivity for imagery. In veterans with severe combat-related trauma exposure, for example, those with PTSD have an above-average capacity for mental imagery (Stutman & Bliss, 1985). Grigsby (1987) highlighted an imagery practice effect in PTSD treatment and opined that severe chronic stress converts average into superior imaginal ability. Later, Bryant and
Harvey (1995) reflected on such ability, arguing that the experience of traumatic imagery can increase capacity to experience non-traumatic imagery. Both they and Grisgby argued that this ability, which is ironically a key to the hallmark symptom of PTSD, may be able to be put to therapeutic use.

### 6.4.4.3 Anger in PTSD

As noted, research is beginning to emphasise anger’s impact on imagery. Given the prominence of dysfunctional anger in PTSD, and the latter’s intimate association with imagery, this impact is likely to be exacerbated in its presence.

To briefly reiterate argument from subsection 6.4.2 and Chapter Five, anger in PTSD has a clear effect on intrusions. This effect is interactive and reciprocal, with uncontrolled imagery leading to high arousal, anger having a primary impact on imagery and rumination mediating the relationship between anger and PTSD. This has been demonstrated in a longitudinal study of assault victims by Orth, Cahill, Foa and Maercker (2008). As argued in Chapter Five, this angry ruminative scrutiny is directed frequently by PTSD sufferers to the loss of control over the imagery process itself, not simply the content of the imaginal memory. As argued in Chapter Five, this angry ruminative scrutiny is directed frequently by PTSD sufferers to the loss of control over the imagery process itself, not simply the content of the imaginal memory.

Cognitive models of PTSD suggest that the frequency and distress of intrusive memories is magnified through ruminations about injustice and blame (Rusting & Nolen-Hoeksema, 1998). The five cognitive characteristics hypothesised in section 6.4.2 to connect anger to imagery - meaning-related appraisals, anger’s interconnectedness with other emotions, culpability, morality and wrong-doing and malevolence - typically take on even greater impact in PTSD.

The power of the link between anger in PTSD and imagery is in part due to the meaning of the event. To illustrate, most people can generate mental images and manipulate them and under normal conditions - where they are known to have an average duration of 250 milliseconds (Kosslyn, 1994). These images decay quickly, however and even with active maintenance people struggle to hold an image in short term memory for more than a few seconds (Pearson et al., 2013). The evidence suggests that only imagery meaning is remembered after a few days (see Baggett, 1975). The impact of meaning is amplified following the experience of extreme stress, where the considerable longevity of post-traumatic imagery is axiomatic clinically.
and empirically. Wilmer (1996), in a review of combat-related nightmares, observed that decades after WW II and the Vietnam War, a majority of combat veterans faithfully or symbolically continue to experience war-related nightmares and recent research suggests that severely traumatic autobiographical memories may have a lifespan in excess of 65 years (Rintamaki, Weaver, Erlbaum, Klama & Miskevics, 2009). Perceptually and imaginically, all such phenomena, while initiated by external stimuli, depend on the interpreted meaning of those stimuli.

In summary, the manifestation of imagery is exacerbated in the presence of disordered emotion, stress and, particularly, traumatic stress. Diminished control of imagery creates a circular problem of amplifying imagery characteristics, with anger in the presence of heightened imagery increasing detection of and scanning for, triggers and cues.

6.5 Conclusion

This chapter has described the various factors likely to affect imagery in anger in PTSD. It documents evidence emphasising the need to understand that the manifestation and management of imagery is subject to demographic, individual and imagery characteristics and that the impact of these factors is exacerbated where there is negative emotion, anger, stress and traumatic stress. Building on them and the preceding chapters, the following chapter presents a consolidated rationale for the empirical task of this thesis.
CHAPTER 7: PLAN OF THIS RESEARCH

Nothing in life is as important as you think it is, while you are thinking about it
Daniel Kahneman, Thinking, Fast and Slow

Based on the literature reviewed and theoretical arguments developed thusfar, this chapter articulates the rationale for this research’s investigative focus on the relationship of anger in PTSD and the role of imagery in that relationship. It initially outlines an integrated hypothetical model for explaining anger in PTSD that emphasises the cognitive and emotional nature and impact of imagery. It then justifies this model by summarising what is known about anger in PTSD from the existing PTSD, anger, anger in PTSD and imagery literatures. Subsequently, it identifies various research questions that bear upon this model, but as yet are un-examined. Finally, it describes the specific objectives of this research and the experimental predictions involved.

7.1 An Integrated Model to Account for Anger in PTSD

This research examines the utility of an explanatory model of anger in PTSD. That model identifies that anger makes direct and indirect contributions of anger to PTSD. It is, however, focused specifically on anger’s indirect contribution to PTSD via imagery and thought. Imagery’s influence is thought to be reflective of the operation of imagery process characteristics (frequency, vividness and absorption) and content (angry imagery). Thought is understood to independently contribute to anger’s relationship with PTSD and to mediate imagery’s impact on anger in PTSD. The model understands that imagery and thought have differing relationships with negative affect, as represented by anger, anxiety and depression. It predicts the roles of imagery and thought, as underlying mechanistic cognitive devices will be amplified in the presence of PTSD. These roles and their impacts are anticipated to be greatest where there is angry rumination of an autobiographical nature characterised by high-intensity, dysregulated imagery, negative-thought and even more prominent where such phenomena are automated or involuntarily manifested. A schematic representation of this model is depicted in Figure 7.1.
Figure 7.1  A preliminary model for understanding the role of imagery in anger in PTSD

7.2  Theoretical and Empirical Evidence Supporting this Model

7.2.1  PTSD-related evidence. As detailed in Chapter Two, PTSD symptoms, anger included, are likely to be associated with extreme stressors, but are far from inevitable. Individual pre-dispositional strengths and weaknesses, potentially pathogenic trauma-related factors and the context of the individual’s life are causally implicated in the aetiology of PTSD in a complex, multifactorial interaction.

The application of a stress-vulnerability paradigm (Meehl, 1962) to PTSD emphasises that subjective appraisal is essential for an event’s traumatic impact to be registered (McNally, 2004). Such appraisal is influenced by high levels of negative emotion and is a cognitive risk factor for PTSD due to its tendency to impair
information processing (Ehlers, 2010; Holmes & Bourne, 2008). Key cognitive processes involved are typically automated and include a negative attributional style, threat-oriented rumination and looming cognitive style. Together such cognitive processes facilitate the interpretation of PTSD symptoms as signifiers of impending catastrophe and impair current and future coping (Elwood et al., 2009).

Prominent explanatory theories of PTSD have also emphasised the role of emotion-overload. Importantly, however, it has been increasingly acknowledged in recent PTSD research and theorising that many individuals exposed to trauma, especially via vocation, do not typically respond with a fear response. Reflective of this, DSM 5’s PTSD stressor criterion eliminated the second DSM IV sub-criterion (A2) which required that an individual respond with fear, helplessness or horror to a traumatic event. It also amended DSM IV by accepting repeated or extreme indirect exposure to aversive event details as sufficient to meet the required stressor criterion. It explicitly illustrates the grounds for this inclusion by reference to the example of policing duties involving repeated exposure to childhood sexual abuse, where resultant emotional responses in responders are typically those of bewilderment, repulsion, disgust and, ultimately, anger.

These DSM 5 alterations were underpinned by several large-scale confirmatory factor analytic studies of PTSD over the last decade. They endorsed a four factor structure involving intrusion, effortful avoidance, negative alterations in cognition/mood and arousal. These developments have both coincided with and facilitated the recognition that PTSD may be characterised by a spectrum of affective presentations and that dominant emotions in PTSD include anger and anger-related feelings, like disgust, guilt and shame. Therefore, alongside the classic fear-based PTSD, various subtypes have either already been endorsed in the official nomenclature (e.g., the DSM 5 dissociative sub-type) or identified as important research targets (e.g., anhedonic and angry subtypes) by key PTSD theorists and researchers (see Friedman, 2011; Friedman & Resicke, 2014; Resick & Miller, 2009).

While these theories emphasise the importance of thought-based appraisals, it is apparent that imagery is critically involved in disordered emotion. Thus, intrusive imagery is widely recognised as the signature of PTSD. Theory has strongly influenced practice and lead to the development of one of the most important current conceptualisations of and treatment approaches to PTSD: emotion processing theory.
It has, in turn, been critically important in understanding and more effectively treating the disorder via prolonged (imaginal and in vivo) exposure (McNally, 2005).

Such imagery has long been recognised as having a retrospective character, as represented in memories of danger(s) or horror. Recently research has begun to emphasise that imagery may also have a powerful prospective role in the maintenance of PTSD - for example, as suggested by the Warning Signal Hypothesis (Ehlers et al., 2004; Ehlers, 2010) and the looming cognitive style identified by Elwood (2009) and associates.

Imagery is not a vulnerability for dysfunction per se, given its critical role across a wide range of adaptive human functions and status as a naturally inert, content-free perpetual mental tool. Rather, in the presence of dysregulated and high-intensity negative emotion, it becomes dysregulated and aversive and a cognitive variable central to the experience of PTSD and the emotion-overload (Foa et al., 1989) and attributional, ruminative, anxiety-sensitive and looming thinking styles proposed to account for PTSD variance (see Elwood et al., 2009).

Thusfar, the clinical importance of imagery has principally been established in relation to anxiety disorders and strong imagery connections have been made in research of anxiety disorders, especially, PTSD. Within PTSD, there is arguably a connection between imagery and anger that centres on the known or perceived intention of the event perpetrator and the event’s meaning and moral status. Disgust, guilt and shame, repulsion and other non-anxious affects are highly likely to be associated with such morality dimensions. Their downstream presence or absence in the face of horrific or morally-challenging traumata account for the fact that not all trauma-exposed individuals develop PTSD. As reported in previous chapters, the involvement of people in and their culpability for trauma is often causally linked to the emergence and maintenance of PTSD and anger, especially where there is apparent malevolence and immorality of behaviour. Imagery based recollections and prognostications of an intrusive and volitional nature are intimately involved in these powerful affective reactions to the experience of trauma.

7.2.2 Anger-related evidence. Anger is a cardinal human emotion that occurs semi-regularly for the vast majority of people without significant negative impact. In contrast, dysfunctional anger, as defined in Chapter Three (subsection 3.2.2), is uncommon and inherently pathological and over-represented in PTSD populations, where it affects a substantial proportion of those with the disorder.
Dysfunctional anger is multi-factorially determined. Influential aetiological and maintaining factors include maturational experiences, negative temperament, blaming causal appraisals and negative emotions other than anger, psychopathological conditions of both a functional and organic nature, problematic mental states and behavioural disinhibition associated with organic brain conditions (especially acquired brain injury) and intoxication.

It is also correlated with negative life events, especially where these are cumulative or traumatic in nature. In their presence, the anger response is strongly influenced by perceived provocation driven by the (in)actions of others. As detailed in Chapter Six, this perception is appraisal-based, which, in turn, reflects accumulated memory storage and retrieval via imagery, with all of imagery’s attendant biases. The result is the more an event is experienced as provoking and subject to blaming appraisals, the greater the perceptual and cognitive resources likely to be devoted to it and likelihood that imagery with a strong action potential will be manifested.

Multiple mechanisms that connect imagery to negative affect have clear relevance to anger. These include the direct effect of imagery on emotional systems in the brain, tendency for imagery to have a similar impact to that of real events and capacity of imagery to reactivate past feeling states (Holmes & Mathews, 2010). Imagery is central to cognitive processes - for example, via a stimulus salience effect that drives top-down information processing that then directs bottom-up information processing. Research shows that the operation of these processes reflects various facets of imagery (especially its control, absorption and vividness).

As noted in the earlier description of the model be tested in this research, anger has long been understood as a moral emotion and the development of dysfunctional anger is strongly correlated with appraisals regarding social norm-violation and enforcement, more particularly when there is morally questionable behaviour by self or others. In these circumstances, dysfunctional anger can be the product of the tendency to ruminate and the effect of over-generalised, autobiographical memory, manifested not only in thought, but also imagery. Indicative of this, a proclivity for imagery and an angry cognitive set - for example, characterised by event appraisals, attributions of causality, dysfunctional underlying beliefs and schemata and values supporting anger, aggression and violence as acceptable means for conflict resolution - is critical to the development and maintenance of dysfunctional anger.
There is also an indirect connection of imagery to anger through anger’s interconnectedness with other negative emotions that have a proven link with imagery, anxiety being the ultimate example (Hawkins & Cougle, 2011). Research has shown over time that anxiety and anger are innately linked and it is arguable imagery related to anxiety can affect anger. Not all anxiety disorders and anger are equally connected (Moscovitch et al., 2008). It is arguable that those anxiety disorders with a higher association with imagery (i.e., PTSD social anxiety disorder) are more likely to be connected to anger via imagery where the stimuli relate to humans (and not, for example, animal or inanimate fears like heights or thunderstorms).

7.2.3 Anger in PTSD-related evidence. Dysfunctional anger in PTSD is aversive, dysphoric, dimensional in intensity and provocation-related. It is recognised as associated with a multitude of mental contents, manifested intra and interpersonally and becomes automatic, chronic and subject to a loss of control on account of repetition. It is coloured by a preoccupation with the meaning of the initiating traumatic event, the intention of its perpetrators and the moral status of associated behaviours. It is characteristically focused on either those responsible for the traumatic loss or hurt or failing to prevent it. It is also preoccupied with those unable or unwilling to understand the nature of such loss and hurt and its ongoing maintenance. It may also be differentiated thematically from dysfunctional anger. These themes are typically focussed on the inculcation of values in significant others and their protection from future traumatic events. Associated with past hurt and loss, such themes can be focussed in an ongoing manner on events appraised as either warning signs for present and future loss and hurt or cues and triggers to memories of past loss.

Anger in PTSD is more likely to be associated with extreme stressors and there is clear evidence it is differentially associated with trauma type. Natural disasters that do not include a human causal component appear to be the least associated with anger, while human caused events and especially those which are morally culpable in nature and malfeasant, are associated with more severe anger.

The inability to control imagery is powerfully associated with PTSD severity. This is because disordered imagery leads to intrusions and high arousal. Intrusions and anger in combination have a greater effect on PTSD severity than either alone. In turn, rumination, in both its thought and imagery-based forms and anger are inextricably linked. The common connection is arousal and it is known that the PTSD
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hyperarousal symptom group, to which anger belongs, is the predominant predictor of overall PTSD severity.

Logically, the greater the number of risk factors present, the greater the likelihood of anger in PTSD. No single risk factor, however, is likely to result in anger in PTSD and not all individuals exposed to these risk factors develop dysfunctional anger. A stress-diathesis paradigm has strong utility in accounting for this apparent variation in that subjective perception and appraisal of anger-related stimuli influenced by a tendency to experience high levels of negative emotion renders some individuals susceptible to anger in PTSD. Imagery is implicit in these powerful affective outcomes. In summary, anger in PTSD that is ruminative in nature and has strong thought and imagery-based components is decay resistant and persists long after the occurrence of traumatic event.

7.2.4 Imagery-related evidence. As described across Chapters Four to Six, broad-ranging historical and contemporary evidence from the fields of neurology, perception, psychopathology and affect-theory indicates that imagery is an elemental human faculty intrinsic to intellectual, psychological, psychosocial and psychobiological functions. Imagery is thus recognised as central to perception, information processing, imaginative thought and memory.

Evolutionary and perceptual psychology researchers and theorists agree that imagery-based information processing developmentally precedes word-based cognition. Before humans use lexical devices, pictorial tools play a critical role in enabling communication. Imagery plays a clear part in psychosocial development and it has been well-documented since Piaget’s seminal work which showed that before infants develop expressive language, their earliest interpersonal interactions occur non-verbally (see, for example, Kosslyn, 2005a; Pratt, Cooper & Hackman, 2004). This commences with the recognition of caregiver faces and is followed by emotion identification and, finally, the enabling of emotional expression.

There is evidence that imagery plays a role in the maintenance of the human sense of self, preference for imaginal processes being taken to be a reflection of temperament, disposition and personality. At the level of psychological functioning, imagery is considered critical to psychological integrity and much literature attests to its importance in functionality and wellbeing in non-clinical populations.

Imagery is germane to the experience of emotion - especially in so-called primary affects like disgust, worry, fear, sadness, happiness and joy - and is more
powerful than thought in evoking memory of equivalent events which have emotional content. Given imagery’s central important to human functionality, even intrusive emotional imagery can be within the bounds of “ordinary” cognitive processing and not abnormal per se. It is known, nevertheless, that from an early age, some individuals experience emotions more intensely and have greater difficulty regulating them. Relevant to this, researchers have emphasised that there is an optimum level of imagery which, when exceeded, becomes associated with emotional dysregulation and many forms of psychopathology. A preponderance of sensory-imagery encoding of information is associated with persistent, disordered psychological processes, including distorted information processing and disordered, intrusive memories. Consequently, for anger to not involve imagery generally and more so where there is dysfunctional anger, like that in PTSD especially would be inexplicable.

7.2.5 Summary. Perceptual attention is drawn to anger-provoking stimuli. This is phylogenetically programmed. Faced with an information array potentially containing such stimuli, increased brain activation will occur in networks and systems associated with high-intensity negative emotions (especially anger) and imagery.

Humans are not inherently angry and under normal conditions anger responses will be dampened by higher cortical areas and top-down input processing. Due to chronicity of stimulation, however, biases can develop which lead to the instigation of imagery that is threat driven and of an intensity that is beyond the capacity of individuals to easily regulate. Under these conditions, intrusions occur which reflect imagery-driven perceptual recognition and memory retrieval processes which are autobiographical in nature, over-generalised and negatively-biased and which inevitably become the subject of rumination. Autobiographical memory, when unbidden and associated with negative affect, especially anger, has a greater impact than non-anger-associated autobiographical memory. All of this further serves to amplify the impact of intrusions, leads to more dysphoria, anger included and recursively exacerbates imagery (and automated thought) in an unintended practice effect.

The rumination involved in anger and angry imagery typically involves others and their behaviours. From visual imaging studies it is known to be easier to produce imaginal constructs when instructions are given to include others and even more so when significant others are invoked. This will lead to more rumination. This,
in turn, is enhanced by the salience or meaning of the event, with contravention of social norms and morally-questionable behaviour being especially provocative. This dysregulation of the image system in PTSD is a reflection of innate ability and imagery qualities. Thus, while normally a benign, content-free, tool, under conditions of extreme stress and scenarios involving moral questions, a proclivity for imagery production becomes a vulnerability for imagery dysregulation and, ultimately, dysfunctional anger. As identified in Chapters Five and Six, increased imagery proneness leads to increased impact of anger on PTSD on the basis that imagery acts as a mediator of anger, as it does in anxiety.

On the basis of the above research, due consideration was given to the directionality of the proposed model. It was decided that the best conceptualisation of the relationship of imagery to anger in PTSD for testing is one which understands dysregulated anger causally determines dysregulated imagery. This emphasises that is imagery and for that matter thought, which is increased in the context of psychopathology, rather than the reverse. Thus, the proposed model outlines a causal path from anger to imagery and thought that continues on to PTSD.

### 7.3 Key Research Questions

Notwithstanding the phenomenological, empirical and theoretical knowledge confirming and describing the relationship of anger to PTSD and the evidence presented for the role of imagery in that relationship, there are unanswered questions about anger and imagery in PTSD that call for investigation.

In the first instance, although anger is strongly associated with PTSD, factors influencing that relationship’s strength are not clearly understood. There are many potential factors of influence. While it is beyond the ambit of this research program to explore all such factors, obvious areas requiring investigation relate to the impact of the stressor and its nature, intensity and dose-rate, on anger in PTSD. A prime example of this is the need to establish why and how those exposed to cumulative occupational trauma (e.g., emergency services and military personnel), differ in their responses and in turn from the general community response to trauma. Correspondingly, the importance of individual risk-characteristics is as yet undetermined. To exemplify, while it is known that PTSD intensity in adults does not necessarily vary with age of onset, the experience of anger does vary across the lifespan. In this regard, it would valuable to know whether there is an interactive
effect, such that later-occurring PTSD is differentially associated with anger intensity and trajectory and, if so, on what bases this exists. Another area for investigation relates to how the impact of anger on PTSD compares to other negative affects (especially anxiety and depression) and whether they have mediational or moderational effects. Another knowledge gap the research literature has yet to comprehensively address relates to whether the association of anger and PTSD is causal in nature and, if so, what direction that cause takes.

Similarly, despite the central association of imagery with PTSD, there are unanswered questions relating to the role and impact of imagery in anger in PTSD. The most obvious relates to the extent to which imagery associated with anger affects PTSD as compared to anger’s direct relationship with PTSD.

Another question relates to which imagery characteristics are involved and the relative strength of any such association with anger. Research on imagery in PTSD and in general has tended to focus on the role of vividness. This is not surprising, given the common use of long-developed and psychometrically sound measures of vividness (see Chapters Six and Eight). Other imagery characteristics deserving of attention, but as yet not as well-examined as vividness, include control and absorption ornowness.

A less obvious, but important question, pertains to how the strength of imagery’s relationship with anger in PTSD compares to that of other affects (especially anxiety and depression). Answering this question has the potential to lead to a more integrated and through understanding of how negative emotion affects PTSD through imagery. Dismantling the role of negative affect and imagery also has high potential value in the treatment of and anger in PTSD and PTSD itself.

Another question relates to the precise role of negative thought in PTSD and its relationship with imagery. The influence of dysregulated imagery in PTSD is axiomatic, but typically accompanied by impactful maladaptive thought. Cognitive restructuring treatments for PTSD have been shown to be clinically effective (see Mueser et al., 2009), yet various consensus views and guidelines, which summarise the now considerable body of treatment evidence, emphasise that cognitive therapy typically does not add to the overall impact of prolonged exposure (Foa et al., 2005). This is clinically counter-intuitive and antithetical to the many critically important descriptive treatment frameworks and models which advocate for a staged approach to interventions (e.g., those by Briere, 1996; Cloitre, Cohen & Koenen, 2006; Keane,
There are attempts to integrate exposure-focused and cognitive treatment approaches in programs and types of treatment - see, for example, the work of Briere, 1996) and Cloitre and colleagues (2006) - and to better understand the imagery and thought inter-relationship is important. This will especially important in angry posttraumatic rumination, a place in PTSD where imagery and thought meet.

7.4 Key Aims, Objectives and Predictions of This Research Program

This research program sought to examine the influence of imagery in anger in PTSD across a range of populations. It did so via three studies which examined the relationship of anger to PTSD, the role of cognition (imagery and thought) in anger and negative affect and in anger in PTSD relate to other negative affects. The following sets of aims, objectives and hypotheses were pursued.

7.4.1 Aims, objectives and predictions pertaining to anger in PTSD (studies one, two and three). The overall aim of this element of this research program was to assess the strength of the relationship between anger and PTSD. Four specific objectives were pursued. The first was to examine the specific relationship of PTSD to anger phenomena. The second was to explore whether this relationship could be clarified by contrasting it with those pertaining to PTSD to anxiety and depression. The third investigated the impact of change in anger on PTSD and the final objective sought to assess the directionality of this change relationship.

Flowing from these objectives and based on the prevailing research literature, there were four experimental hypotheses. Initially, it was predicted that a positive relationship would exist between anger and PTSD (hypothesis 7.4.1.1). Next, it was expected that this relationship would be as great as, or exceed, the strength of those pertaining to anxiety and depression and PTSD (hypothesis 7.4.1.2). Following on from this, it was hypothesised that with treatment, changes in anger would lead to changes in PTSD (hypothesis 7.4.1.3). Finally, across the three populations involved in the studies of this research program, it was expected that there would be differences in negative affect due to psychopathology. Thus, it was expected that anger scores would be elevated in PTSD populations, correlated strongly with PTSD, albeit subject to the effect of occupational group and trauma type and would inversely reflect the effect of age (hypothesis 7.4.1.4).

These objectives and hypotheses operate as hurdle requirements for the studies that investigated the relationship of imagery to anger and negative affect (as
measured by anxiety and depression) and such affects in the presence of PTSD. They are understood as a critically important means for “proof of concept” testing of the proposition that anger is strongly related to PTSD of itself and in comparison to other major negative effects.

From this platform, it will be possible to explore the remaining objectives and hypotheses regarding the role of imagery. These objectives and hypotheses pertain to the relationship of imagery to anger and other negative affects (as measured by anxiety and depression) and such affects in the presence of PTSD and are described in the following two subjections of this chapter.

7.4.2 Aims, objectives and predictions regarding imagery in negative affect, anger and anger in PTSD (studies two and three). The overall aim of this second element of this research program, was to explore the role of imagery in negative affect generally and within the context of PTSD. Three objectives were addressed. The first focused on the strength of the relationship between anger and imagery in general. As part of this objective, that relationship was contrasted with those pertaining to the negative affects of anxiety and depression. The second objective was focused on the relationship of anger and imagery in the context of PTSD. That relationship was contrasted with those pertaining to imagery and anxiety and depression. The role of visual imagery in determining anger-related PTSD patterns and the directionality of these relationships was also explored. Consequent of these first two objectives, this research sought to compare such relationships across general and PTSD populations.

Emanating from these objectives and based on the prevailing research literature, there were four hypotheses. Initially, it was hypothesised that a positive relationship would exist between imagery and anger generally and in the presence of PTSD (hypothesis 7.4.2.1). Next, it was predicted that this relationship would be comparable in strength to those pertaining to anxiety and depression, but that different facets of imagery would be associated with different affects (hypothesis 7.4.2.2). Thereafter, was expected that changes in anger in PTSD and differences in the expression of anger, anxiety and depression would be identifiable on the basis of imagery (hypothesis 7.4.2.3). Finally it was hypothesised that while such relationships would hold in both general and PTSD populations, they would be of a greater intensity in the latter population (hypothesis 7.4.2.4).
7.4.3 Aims, objectives and predictions relating to the interaction of imagery and thought in negative affect, anger and anger in PTSD (studies two and three). The overall aim of this final element of this research program, was to compare the role of thought in negative affect generally and within the context of PTSD. Three specific objectives were addressed. The first focused on the strength of the relationship between anger and thought in a general population. As part of this objective, that relationship was contrasted with those pertaining to the negative affects of anxiety and depression. The second objective contrasted the impact of imagery on anger with that of negative thought. As part of this objective, the inter-relationship of such imagery and thought impacts was investigated. This impact was examined generally and in the context of PTSD.

Arising from these objectives and based on the prevailing research literature, there were four experimental hypotheses. Initially, it was predicted that a positive relationship would exist between negative thought and anger in PTSD (hypothesis 7.4.3.1). Allied to this, it was hypothesised that this relationship would be comparable in strength to those with anxiety and depression and that this would apply in both general and PTSD populations (hypothesis 7.4.3.2). It was additionally hypothesised that the inter-relationship of imagery and thought in such relationships would be differentiable on an affect-by-affect basis (hypothesis 7.4.3.3). Finally, it was hypothesised that while such relationships would hold in general and PTSD populations, they would be elevated in PTSD populations, correlated more strongly with PTSD where there was moral injury and inversely reflect the effect of age (hypothesis 7.4.3.4).

7.5 Conclusion

By testing the above model of anger in PTSD and the associated hypotheses, this research sought to illuminate the role of visual imagery as a mechanistic device underlying anger and anger in PTSD. It was anticipated that by adopting the approach outlined, it would be able to explicate whether imagery functions as a thought-independent or related pathway to anger in PTSD. The three studies which sequentially test these propositions and build toward a consolidated understanding are described in the following three chapters.

Multiple mechanisms proposed to connect imagery to negative affect have clear relevance to anger. These include the direct effect of imagery on emotional
systems in the brain, tendency for imagery to have a similar impact to that of real events and capacity of imagery to reactivate past feeling states.

Imagery functions as overarching top-down cognitive processes that strongly influences top-down information which strongly influences bottom-up processing and reflects the individual’s experience of several facets of imagery (i.e., its control, absorption, vividness and anger content). There is also an indirect effect of imagery on anger via anger’s interconnectedness with other negative emotions which have a proven link with imagery, like anxiety.

Enduring dysfunctional anger in the context of PTSD is often the product of the tendency to ruminate via over-generalised, autobiographical memory, manifested in not only thought, but also imagery. Indicative of this, a proclivity for imagery and an angry cognitive set is critical to the development and maintenance of dysfunctional anger. Anger has long been understood as a moral emotion and the development of dysfunctional anger is strongly correlated with appraisals regarding social norm-violation and enforcement, more particularly when there is morally questionable behaviour by self or others.
CHAPTER 8: STUDY 1: EXAMINATION OF THE PREVALENCE, VARIABILITY AND EFFECT OF ANGER ON PTSD

When you are offended at any man's fault, turn to yourself and study your own failings. Then you will forget your anger.

Epictetus

The overall research project, in which this study was embedded, was conducted over seven years from 2008 to 2015 and involved three separate, yet theoretically and empirically inter-related, studies. This chapter, and the two which follow, sequentially describe those studies. Each chapter reports the respective study research design, participants involved, procedures and measures used to test experimental predictions, approach to data analysis, results, and, thereafter, a brief discussion of the meaning of those results.

8.1 Research Design

This study investigates the relationship between anger and PTSD by examining cross-sectional pre and post-treatment data. It also explores the stability of that relationship over time. These data are contrasted with relationships pertaining to anxiety and depression, as these two negative affects are well-established in the theoretical and empirical literature as strongly associated with PTSD.

To facilitate these objectives, this study retrospectively analysed data gathered from veterans who received psychiatric and/or psychological treatment for trauma-related mental health conditions at the Psychological Trauma Treatment Service (PTRS) of Austin Health in programmatic group and individual PTSD treatment. These data were collected at the time of entry into treatment (pre-treatment) and again 12 months later (post-treatment).

The PTRS is a specialised service of Austin Health for the assessment and treatment of individuals with trauma-related mental health conditions. Austin Health is a major federal and state government funded tertiary-level teaching hospital incorporating a broad range of inpatient and outpatient service facilities.

This study was approved by the Austin Health Human Research and Ethics Committee (AHHREC). All participants had previously provided informed consent.
when completing the metrics involved as part of the process of entering into treatment. The Patient Informed Consent Forms approved for use by the AHHREC are attached at Appendix D. No monetary payment was made to any individual for participation in this study.

**Figure 8.1** Participant flow summary

### 8.2 Research Method

#### 8.2.1 Participants. The participants in this study were former serving (veteran) Australian Defence Force (ADF) personnel. They were drawn from the three branches of the ADF; that is, the Royal Australian Airforce (RAAF), Royal Australian Navy (RAN) and Australian Army (Army). They comprised a total of 357 Vietnam veterans who were Regular (vocational) RAAF, RAN and Army personnel or National Service (conscripted) Army Members. They were drawn from over 846 consecutively admitted veteran participants who had completed the Austin Health PTSD Treatment program between its inception in 1995 and the data-inspection point of November 2008 (see figure 8.1). A total of 499 participants were excluded due to missing data on one or more of the study measures at time one or time two. The majority of these related to time two data collection. At both time points, the most
common reasons for the non return of data included choosing to decline the invitation to provide data, being lost to follow up, the purported failure of the mail service and apparent accidental failure to complete all aspects of the assessment battery. Thereafter, four participants were excluded due to their status as multivariate and univariate outliers. The adjusted sample, after correction for these incomplete data sets and outliers comprised 353 cases, with 256 of these cases providing all required data at time one and two.

8.2.2 Measures

8.2.2.1 Demographic and Service Data. Demographic information was gathered via simple survey. It consisted of age, gender, education, employment and relationship status data, along with information about service length and the military branch in which the participants served the majority of their service.

Participant demography is detailed in Appendix E. It shows that at the time of pre-treatment test completion, participants were aged from 43 to 75 years, with a mean age of 53.73 (SD = 4.51). All were male. Most (55%) had been educated to late high school, with 39 per cent attaining a trade or industry certificate, and six per cent obtaining a university degree or higher. Twenty two per cent were employed. Sixty-five per cent were in receipt of a DVA pension for a medical or psychiatric condition and thirty-five per cent were applying for a DVA pension at the time of entry into treatment. Seventy-nine per cent were married and only four per cent had never been in a marriage-like relationship. All served in Vietnam (56% as conscripted National Servicemen and 44% as regular ADF personnel) and the vast majority were past serving members of the Army (94%), with only one per cent from the RAAF and five per cent from the RAN. Co-morbidity in the sample (as rated by the assessing PTRS psychiatrist, psychiatry registrar or clinical psychologist) was common. Where data was available (in approximately 50% of cases), diagnosis of alcohol-substance abuse/dependence (43%), depression (42%) and another anxiety disorder (15%) were the most common diagnoses.

8.2.2.2 Clinical Data. The following measures of anxiety, depression, anger, PTSD and substance abuse were used to obtain clinical data in this study (see Appendix F for copies of all instruments used in this study). They were adopted for a range of reasons. Principally, it was because they comprise a subset of the battery of psychometrics approved for use as outcome measures by Phoenix Australia for accredited Australian veteran PTSD treatment programs. (Phoenix Australia, formerly
the Australian Centre for Posttraumatic Mental Health, accredits veteran PTSD treatment programs on behalf of the Department of Veterans’ Affairs). Second, their use permits comparison of PTSD treatment data derived from Australian populations with data from similar international studies. Most notably these are from the US, where there is a longstanding and substantial amount of work published in the international literature on their use with military populations, including PTSD treatment programs (see Fontana & Rosenheck, 1997; Johnson et al., 1996; Elhai et al., 2003). Importantly, in this regard, US and Australian veterans have been described as having strikingly similar profiles (Elhai et al., 2003). Third, each is empirically-supported measurement instruments of PTSD and its comorbidities and possesses strong psychometric properties.

**Depression, Anxiety and Anger**

The Hospital Anxiety and Depression Scale (HADS; Zigmond et al., 1983) was used to measure anxiety and depression. The HADS is a 14 item, self-report measure for detecting states of anxiety (HADS-a, 7 items) and depression (HADS-d, 7 items). Each item response is scored from ‘0’ to ‘3’ with scores summed to form a total score for each sub-scale with values ranging from 0 to 21. Scores of 11 or more on either subscale are considered to indicate a significant ‘case’ of psychological morbidity, while scores of 8-10 represents 'borderline' and 0-7 'normal' cases. The internal consistency of the HADS is acceptable [Cronbach's alpha = 0.83 for the anxiety (HADS-a) and 0.82 for the depression (HADS-d) subscales] (Bjellard, Dahl, Haug & Neckelmann, 2002). In this study, the HADS-a and HADS-d respectively attained internal consistencies as measured by Cronbach’s alpha (coefficients) of 0.76 and 0.71.

The Dimensions of Anger Reactions Scale (DAR scale; Novaco, 1975) was used to assess dispositional anger directed toward others. The DAR scale comprises seven items rated on an eight point Likert scale from 0 (“Not at all”) to 7 (“Exactly so”), with higher scores indicating greater anger proneness. Possible scores range from 0 to 49. The DAR scale possesses good internal consistency (α = 0.89) and reflects a single factor (Novaco, Swanson, Gonzalez, Gahm & Reger, 2012), as well as being a reliable and sensitive measure of anger (d = .54; p < .001) (Forbes et al., 2004). In this study, the DAR Scale attained a Cronbach’s alpha of 0.92.
PTSD and combat exposure

The PTSD Checklist “military” version (PCL-M: Weathers, Litz, Herman, Huska & Keane, 1993) was used to measure PTSD. The PCL-M is a 17-item self-report scale for assessing PTSD symptoms as defined by DSM IV criteria. It has been used in a large range of national and international studies examining PTSD in military and veteran populations. In completing the PCL-M, respondents rate each item from 1 ("not at all") to 5 ("extremely") to indicate the degree to which they have been affected by that particular symptom over the past month. Thus, total possible scores range from 17 to 85. The PCL-M has demonstrated high levels of diagnostic accuracy (using a cut off of 50) when evaluated against “gold standard” structured interview measures like the PTSD component of the Structured Clinical Interview for DSM IV (SCID: Spitzer, Williams, Gibbon & First, 1995) and the Clinician Administered PTSD Rating Scale (CAPS: Blake et al., 1990), at a single time point (Blanchard, Jones, Alexander, Buckley & Forneris, 1996) and over the course of treatment and follow up (Forbes, Creamer & Biddle, 2001). The PCL-M has strong test-retest reliability (.96) and high levels of internal consistency (.97). In this study it attained a Cronbach’s alpha level of 0.85.

The PCL is, however, not a diagnostic measure. Because a diagnosis of PTSD is a condition for entry into the PTRS PTSD Treatment Program (and indeed all such Australian DVA-funded PTSD Treatment Programs), PTSD diagnosis was assessed and confirmed by use of the CAPS for DSM IV. In completing the CAPS, the clinician makes a rating for both the frequency and intensity of each of the 17 symptoms of PTSD. Blake and colleagues (1990) recommended that a frequency score of 1 (scale 0 = “none of the time” to 4 = “most or all of the time”) and an intensity score of 2 (scale 0 = “none” to 4 = “extreme”) was required for a particular symptom to meet criterion. A diagnosis is made by determining the requisite symptoms meeting criterion for a DSM diagnosis. A severity score for each symptom can also be calculated by summing the frequency and intensity scores. Thus, the total range of the instrument is 0-136. The CAPS is known to have strong reliability and validity (Weathers, Ruscio & Keane, 1999). Among pre DSM 5 interviews for PTSD, of which the most commonly used are the CAPS, the PTSD Symptom Scale (Foa et al., 1993) and the Structured Interview for PTSD (Davidson, Malik & Travers, 1997) - the CAPS is considered the gold standard diagnostic tool for assessing the presence
of PTSD. In this study, the CAPS obtained an internal consistency, as measured by Cronbach’s alpha coefficient, of 0.93.

Participant level of combat exposure was rated using the Combat Exposure Scale (CES; Keane et al., 1989). The CES is a widely used measure of combat exposure among veterans. It is a seven item measure and provides a total score (range 5-35) which may be broken down into various categories ranging from light to heavy combat. The measure has demonstrated an internal consistency alpha coefficient of .95 and test-retest reliability of .97. In this study, the CES attained an internal consistency as measured by Cronbach’s alpha (coefficient) of 0.82.

Alcohol Misuse

The Alcohol Use Disorders Identification Test (AUDIT; Babor et al., 1989) was used to assess alcohol misuse. The AUDIT is a self-report measure for assessing alcohol abuse/dependence. Scores range from zero to 40. A score of eight or more suggests clinically significant problems with alcohol consumption. High scores on questions one to three suggest hazardous alcohol use. High scores on questions four to six suggest the presence or emergence of alcohol dependence. High scores on questions seven to 10 suggest harmful alcohol use. The AUDIT was developed by the World Health Organisation as a screening instrument for hazardous and harmful alcohol consumption. It has strong test-retest reliability and high levels of internal consistency (.85 to .89) (O’Hare, Sherrer, La Buult & Emrick, 2004). In this study, it attained a Cronbach’s alpha level of 0.94.

8.2.3 Procedure. Data for this study was collected from previous participants of the Austin Health PTSD Treatment Program (the Program) for current-serving military personnel and veterans. Candidates for the Program are referred from various sources. These include general practitioners, private psychiatrists, psychologists, the Vietnam Veterans and Families Counselling Service [a community-based counselling service for veterans and current serving Australian Defence Force personnel funded by Australia’s Department of Veterans Affairs], inpatient and or outpatient services within Austin Health (both psychiatric and general medical) and self-initiated referrals.

Upon referral to the Austin Health PTSD Treatment Program, veteran and military candidates are triaged and then assessed by a member of the Psychological Trauma Recovery Service Assessment and Treatment Planning Team. When deemed appropriate, they are referred to the PTSD team. This assessment aims to determine
the veterans’ PTSD status, nature of comorbidities, physical health, substance use profile, marital and family situation, vocational and social status and eligibility for entry into the treatment program.

The Program is cognitive-behavioural in orientation. Key components include psycho-education modules, trauma-focused sessions which address common trauma-related themes (rather than group exposure), arousal management (including anger and anxiety management), depression management and alcohol and substance management modules. Details of the nature of the Program have been published by Creamer, Morris, Biddle and Elliot (1999).

Eligibility criteria for entry the program included a diagnosis of PTSD, being detoxified from the acute effects of alcohol and other drugs of addiction, stability of recent treatment (represented by the absence of any major changes in psychotherapy or medication in the few weeks prior to the program), an absence of acute suicidal or violent ideation or current psychosis, a cognitive capacity adequate for learning in the program and an absence of major or current life crises.

As outlined earlier in the measures section of this chapter, pre-treatment, PTSD diagnoses are confirmed using the Clinician Administered Scale for PTSD (CAPS; Blake et al., 1990) administered predominantly by psychiatrists and clinical psychologists. All clinical staff administering the CAPS are trained in its administration. The assessment also includes the gathering of key demographic information like marital, employment and compensation seeking status as well as military history details including their units and dates of military service.

Once accepted for the Program, participants are invited to attend an orientation session where they meet with the staff of the treatment program and are familiarised with the treatment setting. Where this orientation does not occur (for logistical reasons it can be difficult to assemble cohorts), participants are seen individually by a member of the treatment team before the first day of the Program.

The Program is 10 or 12 weeks duration, depending on the logistics of the time of year and the need to account for treatment-continuity-interfering factors such as government gazetted public holidays, school holidays and the Easter and Christmas vacation periods. The Program consists of a six week intensive phase of three days per week phase followed by a graduated step-down phase of one week of two days, followed by four weeks of one day per week. It also includes a partners component, usually consisting of a day-long sessions for each of the weeks of the Program.
Weekly individual psychology treatment is also provided to participants over the course of the Program. These individual treatment sessions primarily focus on trauma-focused work (prolonged exposure), but provide a significant ongoing opportunity to address any other key psychosocial problems (including other psychiatric problems and commonly familial or vocational problems).

In most instances data collection occurred before, at the commencement, or early in treatment. It was considered important to administer the measures once participants had already been accepted into the treatment program for two reasons. First, it was anticipated that this would minimise the potential for exaggerated or invalid response sets motivated either as pleas for inclusion into the program or where the participants may have misconceptions that acceptance into the program had implications for compensation processes. Second, it separated the completion of the study measures from completion of the minimum outcome data set to minimise the assessment burden on the participants at any one time point and therefore minimise both potential effects of fatigue or frustration on endorsement patterns.

Re-administration of the study metrics (time two) occurred twelve months post the completion of the initial administration of this battery of outcomes measures. For a minority of participants, for various reasons (e.g., reported failure of the postal service or unavailability due to absence), this did not occur then and the two-time point data collection occurred more than 12 twelve months apart. All measures in this study were completed by manually (i.e., using “pencil and paper tests”).

8.3 Preliminary Data analysis

Data in this, and all subsequent studies, was analysed by use of the Statistical Package for the Social Sciences (SPSS) version 21.0. Prior to analysis, all scale scores were checked for missing data, normality of distribution and outliers. There was a small amount of missing data in the Phoenix Australia data subset, due to participant failure to complete each item of every questionnaire included in the measurement battery. Consistent with the recommendations of Elliot and Hawthorne (2005) and Hawthorne and Elliot (2005), several approaches were used to address various data-gaps. For cases with single or several missing items of data, the closest match method (i.e., the insertion of cases missing timepoint data with values from the participant in the dataset who has the closest scores on same variables) was used. For cases where at
least half of the items of a scale were unanswered, mean substitution (i.e., replacing all missing data in a variable by the mean of that variable) was employed.

Tests of normality were subsequently performed on all scales included in the study. These tests indicated almost all scale scores were within acceptable limits of skewness and kurtosis. The sole exception was the age of the sample, which, while not skewed, was leptokurtotic in distribution, reflecting the fact that the sample was comprised of Vietnam veterans.

A variety of statistical techniques were used to test the study hypotheses. Cohen’s $d$ was used to establish effect size differences between pre-treatment and post-treatment measures, while Cronbach’s alpha was calculated for all pre-treatment scale scores to establish their internal consistency. Correlation analysis was used to compare the strength of relationship between PTSD and anger compared to other comorbid mood states and conditions that typically accompanying the conditions (i.e., anxiety, depression and alcohol abuse). A series of multiple regression analyses was then performed to further explore the relationships between the study’s variables. To eliminate the possible confounding of outcomes by the retention of anger probes in both independent and dependent variables, the PTSD Total and Arousal Subscale scores were adjusted by elimination of its anger item (item 14) in all analyses in which anger was the independent variable.

### 8.4 Results

#### 8.4.1 Treatment commencement (time one) data

Table 8.1 reports the mean scores, standard deviations, and significance and alpha levels for all scales for the population sub-sample of this study. The profile of scores was comparable with that previously reported for similar Vietnam veteran samples. For example, Creamer, Elliott, Forbes, Biddle and Hawthorne (2006) reported mean scores of 14.8 ($SD = 3.5$) and 11.9 ($SD = 4.0$) respectively for HADS-a and HADS-d scores, and Forbes and colleagues (2004) reported a mean of 32.7 ($SD = 12.2$) for Vietnam Veterans on the DAR scale.

The mean CAPS severity score at the time of intake was 76.7 ($SD = 18.3$). This reflects moderate-to-severe PTSD and is consistent with the mean score of 77.7 ($SD = 16.8$) applicable to participants of Australian PTSD treatment programs (Elhai et al., 2003). The mean PCL-M score similarly approximated the score of 67.3 ($SD = 9.7$) reported for Vietnam veterans by Creamer and colleagues (2006).
Table 8.1

*Study scale and subscale means and standard deviations at pre- and post-treatment with tests of reliability (Cronbach’s alpha) and effect size (Cohen’s d) (n = 353)*

<table>
<thead>
<tr>
<th>Variable</th>
<th>Pre-treatment</th>
<th>Post-treatment</th>
<th>t</th>
<th>d</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>M</td>
<td>SD</td>
<td>M</td>
<td>SD</td>
<td></td>
</tr>
<tr>
<td>HADS-a (Anxiety)</td>
<td>14.8</td>
<td>3.3</td>
<td>.76</td>
<td>12.7</td>
<td>3.6</td>
</tr>
<tr>
<td>HADS-d (Depression)</td>
<td>11.8</td>
<td>3.9</td>
<td>.71</td>
<td>10.4</td>
<td>4.1</td>
</tr>
<tr>
<td>DAR Scale (Anger)</td>
<td>29.4</td>
<td>14.4</td>
<td>.92</td>
<td>26.1</td>
<td>14.5</td>
</tr>
<tr>
<td>Audit (alcohol)</td>
<td>12.6</td>
<td>9.9</td>
<td>.94</td>
<td>11.2</td>
<td>9.0</td>
</tr>
<tr>
<td>PCL-M (PTSD) Total</td>
<td>65.3</td>
<td>9.1</td>
<td>.85</td>
<td>58.1</td>
<td>11.2</td>
</tr>
<tr>
<td>PCL-M (Intrusions)</td>
<td>17.5</td>
<td>4.2</td>
<td>.86</td>
<td>15.2</td>
<td>4.3</td>
</tr>
<tr>
<td>PCL-M (Avoidance)</td>
<td>26.8</td>
<td>4.1</td>
<td>.66</td>
<td>24.1</td>
<td>5.2</td>
</tr>
<tr>
<td>PCL-M (Arousal)</td>
<td>20.9</td>
<td>3.0</td>
<td>.70</td>
<td>18.9</td>
<td>3.6</td>
</tr>
</tbody>
</table>

The mean participant CES score was 19.25 \( (SD = 8.08) \), reflecting moderate combat exposure. This is consistent with previously reported scores for similar populations [e.g., the mean of 19.27 \( (SD = 10.05) \) reported by Creamer et al., 1996].

Table 8.2 reports Pearson product-moment correlation coefficients for age, alcohol consumption, depression, anxiety and anger with PTSD and its subscales at the time of entry into treatment. It indicates that associations with PTSD were generally of moderate strength. Exceptions included the higher correlation between anxiety and PTSD total score and the lesser correlations pertaining to depression and anger scores with PTSD intrusion scores. Negligible relationships existed between age and alcohol consumption pre-treatment and PTSD \( (r = -.11 \text{ and } .01 \text{ respectively, } p = ns) \) and on this basis both were eliminated from all subsequent analyses.
Table 8.2

*Relationships of age, negative affect, alcohol abuse and PTSD at pre-treatment (Pearson’s r) (n = 353)*

<table>
<thead>
<tr>
<th></th>
<th>Hads-d</th>
<th>DAR</th>
<th>Alcohol</th>
<th>PCL-Tot</th>
<th>PCL-Int</th>
<th>PCL-Av</th>
<th>PCL-Ar</th>
</tr>
</thead>
<tbody>
<tr>
<td>HADS-a (Anxiety)</td>
<td>.59**</td>
<td>.46**</td>
<td>.06</td>
<td>.65**</td>
<td>.48**</td>
<td>.52**</td>
<td>.60**</td>
</tr>
<tr>
<td>HADS-d (Depression)</td>
<td>.34**</td>
<td>.00</td>
<td>.54**</td>
<td>.30**</td>
<td>.58**</td>
<td>.43**</td>
<td></td>
</tr>
<tr>
<td>DAR Scale (Anger)</td>
<td>.15**</td>
<td>.46**</td>
<td>.35**</td>
<td>.41**</td>
<td>.39**</td>
<td></td>
<td></td>
</tr>
<tr>
<td>AUDIT (Alcohol)</td>
<td>.04</td>
<td>.04</td>
<td>.01</td>
<td>.05</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PCL-Tot (PTSD Total)</td>
<td>.82**</td>
<td>.88**</td>
<td>.82**</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PCL Int (Intrusion)</td>
<td></td>
<td>.53**</td>
<td>.53**</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PCL-Av (Avoidance)</td>
<td></td>
<td></td>
<td></td>
<td>.61**</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* p < .05, ** p < .01

Such data indicate pre-treatment, anger was significantly related to PTSD overall (PCL-Tot) [r (353) = .46], albeit to a lesser extent than anxiety (Hads-a) and depression (Hads-d) [r = .65 versus .54]. In terms of PTSD sub-clusters, anxiety was most highly related to arousal (PCL-Ar), while depression and anger were most related to avoidance (PCL-Av). All relationships were statistically significant (p < .01).

Table 8.3 indicates that, in the three stepwise regression equations relating to PTSD Total and the Intrusion and Arousal sub-clusters, anxiety was the first predictor entered (as having the greatest unique contribution). Anger and depression, as the next predictors entered, added marginally to the amount of variance explained for PTSD Total Scores (4 & 3% respectively). For Intrusion and Arousal sub-cluster scores, only anger, albeit again marginally, added to the amount of variance explained (2 & 4% respectively). For Avoidance sub-cluster scores, depression was the first predictor entered, followed by anxiety, then anger. Both anxiety (8%) and anger (2%) made minor contributions to the explained variance.
These results provided support for the further exploration of the experimental predictions regarding the interaction of anger, anxiety and depression with PTSD. Accordingly, stepwise multiple regression analyses were run with anxiety, depression and anger as independent variables, and PTSD and its (DSM IV) sub-clusters as dependent variables. The results of these analyses show all three affects were significant predictors for PTSD Total ($r^2 = .50, F(3,350) = 116.61, p < .001$), Avoidance cluster ($r^2 = .43, F(3,348) = 87.56, p < .001$) and Arousal cluster scores ($r^2 = .43, (3,350) = 90.33, p < .01$), while only anxiety and anger were significant predictors for Intrusion cluster scores ($r^2 = .25, F(3,350) = 59.08, p < .001$).

**8.4.2 Post-treatment (time two) data.** Post-treatment, mean scale scores for all independent and dependent variables decreased relative to pre-treatment scores (see Figure 8.2). These changes, as well as the means and effect sizes shown in Table 8.1, demonstrate that clinically mild-to-moderate changes in scores applied to most variables. The greatest changes were those for Total PTSD ($d = .65$) and anxiety scores ($d = .57$). Anger score change ($d = .48$) was clinically meaningful, but not as
strong. The least change occurred in scores for depression ($d = .34$) and alcohol abuse ($d = .16$).

![Figure 8.2 Change in mean scores for all variables pre and post-treatment](image)

Table 8.4 reports Pearson product-moment correlation coefficients for post-treatment depression, anxiety, and anger with PTSD measures post treatment (that is, 12 months post commencement of treatment). It indicates that, in each instance, strength of associations were consolidated from pre-treatment without any change in the order of the magnitude of correlations with PTSD overall or its sub-clusters. Thus, anxiety retained the greatest association with PTSD overall, followed by depression and then anger. With regard to PTSD’s sub-clusters, anger was most related to arousal and, surprisingly, least to PTSD intrusions. Anxiety was most related to arousal, while
depression was mostly strongly related to avoidance. Anger’s relationships with the dependent variables tended to strengthen most from pre-treatment to post-treatment.

Table 8.4

*Relationships of negative affects, alcohol abuse and PTSD at post-treatment (Pearson’s r) post treatment.*

<table>
<thead>
<tr>
<th></th>
<th>Hads-d</th>
<th>DAR</th>
<th>PCL-Tot</th>
<th>PCL-Int</th>
<th>PCL-Av</th>
<th>PCL-Ar</th>
</tr>
</thead>
<tbody>
<tr>
<td>HADS-a (Anxiety)</td>
<td>.64**</td>
<td>.54**</td>
<td>.68**</td>
<td>.52**</td>
<td>.60**</td>
<td>.61**</td>
</tr>
<tr>
<td>HADS-d (Depression)</td>
<td>.48**</td>
<td>.63**</td>
<td>.43**</td>
<td>.63**</td>
<td>.53**</td>
<td></td>
</tr>
<tr>
<td>DAR Scale (Anger)</td>
<td>.56**</td>
<td>.44**</td>
<td>.46**</td>
<td>.54**</td>
<td></td>
<td></td>
</tr>
<tr>
<td>PCL-Tot (PTSD Total)</td>
<td></td>
<td>.81**</td>
<td>.88**</td>
<td>.85**</td>
<td></td>
<td></td>
</tr>
<tr>
<td>PCL Int (Intrusion)</td>
<td></td>
<td></td>
<td>.53**</td>
<td>.58**</td>
<td></td>
<td></td>
</tr>
<tr>
<td>PCL-Av (Avoidance)</td>
<td></td>
<td></td>
<td></td>
<td>.65**</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* p < .05, ** p < .01

These results provided support for the further exploration of the experimental predictions regarding the interaction of anger, anxiety and depression with PTSD. Accordingly, multiple regression analyses were run utilising negative affect (depression, anxiety and anger) and PTSD total score at pre-treatment as independent variables, and post treatment PTSD symptoms as the dependent variable. The pre-treatment PCL score was included on the basis of the robust research finding that PTSD severity is the greatest predictor of PTSD outcome post-treatment. Given this and the tendency for anger correlations tended to strengthen more than the other independent variables in its relationship with PTSD variables, this analysis was run as a hierarchical regression, with PTSD and then anger, anxiety and depression at time one entered sequentially into the equation.
### Table 8.5

**Hierarchical multiple regression analysis results utilising treatment commencement PTSD total score and negative affect as independent variables and post treatment PTSD symptoms as dependent variables**

<table>
<thead>
<tr>
<th>Dependent variable</th>
<th>Variables in the equation</th>
<th>$R^2$</th>
<th>$\Delta R^2$</th>
<th>$\beta$</th>
</tr>
</thead>
<tbody>
<tr>
<td>PTSD-Total T2</td>
<td>PTSD T1</td>
<td>.28</td>
<td>-</td>
<td>.41**</td>
</tr>
<tr>
<td></td>
<td>Anger T1</td>
<td>.32</td>
<td>.04</td>
<td>.25***</td>
</tr>
<tr>
<td>PTSD-Intrusion T2</td>
<td>PTSD T1</td>
<td>.21</td>
<td>-</td>
<td>.43***</td>
</tr>
<tr>
<td></td>
<td>Anger T1</td>
<td>.23</td>
<td>.02</td>
<td>.16**</td>
</tr>
<tr>
<td>PTSD-Avoidance T2</td>
<td>PTSD T1</td>
<td>.17</td>
<td>-</td>
<td>.25***</td>
</tr>
<tr>
<td></td>
<td>Anger T1</td>
<td>.21</td>
<td>.04</td>
<td>.23***</td>
</tr>
<tr>
<td>PTSD-Arousal T2</td>
<td>PTSD T1</td>
<td>.19</td>
<td>-</td>
<td>.27***</td>
</tr>
<tr>
<td></td>
<td>Anger T1</td>
<td>.24</td>
<td>.05</td>
<td>.24***</td>
</tr>
</tbody>
</table>

* $p <.05$, ** $p <.01$ *** $p <.001$

Table 8.5 reports the results of this hierarchical multiple regression analysis. It shows that PTSD and anger scores at pre-treatment were the only two significant predictors of the dependent variables. The results of this analysis show that pre-treatment PTSD was the greatest overall predictor of PTSD post-treatment scores ($r^2 = .28, F(3,249) = 96.74, p <.001$) and anger was the only other predictor to be retained in the prediction equation ($r^2 = .32, F(2,250) = 80.1, p <.001$). Against expectations, neither anxiety nor depression at pre-treatment explained any additional variance either for PTSD total or its subscales. This anger-as-second predictor of PTSD function was true for all PTSD sub-clusters. The results of this analysis show that for PTSD Intrusion scores, pre-treatment anger explained an additional 2% of the PTSD score variance above the T1 PTSD score ($F(2,250) = 52.5, p <.001$), for PTSD Avoidance scores anger explained an additional 4% of the variance ($F(2,250) = 47.9, p <.001$) and for Arousal Scores, anger explained an additional 5% of the variance ($F(2,250) = 55.8, p <.001$) above the pre-treatment PTSD score.
Table 8.6

Hierarchical multiple regression analysis results utilising treatment commencement negative affect scores as independent variables and post-treatment PTSD scores as the dependent variable

<table>
<thead>
<tr>
<th>Dependent variable</th>
<th>Variables in the equation</th>
<th>R²</th>
<th>Δ R²</th>
<th>β</th>
</tr>
</thead>
<tbody>
<tr>
<td>PTSD-Total T2</td>
<td>Anger T1</td>
<td>.20</td>
<td>-</td>
<td>.34***</td>
</tr>
<tr>
<td></td>
<td>Anxiety T1</td>
<td>.24</td>
<td>.04</td>
<td>.23***</td>
</tr>
<tr>
<td>PTSD-Intrusions T2</td>
<td>Anger T1</td>
<td>.11</td>
<td>-</td>
<td>.24***</td>
</tr>
<tr>
<td></td>
<td>Anxiety</td>
<td>.14</td>
<td>.03</td>
<td>.20**</td>
</tr>
<tr>
<td>PTSD-Avoidance T2</td>
<td>Anger T1</td>
<td>.15</td>
<td>-</td>
<td>.31***</td>
</tr>
<tr>
<td></td>
<td>Depression T1</td>
<td>.25</td>
<td>.05</td>
<td>.23***</td>
</tr>
<tr>
<td>PTSD-Arousal T2</td>
<td>Anger T1</td>
<td>.16</td>
<td>-</td>
<td>.30***</td>
</tr>
<tr>
<td></td>
<td>Anxiety T1</td>
<td>.20</td>
<td>.04</td>
<td>.23***</td>
</tr>
</tbody>
</table>

*p < .05, **p < .01 *** p < .001

In an attempt to illuminate the relationship of negative to chance in PTSD, two further explorations of the data were undertaken. Both series of analyses were conducted without PTSD at time one. Both analyses assessed the predictive impact of negative effects on PTSD change in the absence of initial PTSD score as a predictor variable. The first of these analyses involved the use of simple regression analyses to check the effect of pre-treatment anger, anxiety and depression scores on post-treatment PTSD scores. These regressions identified anxiety ($r^2 = .13$, $F(1,252) = 39.04$, $p < .001$), depression ($r^2 = .10$, $F(1,252) = 29.28$, $p < .001$) and anger ($r^2 = .19$, $F(1,252) = 61.14$, $p < .001$) to be predictive of change in PTSD.

Following on from these analyses, the next step involved a stepwise multiple regression analysis involving all three affects at time one as predictors and post-treatment PTSD as the dependent variable. The results of this analysis are reported in Table 8.6. It showed that anger was the primary predictor for PTSD overall and for each of its sub-clusters. In three out of four instances (PTSD total, Intrusion and Arousal Scores), anxiety at time one entered the equation as the second predictor, while depression at time one entered the equation for PTSD avoidance. For PTSD total, intrusion, avoidance and arousal scores anger explained between 11% and 20% of score variance.
8.5 Discussion

This study investigated the relationship of negative affect (depression, anxiety and anger) to PTSD scores in a treatment seeking population of Australian Vietnam veterans with PTSD. This association was examined pre and post-treatment and in terms of the change relationships between those time-points.

By these means, it sought to answer several questions. The first related to the extent to which anger was associated with PTSD. The second concerned the degree to which anger is associated with PTSD, compared to anxiety and depression, affects known to be strongly associated with PTSD. The final question connected to the extent to which changes in anger would be associated with changes in PTSD.

The evidence from this study relevant to each question is now summarised, preparatory to a more detailed consideration in the concluding general discussion (Chapter Eleven) of the meaning of such results and their theoretical and clinical implications. The strengths and weaknesses of the study’s design and possible future research directions will also be discussed in detail there.

In line with the first experimental hypothesis, this study showed anger was positively associated with PTSD. This was the case pre and post-treatment. Although this was consistent with expectation, pre and post-treatment the strength of these anger-PTSD relationships was less than those applicable to anxiety and depression. At both time points, anxiety continued to have the strongest association with PTSD. Drilling down to DSM IV PTSD sub-cluster relationships, anxiety was the affect most associated with PTSD intrusions and arousal, while depression was the affect most associated with PTSD avoidance. Consequently, contrary to expectation, the second prediction, that anger’s relationship with PTSD would be greater than those for anxiety and depression, was not supported.

In contradistinction, as scores decreased from pre to post-treatment, PTSD and anger scores decreased the most and PTSD’s correlations with anger tended to strengthen most, compared to anxiety and depression. This occurred via an increased association of anger with PTSD’s intrusion and arousal sub-clusters. Reflective of this, multiple regression analyses indicated that both PTSD and anger scores pre-treatment were predictive of post-treatment PTSD scores. Pre-treatment PTSD was the greatest predictor, but these results importantly confirmed the study’s third prediction that anger would be predictive of PTSD’s trajectory. When PTSD was not included in the prediction equation, the relative impact of negative emotion on PTSD
became clearer again, with anxiety entering the equations for Intrusions and Avoidance, while depression entered the equation for Avoidance. In all cases their contribution was, however, secondary to that of anger, which was the primary predictor for PTSD overall and for each of its sub-clusters.

To conclude, this study demonstrated a clear, predictive relationship between anger and PTSD. As planned, the next stage of this research project investigates of the role of imagery in the development and maintenance of negative and angry affect.
The previous study investigated the link between anger and PTSD, relative to anxiety and depression. It established anger has an important link to PTSD, albeit not as strong as that of anxiety and depression. Importantly, it also demonstrated that there is a positive predictive link between anger and PTSD treatment outcome, such that anger at pre-treatment and changes in anger are longitudinally related to PTSD scores and prospective change in PTSD scores. There was no evidence that an equivalent relationship with PTSD applied to anxiety and depression.

As the second step in this research program, this study sought to investigate the relationship of imagery to non-PTSD related anger. As in the previous study, anger was compared with anxiety and depression. By including a measure of negative thought, it also aimed to investigate the comparative relationships of imagery and thought with anger compared to those applicable to anxiety and depression.

9.1 Research Design

This study explores the cross-sectional association between visual imagery and anger in a non-clinical population. It contrasts these data with the association of imagery with anxiety and depression, since those affects are considered to have strong and weak relationships with imagery respectively. These data are further assessed against data pertaining to the relationship of negative thought to each affect.

9.2 Method

9.2.1 Participants. The participants in this study were undergraduate psychology students of Swinburne University of Technology in Melbourne, Australia. At the time of testing, the initial participant pool comprised 208 students. A total of 11 participants were excluded due to their status as multivariate and univariate outliers. The adjusted sample of 197 participants comprised 150 women and 47 men.
Anger-in-PTSD: The Role of Visual Imagery

(see figure 9.1). This participant gender profile reflects the enrolment distribution of undergraduate male and female psychology students at Swinburne University and across Australian Universities. There was no significant change in the sample demographic characteristics after this correction.

**Figure 9.1**  Community sample flow summary

9.2.2 **Measures.**

9.2.2.1 **Demographic Data.** Demographic information was gathered via simple survey. It consisted of age, gender, education, employment and relationship status data.

Participant demography is detailed in Appendix E. It shows that at the time of testing, the ages of study participants ranged from 17 to 67, with a mean age of 22.37 (SD = 7.68). The majority of whom were female (159 women, 49 men), in full or part-time employment (79%) and single (85%).

9.2.2.2 **Clinical Data.** The following measures of anxiety, depression, anger, imagery and negative thought were used to obtain clinical data in this study (see Appendix G for copies of all instruments used in this study). They were adopted for the reasons described below.
Imagery

A thorough review of available psychological tests of imagery capacity was conducted in order to establish the most suitable test for the experimental tasks concerned in this study. It revealed there are a variety of means for measuring human imagery. It was understood that, in order to validly assess the range of imagery characteristics targeted in this research a broad ranging measure/set of measures was necessary. On the basis of previous research and theory, an approach was taken that proved consistent with the position subsequently advocated by Pearson and associates (2013) in their review of imagery methods.

Pearson and associates did not attempt to review the validity or psychometric properties of existing tests, since that had already been undertaken in previous reviews such as those by MacInnis (1987) and Richardson (1994). Rather, their objective was to describe the range of tasks and metrics available to researchers, and to articulate a framework for guiding researchers in choosing measures for assessing the aspect of imagery most relevant to their research focus. Their review identified what they described as the extensive, diverse and eclectic means for measuring imagery. Tasks described by them included script driven imagery processes, intrusion triggering tasks, and intrusion diaries. Scales included those which assess general imagery use, imagery re-experiencing, bias in imagery and prospective imagery.

Although their review post-dated the point at which this research was designed, the imagery measures considered for use in this research and the ultimate imagery measure of choice are consistent with their recommendations. Several often used measures were considered, including Betts Questionnaire Upon Mental Imagery (QMI; Richardson, 1969), the Modified Gordon Test of Visual Imagery Control (TVIC; Richardson, 1977) and the Vividness of Visual Imagery Questionnaire (VVIQ; Marks, 1973). These three scales have sound psychometric properties and are reliable and valid measures of the imagery facets they attempt to measure. They were not considered appropriate for the current research, however, given their narrowness of focus and, in the case of the VVIQ, structured interview format. That format, due to the potential burden it posed to participants, was rejected in favour of a self-report measure.

Given the breadth of imagery characteristics targeted in this and the next study, an omnibus test was required to adequately test the research hypotheses. The only such test available is the Imaginal Process Inventory (IPI). The IPI was
developed by Singer and Antrobus (1970; 1972) to investigate what they referred to as daydreams, but is now understood more broadly as an instrument for measuring mental imagery. It has been used to assess imagery in research of constructs as diverse as mindfulness, sleep quality, psychological wellbeing, relaxation, spontaneous thought, absorption, disengagement and dissociation and brain activity and its short form has become the preferred measure of visual imagery (Huba et al., 1982).

As far as could be established, the IPI has not previously been used in affect-related research, anger included. It’s use in the wide range of research domains noted immediately above, the extensive range of content domains assessed by its 28 subscales and 344 questions and the strong psychometric properties of it and its short form, made it a logical choice for the objectives of this research.

Four subscales from the IPI were selected to assess visual imagery in the current study. The resultant 56-item measure, hereafter referred to as the IPI-abridged version (the IPI-AV), comprised the IPI Daydream Frequency Subscale (DDFS; 12 items), the IPI Absorption in Daydreams Subscale (DDAS; 20 items), the IPI Daydream Vividness Subscale (DDVS; 12 items) and the IPI Hostility in Daydream Subscale (DDHS; 12 items). [For clarity, the word ‘imagery’ will be used in this thesis instead of ‘daydream’ when referring to IPI-AV subscales. Also, given the current meaning of the term ‘hostility’ - which includes personality dimensions that were not clearly delineated when the IPI was created (see McHugh et al., 2012) - the word ‘anger’ will be used to describe imagery of an irritable nature.] Participants respond to each probe using 5-point Likert scales. Possible scores on the three 12-item subscales range from 12 to 60 and from 20 to 100 on the 20-item absorption scale. Higher scores on all scales indicate greater presence of the target phenomenon.

These four sub-scales were chosen because they directly measure constructs pertinent to imagery’s relationship with negative affect. The first three have strong associations with negative affect, including depressed mood, anxiety and the mixed affective-distress of PTSD (Bywaters et al., 2004b; Day, Holmes & Hackmann, 2004; Laor et al., 1999; Rauch, Foa, Furr & Filip, 2004); the last was included given the study’s focus on anger. The inclusion of the anger the subscale of the IPI was logically justified given the focus of this research on anger. Its inclusion engendered a contrast of the effect of a content facet of imagery on negative affect via a comparison of its effect on anger, anxiety and depression. It served as a useful “proof of concept”
check on the grounds that if there was no or a negligible association of angry imagery with anger, then this would be a clear indication of a null imagery effect. It also enables a contrast to be undertaken between the effect of a specific content (anger) and process characterises (frequency, vividness and absorption).

A fifth scale of the IPI was included in the imagery measure as provided to participants in this study (and Study Three). This scale, the IPI Presence of Imagery subscale (DDNS; 12 items), is ostensibly a measure of cognitive style (as assessed by descriptors mapping the tendency to preferentially use imagery, as opposed to thought, in information processing and cognition) (Appendix H). It proved to be unreliable (it obtained a Cronbach alpha reliability of .62 in this study). Attempts at eliminating items to improve it reliability and establishing an effective truncated structure via confirmatory factor analysis proved unsuccessful. The DDNS was consequently omitted from all analyses in this research.

Such scales were deemed a sufficient abridgement of the IPI, given that factor analysis suggests it contains redundancy (Huba et al., 1982) and its total application may be an unnecessary burden on respondents. The full IPI has adequate levels of internal consistency, with its 28 subscales attaining Cronbach’s alphas of between 0.70 and 0.90 (Huba et al., 1982), test-retest reliabilities of between 0.65 and 0.91 (Giambra, 1999a; 1999b), and sensitivity to the effect of aging on imagery frequency (Giambra, 1993). In this study, the DDFS, DDAS, DDV and DDHS attained Cronbach’s alphas of between .90 and .95.

An omnibus imagery measure was constructed by summing the totals of these four measures. This enabled an index of overall imagery capable of contrast with the thought measure (described immediately below). This General Imagery Measure (GIM) attained a Cronbach’s alpha of .73 in this study.

Negative Thought

The negative automatic thoughts subscale (the NAT Subscale) of the Automatic Thoughts Questionnaire-Revised (ATQ-R; Kendall, Howard & Hays, 1989) was used to measure thought. The ATQ-R is an internally consistent 40-item scale with demonstrated sensitivity to treatment effects and good reliability and validity in measuring automatic thought frequency (Burgess & Haaga, 1994; Hill, Oei & Hill, 1989). The NAT Scale’s 30 items are scored on a five point (0 - 4) Likert scale. Possible scores range from 0 to 120, with higher scores indicating greater
negative thought frequency. In this study, the NAT Scale attained a Cronbach alpha of .95.

**Anxiety Depression and Anger**

The anxiety and depression subscales from the Depression, Anxiety and Stress Scale (DASS; Lovibond & Lovibond, 1995) were used to assess symptoms of anxiety and depression. Each scale consists of seven items rated on a 4-point Likert scale. Higher scores indicate greater levels of depression or anxiety. The DASS was chosen for use in this study on account of its reliable and valid psychometric properties. It is noted to have excellent reliability, as well as convergent and discriminant validity (Crawford & Henry, 2003). In this study, the anxiety and depression subscales respectively attained Cronbach alphas of .74 and .83.

The Dimensions of Anger Reactions Scale (DAR Scale; Novaco, 1975), as previously described in Chapter Eight, was used to assess anger. In this study, the DAR Scale attained a Cronbach alpha of .88.

### 9.2.3 Procedure

To facilitate these objectives, participants were recruited via advertisement and asked to complete an internet survey program (Opinio) posted on the Swinburne University of Technology website. Participants accessed the study questionnaires by entering the advertised link into their preferred internet browser. They completed the battery of questionnaires involved on a voluntary basis and respondents were advised they were free to withdraw at any point without any consequence. Informed consent was implied by the submission of completed questionnaires onto the Opinio system as described above. Questionnaire completion was estimated to require approximately 30 minutes. Data from the completed questionnaires was downloaded into an SPSS file through the Opinio program. This study was approved by the Swinburne University Ethics Committee (see Appendix I). Course credit was provided for participation. No monetary payment was made to any individual for participation in this research.

### 9.3 Preliminary Data analysis

Prior to analyses, all scale scores in this study were checked for missing data and normality of distribution. There was no missing data. Normality tests detected a small number of outliers. Univariate and multivariate outliers were removed from the data set by applying a z-score cut-off of 3. This resulted in a decrease in sample size and improved the typically positively skewed nature of the data. Variable
transformation to remove skewness was considered (e.g., via natural log), but was
decided against given the observations of Tabachnik and Fidel (2013) that, if all
variables are similarly moderately skewed, improvements due to transformation are
often marginal. The data were deemed to be within acceptable limits of normality.
Multivariate and univariate analyses of variance were conducted to detect any gender
bias. Although there were significant mean score differences between male and
female participants overall, these pertained to three of the four imagery scales only
(see Table 1). The differences on the three (frequency, absorption and anger-related)
IPI-AV subscales rendered no significant differences for either the correlations or
regression analyses conducted.

Correlation analysis was undertaken on mean scale scores to examine the
relationships between the study’s imagery, thought and affect measures. Williams T, a
statistic for testing the significance of the difference between two independent
correlations (Steiger, 1980), was subsequently calculated and applied to the
correlations to identify any significant between-correlation differences. A series of
hierarchical multiple regression analyses was then performed to further explore the
relationships between the study’s variables. Finally, mediation analysis was deployed
to assess the interaction of imagery and thoughts in the overall relationship with the
study’s affect measures.

9.4 Results

Mean scores for all measures are reported in table 9.1. They indicate IPI
subscale scores considerably higher than those previously reported (see table
footnote). The NAT Scale and DAR Scale scores are, not surprisingly, substantially
lower than those reported for clinical populations [e.g., the mean scores of 55.12
reported by Nezu, Rowan, Meadows & McLure (2000) for the NAT Scale and 32.5
for the DAR reported by Forbes and colleagues (2008)]. DASS scores for anxiety and
depression fell within the middle and lower end of Australia-normed scores
respectively (Lovibond & Lovibond, 1995)

Pearson product-moment correlation analysis was applied to examine the
relationships between the study’s imagery, thought and affect measures (see table
9.2). The correlations between the four (frequency, vividness, absorption and anger)
imagery subscales and three (anxiety, depression and anger) affects were generally
low. The only imagery-affect correlation that attained a moderate intensity was
between angry imagery and anger. In contrast, negative thoughts were moderately-to-highly correlated with all affects and, with the exception of imagery anger, had stronger correlations with the affects than the imagery variables.

Table 9.1
Scale and subscale means and standard deviations by gender with tests of difference (Anova) and reliability (Cronbach’s alpha)

<table>
<thead>
<tr>
<th>Variables</th>
<th>Total</th>
<th>Male</th>
<th>Female</th>
<th>f</th>
<th>p</th>
<th>α</th>
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<td></td>
<td>(n = 197)</td>
<td>(n = 47)</td>
<td>(n = 150)</td>
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<td>Imagery Frequency Scale (DDFS)</td>
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<td></td>
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</tr>
<tr>
<td>M</td>
<td>36.5</td>
<td>33.7</td>
<td>37.4</td>
<td>4.52</td>
<td>.04</td>
<td>.93</td>
</tr>
<tr>
<td>SD</td>
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</tr>
<tr>
<td>Imagery Absorption Scale (DDAS)</td>
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</tr>
<tr>
<td>M</td>
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<td>47.4</td>
<td>52.8</td>
<td>4.70</td>
<td>.03</td>
<td>.91</td>
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<tr>
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<tr>
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<td></td>
<td></td>
</tr>
<tr>
<td>M</td>
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<td>18.8</td>
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<td>.90</td>
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<tr>
<td>M</td>
<td>124.8</td>
<td>119.8</td>
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<td>M</td>
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<td>M</td>
<td>50.4</td>
<td>48.2</td>
<td>51.1</td>
<td>1.56</td>
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<td>.95</td>
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<td>Depression, Anxiety &amp; Stress Scale - Anxiety Subscale (DASSA)</td>
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<td>10.5</td>
<td>2.19</td>
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<td>.74</td>
</tr>
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<td></td>
</tr>
<tr>
<td>Depression, Anxiety &amp; Stress Scale - Depression Subscale (DASSD)</td>
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<td></td>
</tr>
<tr>
<td>M</td>
<td>10.7</td>
<td>10.4</td>
<td>10.8</td>
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<td>.46</td>
<td>.83</td>
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<td>Dimensions of Anger Scale (DAR)</td>
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<td></td>
</tr>
<tr>
<td>M</td>
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<td>19.7</td>
<td>19.8</td>
<td>.00</td>
<td>.95</td>
<td>.88</td>
</tr>
<tr>
<td>SD</td>
<td>9.5</td>
<td>9.7</td>
<td>9.4</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Note: Giambra (1999b) reported the following respective means for males and females for the IPI scales deployed: DDFS - 27.6 (SD = 8.0) and 28.9 (SD = 7.6); DDAS - 36.4 (SD = 8.3) and 39.2 (SD = 9.2) and DDVS - 11.3 (SD = 5.7) and 11.5 (SD = 5.4)
In line with experimental predictions, these results show that imagery and thought were positively and significantly related to negative affect. They indicate angry imagery was most highly correlated with angry affect and that imagery-frequency, absorption and vividness were more strongly associated with depression and anxiety. These relationships were generally comparable in strength. They show negative thoughts to be most highly correlated with depression and thereafter with anxiety, not anger.

Table 9.2
Relationship of Imagery and Thought to anxious, angry and depressive affect
(Pearson’s r)

<table>
<thead>
<tr>
<th></th>
<th>DDAS</th>
<th>DDVS</th>
<th>DDHS</th>
<th>GIM</th>
<th>NATS</th>
<th>DASSA</th>
<th>DASSD</th>
<th>DAR</th>
</tr>
</thead>
<tbody>
<tr>
<td>DDFS</td>
<td>.66**</td>
<td>.31**</td>
<td>.19*</td>
<td>.82**</td>
<td>34**</td>
<td>.23**</td>
<td>.30**</td>
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<td>33**</td>
<td>.31**</td>
<td>.35**</td>
<td>.26**</td>
<td></td>
</tr>
<tr>
<td>DDVS</td>
<td>.44**</td>
<td>.85**</td>
<td>.30**</td>
<td>.35**</td>
<td>.30**</td>
<td>.23**</td>
<td></td>
<td></td>
</tr>
<tr>
<td>DDHS</td>
<td>.70**</td>
<td>.29**</td>
<td>.24**</td>
<td>.23**</td>
<td>.41**</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>GIM</td>
<td>..</td>
<td>.40**</td>
<td>.34**</td>
<td>.38**</td>
<td>.24**</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>NATS</td>
<td>..</td>
<td>..</td>
<td>.54**</td>
<td>.70**</td>
<td>.40**</td>
<td></td>
<td></td>
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<tr>
<td>DASSA</td>
<td>..</td>
<td>..</td>
<td>..</td>
<td>.56**</td>
<td>.43**</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>DASSD</td>
<td>..</td>
<td>..</td>
<td>..</td>
<td>..</td>
<td>.43**</td>
<td></td>
<td></td>
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</tr>
</tbody>
</table>

* p < .05, ** p < .01

Application of William’s T statistic identified several significant differences in correlation magnitudes. First, among imagery-affect correlations, imagery-frequency had a significantly stronger correlation with depression than anger. Imagery-anger had a significantly stronger correlation with anger, compared to
anger and depression. Interestingly, there was no difference between the correlations of imagery-anger and absorption with anger (t = 1.8, p = .07). There were no other significant differences between any of the other imagery-affect correlations. Second, observation of the negative thought-affect correlations showed that negative thoughts had significantly stronger correlations with anxiety and depression than any of the imagery measures. Negative thoughts also had significantly stronger correlations with depression than with anxiety and anger, and with anxiety compared to depression (see Table 9.3).

Table 9.3

*Differences between Imagery, Thought and Affect Correlations (William’s T)*

<table>
<thead>
<tr>
<th></th>
<th>DASSA: DASSD</th>
<th>DASSA: DAR</th>
<th>DASSD: DAR</th>
</tr>
</thead>
<tbody>
<tr>
<td>DDFS</td>
<td>-1.1</td>
<td>1.7</td>
<td>2.7**</td>
</tr>
<tr>
<td>DDAS</td>
<td>-0.6</td>
<td>0.7</td>
<td>1.2</td>
</tr>
<tr>
<td>DDVS</td>
<td>0.8</td>
<td>1.7</td>
<td>1.0</td>
</tr>
<tr>
<td>DDHS</td>
<td>0.3</td>
<td>-2.4*</td>
<td>-2.6**</td>
</tr>
<tr>
<td>GIM</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>NAT Scale</td>
<td>-3.2**</td>
<td>2.2**</td>
<td>5.2**</td>
</tr>
</tbody>
</table>

* * p < .05, ** p < .01

These results provided support for further exploration of the interaction of imagery and thought on affect. Accordingly, a stepwise multiple regression analysis was run, with imagery, frequency, absorption, vividness and negative thoughts as the independent variables and anxiety, depression and anger as the dependent variables. The results showed that, for anxiety, negative thoughts and imagery-vividness were the two significant predictors ($r^2 = .33$, $F_{(2,194)} = 47.9, p < .01$). For depression, negative thoughts and imagery-absorption ($r^2 = .50$, $F_{(2,194)} = 96.9, p < .01$) were significant predictors, while for anger imagery-anger and negative thoughts were both significant predictors ($r^2 = .26$, $F_{(2,194)} = 33.5, p < .01$) (Table 9.4). Notably, in these analyses, negative thoughts were the primary predictor (the first variable entered into the equation as having the greatest unique contribution) for anxiety and depression, while for anger, imagery-anger had primacy over negative thoughts.
The next step was to assess whether the established relationships of significance constituted either moderator or mediator relationships. According to Baron and Kenny (1986), for mediation four requirements must hold: first, the independent variable must predict the dependent variable; second, the independent variable must predict the mediating variable; third, the mediating variable must predict the dependent variable; and, finally, the independent variable must not remain a predictor when the mediator is controlled. Mindful of this, hierarchical multiple regression analyses were performed to further examine the relationships between imagery (using the most appropriate subscale as determined by the previous analysis), negative thought, and affects. These analyses demonstrated several outcomes as illustrated in Figure 9.2.

Beginning with the dependent variable of anxiety, standard regressions showed imagery-vividness was a significant predictor of anxiety ($r^2 = .36$, $F_{(1,195)} = 26.67, p < .01$) and predicted negative thoughts ($r^2 = .30$, $F_{(1,195)} = 19.17, p < .01$). Third (as already noted), negative thoughts predicted anxiety ($r^2 = .29$, $F_{(1,195)} = 80.85, p < .01$) and, finally, imagery-vividness, while remaining a significant predictor, explained only 4% of anxiety variance, once negative thoughts were controlled (see table 4). These results suggest negative thoughts mediate imagery-vividness in
producing anxiety. That is, without negative thought, vivid imagery does not, in itself, produce anxiety.

Next, using depression as the dependent variable, standard regressions showed imagery-absorption was a significant predictor of depression ($r^2 = .35$, $F_{(1,195)} = 26.83, p < .01$) and predicted negative thought ($r^2 = .33$, $F_{(1,195)} = 23.21, p < .01$). Third (as already noted), negative thought predicted depression ($r^2 = .48$, $F_{(1,195)} = 182.44, p < .01$) and, finally, imagery-absorption, while remaining a significant predictor, explained only 2% of depression variance once $NAT$ Scale were controlled (see Table 4). These results suggest negative thoughts mediate imagery-absorption in producing depression. That is, without negative thoughts, imagery-absorption does not, in itself, produce depression.

Finally, taking anger as the dependent variable, a standard regression showed (as noted) that imagery-anger was a significant predictor of anger ($r^2 = .17$, $F_{(1,195)} = 36.69, p < .01$) and predicted negative thought ($r^2 = .09$, $F_{(1,195)} = 18.12, p < .01$). Third (as already noted), negative thought predicted anger ($r^2 = .16$, $F_{(1,195)} = 37.85, P < .01$). In the subsequent hierarchical regression with imagery-anger entered first and negative thought second, imagery-anger remained a significant predictor at the end of the second step, although its beta weight dropped from .32 to .31. Negative thoughts were a significant predictor ($p < .001$) and added an additional 9% to the explained variance in anger and $R^2$ change was significant ($r^2 = .26$, $F_{(1,195)} = 33.52, p < .001$) (see table 4). These results support a partial mediation hypothesis indicating that the impact of imagery-anger on anger was partially determined by levels of negative thought.
Anger variance explained by Imagery Anger (DDHS) and NATS = 26%

Anxiety variance explained by Imagery Vividness (DDVS) and Negative Thought (NATS) = 33%

Depression variance explained by Imagery Absorption (DDAS) and NATS = 50%

Anger-in-PTSD: The Role of Visual Imagery

Figure 9.2  Imagery, thought and anxiety, depression and anger variances
9.5 Discussion

This study cross-sectionally investigated the relationship of imagery and thought to anger, anxiety and depression in a community population. It sought to answer several questions. The first related to the extent to which imagery was associated with anger. The second concerned the comparative strength of imagery’s relationship to anger, anxiety and depression. The third was about the strength of relationship of negative thought to anger in contrast to that applicable to anxiety and depression. The final question was focused on whether there is inter-relationship between imagery, or facets of imagery, and thought in anger, and, if so, how that relationship compared to any pertaining to anxiety and depression.

Evidence from this relevant to each question is now summarised, preparatory to a more detailed consideration in the concluding general discussion (Chapter Eleven) of the meaning of such results and their theoretical and clinical implications. The strengths and weaknesses of the study’s design and possible future research directions will also be discussed in detail there.

As anticipated by its first experimental hypothesis, this study’s findings demonstrated anger was positively, but mildly associated with imagery. Contrary to expectation, however, the second prediction, that imagery’s association with negative affect would follow an anxiety-anger-depression order of magnitude, was not supported and surprisingly when measured via imagery overall (i.e. by the GIM) depression had the greatest association, anxiety an intermediate association and anger the least association with imagery.

Again consistent with the third of the study’s prediction, different facets of imagery exhibited different patterns of association with anxiety, depression and anger. Significant differences between these imagery-affect correlations were few, however, and restricted to those involving imagery frequency and anger. For imagery frequency, the correlation with depression was significantly greater compared to that for anger. In turn, the single greatest imagery-affect correlation in the study was between imagery anger and anger.

Next, the study’s findings also showed that, compared to imagery, negative thought had stronger and significantly different associations with negative affects, the only exception being that of imagery-anger with anger. Negative thought had the strongest association with depression, then anxiety and least with anger. Each thought-affect correlation differed significantly from the other two.
There were several noteworthy findings regarding the interaction of imagery and thought in their impact on affects. First, negative thought significantly predicted all three affects when it and imagery measures were included as independent variables in regression equations. Importantly, while negative thought was the primary predictor for anxiety and depression, angry imagery had primacy for anger. Interestingly, each affect was also predicted by imagery, but by different imagery characteristics: for anxiety it was imagery-vividness; for depression, imagery-absorption; and for anger, imagery-anger.

Those aspects of imagery and thought alone and together had distinct relationships with different negative affects, and more specifically that angry imagery had a closer association to angry affect than vividness was to anxiety or absorption to depression, was not surprising and was in line with expectations and the specific experimental design on this study. To recall from Chapters Five to Six, imagery has a well demonstrated connection to anxiety (see Homes & Matthew, 2010), but not to depression (Fresco et al., 2010). On this basis, imagery was operationalised by reference to the three imagery process characteristics of frequency, vividness and absorption. Although largely unexamined in relation to anger, such characteristics were on the basis of the limited available research evidence, expected to be relevant to it. Because of this research program’s central interest in anger, a measure of angry imagery that was available in an IPI subscale (the DDHS) was specifically included as a measure of imagery content. In operation, the DDHS was expected to have a strong relationship with anger. Via a contrast of that relationship with those pertaining to the operation of imagery process characteristics like frequency, absorption and vividness, this study provided an obvious point of reference for determining their relative impact on negative affect overall and anger in particular.

The findings of this study provide impetus for further investigation of the role of imagery and thought in their separate and joint effects on negative affect and anger, in particular. The next, and final study, in this research program, attempts to measure the effect of imagery, and secondarily thought, on anger, anxiety and depression and PTSD in different populations of PTSD sufferers.
CHAPTER 10: STUDY 3: INVESTIGATION OF THE PREVALENCE, VARIABILITY AND EFFECT OF IMAGERY ON ANGER IN A PTSD-AFFECTED POPULATION

It is often the last key on the ring that opens the door

Ancient Proverb

The first of the three studies in this thesis established a link between anger and PTSD that showed anger to be a significant predictor of change in PTSD severity. The second study demonstrated that negative affect and visual imagery were associated. This relationship was mediated by thought and a different imagery-thought relationship existed for each of anxiety, depression and anger. Under-pinned by these two sets of results, this third study, as the concluding investigative element of this thesis, explored the relationship of imagery to anger in PTSD in different PTSD populations and the stability of that relationship over time. Like Study Two, the relationship of imagery to anger in PTSD was contrasted with those pertaining to negative thought.

10.1 Research Design

To facilitate these objectives, analyses were performed on data gathered from current and former-military personnel (veterans) of the Australian Defence Forces (as an overall group hereafter referred to as ADF-affiliated participants) and current and former police who received psychiatric and/or psychological treatment for trauma-related mental health conditions at the Psychological Trauma Treatment Service (PTRS) of Austin Health, via participation in programmatic group and individual PTSD treatment.

Where possible, these data were sought from participants pre-treatment or failing that in the earliest stages of treatment and thereafter 12 months later. Where data was gathered from ADF-affiliated treatment program participants, elements of it were accessed from databases held by Phoenix Australia and the PTRS. The role and nature of those organisations was described in Study One (see Chapter Eight method section).
This study was approved by the Austin Health Human Research and Ethics Committee (AHHREC). All participants had previously provided informed consent for access to and examination of these data when completing the metrics involved as part of the process of entering into treatment. The Patient Informed Consent Form approved for use by the AHHREC was as per Study One and, as previously indicated, is attached at Appendix D. No monetary payment was made to any individual for participation in this study.

10.2 Methodology

10.2.1 Participants. The participants in this study were current and former-serving (veteran) members of the Australian Defence Force (the ADF) (hereafter referred in this and the next chapter as ADF-affiliated participants) and current and former-serving police with a diagnosis of PTSD who had received either or both psychiatric and psychological treatment for PTSD and its comorbidities at the PTRS. The majority of ADF-affiliated participants and all police participants were contemporaneous consecutive admissions to an Austin Health PTSD Treatment program. A substantial minority of the former group (41%) had previously received programmatic PTSD treatment at the PTRS, but were continuing recipients of outpatient psychiatry and/or psychology care at that Service. The total of 232 participants in the final study sample comprised 109 ADF-affiliated participants and 123 police participants (see figure 10.1).
Figure 10.1 Participant flow summary

10.2.2 Measures. This study used the same affect measures as Study One, apart from the community population version of the PTSD Checklist (the PCL-C: Weathers et al., 1993) (See Appendix J). The PCL-C is an adapted version of the original PCL-M with slight wording changes on some items to increase its applicability to general PTSD and is considered to have the same psychometric qualities as the PCL-M. The imagery and thought measures of Study Two were also used in this study. All such measures were described in Chapters Eight and Nine and justifications for their use were as stated there.

In this study, the HADs-a and HADs-d respectively attained Cronbach alpha coefficients of .83 and .80, the DAR Scale attained a coefficient of .91, the Alcohol Use Disorders Identification Test attained a Cronbach alpha of .88 and the PCL-M and PCL-C respectively attained Cronbach alphas of .98 and .93 (See Table 10.1).
The same imagery measure used in Study Two was used in this study (see Appendix H). Although there is no evidence the IPI has been used in research of PTSD, it was judged to be the most suitable measure for assessing the imagery domains targeted in this research. In this study, these measures attained strong Cronbach alpha coefficients (of between .82 and .92), while the negative subscale of the Automatic Thoughts Questionnaire-Revised attained a Cronbach alpha of .98 (See Table 10.1).

As with study two, a fifth subscale of the IPI, (DDNS) was initially included as a measure of cognitive style (for determining the study sample’s the proportionate levels of imagery and thought). Like study two, the DDNS did not perform as anticipated and had poor internal consistency (it obtained a Cronbach alpha of .66) that was not resolved by factors analyses and item reduction. It was subsequently eliminated from all analyses on the basis of its unreliability.

10.2.3 Procedure. The referral and entry processes for current serving ADF personnel and police participants to the Austin Health PTSD Program are substantially similar to those procedures pertaining to its veteran PTSD Treatment Program as described in Chapter Eight (see sub-section 8.2.2). The majority of the referral sources are also as described there, but additionally include medical officers of the ADF, the Police Association of Victoria and the Workers Compensation Insurance agent with contractual responsibility for police workplace injuries.

Funding for program participation for veterans is as described in Chapter Eight. For current serving ADF Personnel and police participants, irrespective of work status, treatment is funded on a fee-for-service basis under specific service agreements with the ADF and the Victorian WorkCover Authority via its appointed Workcover Insurance agent. The latter is the government instrumentality responsible for the funding of Workers Compensation treatment services for the state of Victoria. Some interstate and national equivalent agencies also provide fee-for-service funding, where participants are not Victorian police.

Eligibility criteria for entry into treatment are as outlined for veterans in Chapter Eight (see sub-section 8.2.2). Police program candidates are also screened in a pre-treatment orientation session to account for the possibility that they may be known to each other. In this relatively common scenario, participants are given the option of participating in another cohort. This likelihood is less common for ADF-
affiliated program participants. They are nonetheless provided with the opportunity to delay participation where another member of the potential cohort is known to them.

ADF-affiliated and Police participants are assessed by a PTRS Clinical Psychologist, Psychiatry Registrar or Consultant Psychiatrist. The CAPS is not routinely used by the PTRS to determine PTSD status for current ADF-personnel or Police candidates for the Program as all have an established PTSD diagnosis under the applicable military and Worker’s Compensation Systems and acceptance of medico-legal liability for their treatment by the Department of Defence or the aforementioned Workcover Insurance agent. Otherwise assessment processes are largely the same.

ADF participants are treated contemporaneously with veteran participants in military cohorts. The nature of these cohorts is detailed in Chapter Eight. Police cohorts are similarly homogenous. The Police Program is, as per the veteran PTSD treatment program, cognitive-behavioural in orientation. Key components include psycho-education modules, trauma-focused sessions that address common trauma-related themes (rather than group exposure), arousal management (including anger and anxiety management), depression management and alcohol and substance management modules. The duration of the program is 10 weeks, with participants attending two consecutive days per week. A partners component is included in the program, but is reduced compared to the police program and consists of five, day-long sessions across the time of the program. The details and outcomes of this Program are yet to be published. In broad terms, however, its major treatment components are similar to those of group treatment programs for veterans (see Creamer et al., 1999).

The capacity for individual treatment of current serving military personnel and police outside the Program is expanded, compared to the entitlement of veterans, where funding is usually limited to the course of the program. Police participants and current serving personnel are frequently seen for some weeks or months prior to the program, as well as subsequent to it.

Re-administration of the study metrics (time two) occurred twelve months post the completion of the initial administration of this battery of outcomes measures. For a minority of participants, for various reasons - most commonly failure to return metrics on initial request - this did not occur within this timeframe and the two-time point data collection occurred slightly 12 twelve or more months apart. All measures in this study were completed by manually (i.e., using “pencil and paper tests”).
Demographic information was gathered via survey. It consisted of age, gender, relationship and employment status and service characteristics. Full details of it are provided in Appendix E. Briefly, it shows that for the total population pre-treatment, participants were aged from 24 to 83 years, with a mean age of 53.73 (SD = 10.43). The majority were men (all women were current serving police) and 78 per cent were married or in a marriage-like relationship and 22 per cent were single, separated or divorced.

Of the ADF-affiliated participants, the majority (78%) were not employed in any form and were in receipt of a pension. The ages of these ADF-affiliated participants ranged from 24 to 83, with a mean age of 58.54 (SD = 11.06). All such participants were male and the majority were married (77.1%). Sixty seven per cent were Vietnam veterans, five percent had served in Rwanda or Somalia, 12 per cent saw duty in East Timor and five per cent were involved in the conflicts of Iraq, Afghanistan or the gulf war. Apart from the Vietnam veterans, all were regular (professional) servicemen. The vast majority were past serving members of the Army (86%), with only seven per cent from the RAAF and six per cent from the RAN. Of the Vietnam veterans the majority were military conscripts (56% were National Servicemen and 44% were career or regular ADF personnel).

That data also indicates that at the time of initial collection, 123 police participants were involved in this study. Briefly, 52 per cent were currently employed as police and 48 per cent were retired from service. Their ages ranged from 33 to 67, with a mean age of 48.64 (SD = 7.83). The vast majority of police participants (91%) were men (109) and most were married (76%).

10.3 Preliminary data analysis

Prior to any analyses, all scale scores were checked for missing data, normality of distribution and outliers. There were occasions of missing data, due to participant failure to complete each and every item of all questionnaires included in the measurement battery. Consistent with the recommendations of Elliot and Hawthorne (2005) and Hawthorne and Elliot (2005), several approaches were used to address various data-gaps. For the small number of cases with single or several missing items of data, the closest match method was used. For the few cases where more than several of the items of a scale were unanswered, mean substitution was
employed. Where no items were completed for more than one scale, that case was eliminated.

Tests of normality were subsequently performed on all scales included in the study. These tests also indicated the majority of scale scores were mildly positively skewed and kurtotic. The only exceptions were the right skewed nature of imagery vividness scale scores and the leptokurtic nature of DAR scale scores. Consistent with Study One, variable transformation to remove such skewness and kurtosis was considered (e.g., via natural log). Again this was not undertaken, given the observations of Tabachnik and Fidel (2013) that, if all variables are similarly moderately skewed, improvements due to transformation are often marginal.

These tests detected a small number of univariate and multivariate statistical outliers. Applying a z-score cut-off of 3, these outliers were removed via reiterative inspections until all were eliminated. These outliers did not differ significantly from the remaining study population (differences as measured by Cohen’s $d$ ranged from .02 to .09 and were not significant) in terms of their raw score characteristics after this correction for either the ADF-affiliated/police population samples or aggregated population sample. The adjusted sample, after correction for outliers ($n = 30$; 18 ADF-affiliated and 12 police participants) comprised 232 (109 ADF-affiliated and 123 police) participants.

Multivariate (MANOVA) and univariate (ANOVA) analyses of variance were also conducted to detect any biasing effect on results of age, gender or group (ADF-affiliated versus police participant) membership. Because all female participants were police, analyses pertaining to gender were, accordingly, only run on the police participant sub-sample data. Applying Wilks Lambda, these analyses revealed no overall effect of gender on results ($F(14,107) = 1.54, p = .05$) (see Appendix K for a comparison of mean scores by gender). Consistent with Study Two’s finding of significant gender differences on several of the scales common to this study, ANOVAs subsequently run on the police participant data set showed that female police participants had higher imagery frequency ($F(1,121) = 11.67, p = .001$) and were more absorbed in visual imagery ($F(1,121) = 5.34, p = .02$). It was also the case that women police tended to experience more PTSD intrusions than their male counterparts ($F(1,121) = 3.30, p = .07$).

Group membership had a significant effect on overall scores ($F(1, 231) = 3.50, p < .001$), with ADF-affiliated participants, and particularly non-Vietnam
veteran participants, exhibiting significantly higher mean scores compared to police participants across the majority of the independent variables (total imagery score and its subscale derivatives relating to imagery vividness, absorption and anger) and dependent variables (HADs-a, Hads-d, DAR Scale, PCL and each of its subscale scores) (Appendix L). Non-significant differences in scores were apparent on only two independent variables (imagery frequency and automatic thought) and one dependent variable (alcohol abuse).

In an initial set of analyses focused on the total population sample, Pearson product-moment correlation analysis was undertaken on mean scale scores to examine the relationships between the study’s imagery, thought, affect, alcohol abuse and PTSD measures. Given this study’s ultimate goal of testing the explanatory model of anger in PTSD articulated in Chapter Seven, while establishing the direction(s) of causality within that model and consequent of the array of variables involved, Path Analysis using Structural Equation Modelling (SEM) was seen as the ideal analytic fit for the tasks involved. SEM permits simultaneous calculations of multiple direct and indirect relationships. In the model an arrow between variables denotes the relationship examined. Where there is one arrow point, the head of the arrow represents the variable that is regressed on the variable located at the tail of the arrow. Where arrowheads exist at both ends of the represented path, this represents a correlational rather than causal relationship. When the path between two variables includes two or more arrows, this represents the operation of direct and indirect relationships, with the intervening variable being a mediator between the two variables located at either end of the path. Regression coefficients (β) measure the strength of a relationship between variables, with a higher standardised value signifying a stronger relationship (βs of .10 are considered a small effect, with values of 0.3 to 0.5 representing medium-size effects and .50 and above representing large effects). Mplus version 7.01 (Muthen & Muthen, 2011) was accordingly used to examine variation among the relationships between the variables of interest at Time One, Time Two and in score changes from Time One to Time Two.

As with Chapter Seven, in all analyses involving anger and PTSD Total and Arousal as variables, item 14 of the PCL (its sole anger item), was removed in order to eliminate redundancy in all equations involving both variables. Precautionary multiple regression analyses suggested that the Negative Automatic Thoughts Scale (NATS) had the capacity to operate as a proxy measure for depression and thereby
create issues of covariance capable of confounding the interpretation of the relationship between thought and PTSD. To guard against the potential for this, in all analyses where the NATS and PTSD total and avoidance scores were included as variables, items nine to twelve of the PCL were removed.

10.4 Results

10.4.1 Imagery, thought and PTSD: time one. Table 10.1 and Appendix L report the mean scores, standard deviations, significance and alpha levels for all scales for the total population and military and police population sub-samples of this study. They shows that as far as the study’s independent variables were concerned, the IPI-AV subscale scores were considerably higher than those previously reported by researchers (see Chapter Eight, Table 8.1 footnote). The NAT Scale scores were similarly substantially higher than those previously reported for clinical populations - for example, the previously cited mean score of 55.1 reported by Nezu and colleagues (2000) in a clinically depressed population. This was the case irrespective of gender or group membership, although the highest mean scores obtained on all such variables were those of ADF-affiliated participants.

In contrast, scores on the dependent variables were lower than previously reported for PTSD populations. This was true of PTSD scores - as illustrated by the score of 67.3 (SD = 9.7) reported for the PCL for Vietnam veterans by Creamer, Elliot, Forbes, Biddle and Hawthorne (2006). It was also the case for affect scores - as illustrated by the 14.8 (SD = 3.5) and 11.9 (SD = 4.0) respectively reported by Creamer and colleagues (2006) for the HADS-a and HADS-d in Vietnam veterans and the 32.5 (SD = 12.2) reported by Forbes and colleagues (2008) for the DAR scale in Vietnam veterans. This difference substantially reflected the effect of significantly lower scores on all scales by police participants, for whom there are neither normative nor comparable published research data. ADF-affiliated participant scores were, nevertheless, still noticeably lower than those previously reported (see table 10.1).

Pearson product moment correlations measuring the association of imagery predictor variables with the PTSD measures on the total population were positive, but typically low-to-moderate in magnitude (Table 10.2). Of the imagery subscales, the least strong imagery associations with PTSD measures were those for imagery anger and the strongest were typically those for vividness and absorption. Negligible correlations between all variables and alcohol abuse (the Audit) were evident. This
was similarly the case for age with correlations under .11 (ns) being obtained for all but HADS-d ($r = .22$, $p < .01$) and imagery frequency ($r = -.19$, $p < .01$) scores. Both audit scores and age were subsequently eliminated from all further Time One analyses. For negative affects, there was a clear pattern of association in anger’s stronger relationships with each of the imagery measures. Negative automatic thoughts obtained the highest correlations for each of the PTSD-related variables. This tended to reflect its relationship with PTSD avoidance ($r = .49$, $p < .01$).

Table 10.1

<table>
<thead>
<tr>
<th>Variables</th>
<th>Time One</th>
<th></th>
<th>Time Two</th>
<th></th>
<th></th>
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</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$(n = 232)$</td>
<td>$M$</td>
<td>$SD$</td>
<td>$(n = 152)$</td>
<td>$t$</td>
<td>$p$</td>
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<td>General Imagery Measure (GIM)</td>
<td>146.8</td>
<td>33.07</td>
<td>0.82</td>
<td>136.1</td>
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<td>Imagery Frequency (DDFS)</td>
<td>40.0</td>
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<td>0.91</td>
<td>36.7</td>
<td>10.8</td>
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<td>Imagery Absorption (DDAS)</td>
<td>55.1</td>
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<td>51.9</td>
<td>14.6</td>
<td>4.34</td>
</tr>
<tr>
<td>Imagery Vividness (DDVS)</td>
<td>24.0</td>
<td>8.2</td>
<td>0.88</td>
<td>22.2</td>
<td>8.8</td>
<td>16.70</td>
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<tr>
<td>Imagery Anger (DDHS)</td>
<td>27.7</td>
<td>10.5</td>
<td>0.92</td>
<td>25.3</td>
<td>10.3</td>
<td>9.89</td>
</tr>
<tr>
<td>Negative Thoughts (NATS)</td>
<td>78.9</td>
<td>29.3</td>
<td>0.98</td>
<td>75.4</td>
<td>31.6</td>
<td>3.36</td>
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<tr>
<td>Anxiety (HADS-a)</td>
<td>10.0</td>
<td>3.6</td>
<td>0.83</td>
<td>9.5</td>
<td>4.6</td>
<td>8.80</td>
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<td>3.8</td>
<td>0.80</td>
<td>11.4</td>
<td>4.1</td>
<td>42.23</td>
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<td>Anger (DARS)</td>
<td>27.2</td>
<td>13.0</td>
<td>0.91</td>
<td>24.7</td>
<td>13.6</td>
<td>10.90</td>
</tr>
<tr>
<td>Alcohol (Audit)</td>
<td>12.1</td>
<td>9.1</td>
<td>0.88</td>
<td>11.0</td>
<td>8.8</td>
<td>1.75</td>
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<tr>
<td>PTSD Overall (PCL-Tot)</td>
<td>56.0</td>
<td>13.0</td>
<td>0.92</td>
<td>52.0</td>
<td>16.7</td>
<td>16.60</td>
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<td>PTSD Intrusions (PLC-Int)</td>
<td>15.3</td>
<td>4.5</td>
<td>0.88</td>
<td>14.4</td>
<td>5.4</td>
<td>9.97</td>
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<td>PTSD Avoidance (PCL-Av)</td>
<td>22.9</td>
<td>6.2</td>
<td>0.82</td>
<td>20.7</td>
<td>7.3</td>
<td>18.31</td>
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<td>PTSD Arousal (PCL-Ar)</td>
<td>17.9</td>
<td>4.1</td>
<td>0.81</td>
<td>16.7</td>
<td>5.1</td>
<td>9.36</td>
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</table>

These results indicate imagery and thought were each significantly, but moderately, associated with PTSD overall. Drilling down to PTSD sub-cluster scores, all imagery subscales tended to be most strongly associated with PTSD avoidance. Imagery frequency and anger were less associated with PTSD and its symptom cluster.
scores. Such results also indicate that thought has a greater association with PTSD overall and in all its symptom clusters, especially avoidance.

These results indicate imagery and thought were each significantly, but moderately, associated with PTSD overall. Drilling down to PTSD sub-cluster scores, all imagery subscales tended to be most strongly associated with PTSD avoidance. Imagery frequency and anger were less associated with PTSD and its symptom cluster scores. Such results also indicate that thought has a greater association with PTSD overall and in all its symptom clusters, especially avoidance.

Although not an intended focus of this study, preliminary stepwise regression analyses testing the relationship between facets of imagery (frequency, vividness, absorption and angry imagery) and negative affects and PTSD highlighted different relationships with negative affects and PTSD and its subscales (see Appendices O & P). These analyses showed that imagery absorption was predictive of anxiety and depression, while imagery angry and frequency predicted anger.

Supportive of the study hypotheses, these initial analyses provided grounds for further exploration of the interaction of imagery and thought with PTSD. In light of both sets of information and, as previously justified, the possible interplay between affects (anxiety, depression and anger), imagery (frequency, absorption, vividness and anger), thought, population type and PTSD and its symptoms clusters, structural equation modelling (SEM) was applied to determine the causal relationships among the data sets.

Four observed, or directly measured, variables and a single latent variable were included. The observed variables were anger, thought, imagery and PTSD. The latent variable, group membership, was calculated from observed variables ADF-affiliated and police participant membership status. Given this latent construct was a grouping variable, a multi-group format of SEM was applied.
Table 10.2

**Time One Relationships of Imagery and Thought to Alcohol Abuse and PTSD measures (Pearson’s r) for the Total Population**

<table>
<thead>
<tr>
<th></th>
<th>DDFS</th>
<th>DDAS</th>
<th>DDVS</th>
<th>DDHS</th>
<th>NATS</th>
<th>HADS-a</th>
<th>HADS-d</th>
<th>DARS</th>
<th>Audit</th>
<th>PCL-Tot</th>
<th>PCL-Int</th>
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<td>General Imagery Measure</td>
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<td>.76**</td>
<td>.73**</td>
<td>.49**</td>
<td>.27**</td>
<td>.26**</td>
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<td>Imagery Frequency (DDFS)</td>
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<td>.07</td>
<td>.30**</td>
<td>.20**</td>
<td>.32**</td>
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<td>Imagery Absorption (DDAS)</td>
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<td>.59**</td>
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<td>.35**</td>
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<td>.49**</td>
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<td>PTSD Overall (PCL-Tot)</td>
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<td>.86**</td>
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<td>.66**</td>
<td>.63**</td>
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<td>PTSD Avoidance (PCL-Av)</td>
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<td></td>
<td>.63**</td>
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* p < .05, ** p < .01
Table 10.3

*Goodness of fit indices for structural equation model at time one, time two and from time one to time two*

<table>
<thead>
<tr>
<th>Fit Index</th>
<th>Time 1</th>
<th>Time 2</th>
<th>Change from T1: T2</th>
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<td>1.99</td>
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<td>.92</td>
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<td>TLI</td>
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<td>.06</td>
<td>.05</td>
<td>.07</td>
</tr>
</tbody>
</table>

Note: df = 69 for all time points, $p = .000$

Before interpreting whether the model adequately represented the data, it was assessed against various goodness of fit indices. Chi-square was used to determine any discrepancy between the data sample the model as the traditional means for evaluating overall model fit (Hu & Bentler, 1999). Given the size of the current sample was quite large, the chi-square to degrees of freedom ratio ($\chi^2$/DF; Wheaton, Muthen, Alwin & Summers, 1977) was also used. A non-significant result at a $p<.05$ threshold indicates a good model fit as the model accounts for the co variation among the variables well (Barrett, 2007). This test, however, is affected by sample size whereby the Chi-Square statistic nearly always rejects the model when a large sample is used (Bentler and Bonnet, 1980; Byrne, 2001; Jöreskog and Sörbom, 1993) and several other indices were accordingly used. These included the, Comparative Fit Index (CFI: Bentler, 1990), the Tucker-Lewis Index (TLI: Tucker & Lewis, 1973) and the Standardised root mean square residual (SRMR; Bentler, 1990) and the Root Mean Square Error of Approximation (RMSEA: Diamantopoulos & Siguaw, 2000). The TLI is acceptable where the model is not complex (Marsh, 1989). Values for the SRMR range from 0-1.0 with well fitting models obtaining values less than .05 (Byrne, 1998; Diamantopoulos and Siguaw, 2000), however values as high as 0.08 are deemed acceptable (Hu and Bentler, 1999). The RMSEA is regarded as one of the most informative fit indices (Diamantopoulos & Siguaw, 2000), due to its sensitivity to the number of estimated parameters in the model. It indicates how well the model, with unknown but optimally chosen parameter estimates, would fit the population’s covariance matrix (Byrne, 1988). It requires a value between .05 and 07 to indicate that the data fits the model well (Steiger, 2007). A value is below .05 suggests the model is saturated and above .07 indicates the data is not a good fit for the model (Hooper et al., 2008). The performance of these fit indices is presented in Table
10.3. It indicates that the model met critical value criteria for the CFI (.90 or better), RMSEA (under .08), TLI (above .90) and IFI (above .90). Hence, the model well-fitted the data.

Analysis of Time One data using SEM (see Figure 10.2) showed that anger had a strong, positive and direct causal relationship with PTSD (for the total sample, $\beta = .45$, $p = .001$). The regression equations (beta weights) for both ADF-affiliated and police participation were equal, indicating there was no differentiation in anger’s direct impact on PTSD on the basis of group membership. What specifically contributes to this relationship was untested and cannot be known for this and all subsequent SEM analyses which follow. The direct impact of anger was, however, particularly noteworthy.

Anger also had a moderate-to-large indirect causal impact on PTSD. In line with the hypothesised explanatory model, that impact was due to the independent operation of anger-determined imagery and thought pathways. The causal impact of anger on thought and imagery for the overall sample were significantly different when measured by defence to $z$ scores ($\beta = .28$ and .49, $p < .01$ respectively). There were also clear and significant differences between ADF-affiliated and police participants in terms of the effect of anger on imagery ($\beta = .59$ and .32, $p < .05$). Although there were differences between ADF-affiliated and police participants in terms of the effect of anger on thought, these differences were less prominent than those for imagery ($\beta = .33$ and .28) and were not significantly different when measured by $z$ scores.

Imagery had a direct causal impact on thought. This was affected by group membership, such that standardised beta weights were notably more elevated in ADF-affiliated participants ($\beta = .28$, $p = .03$) compared to police participants ($\beta = .46$, $p < .01$). Thought made a high predictive contribution to PTSD for police participants, but had a low and non-significant effect on PTSD for ADF-affiliated participants ($\beta = .40$ versus .17), the difference between the two being significantly ($p < .05$). The impact of imagery on PTSD was low or negligible and non-significant for both groups.

Together these direct and indirect anger paths explained 57 per cent of pre-treatment PTSD score variance. Thought accounted for twice the indirect impact of anger on PTSD compared to imagery (8% versus 4%). Its effect was much more prominent for police, while imagery was more predictive of PTSD among ADF-affiliated participants.
Table 10.4

*Standardised and unstandardised coefficients, standard error and significance values for model at time one (n = 232)*

<table>
<thead>
<tr>
<th>Observed variable</th>
<th>Standardised</th>
<th>Unstandardised</th>
<th>SE</th>
<th>C.R.</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anger → PTSD</td>
<td>.45</td>
<td>.12</td>
<td>.02</td>
<td>5.661</td>
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</tr>
<tr>
<td>Anger → Imagery</td>
<td>.49</td>
<td>.24</td>
<td>.04</td>
<td>6.276</td>
<td>***</td>
</tr>
<tr>
<td>Anger → Thought</td>
<td>.28</td>
<td>.63</td>
<td>.16</td>
<td>4.046</td>
<td>***</td>
</tr>
<tr>
<td>Imagery → Thought</td>
<td>.40</td>
<td>1.5</td>
<td>.36</td>
<td>5.158</td>
<td>***</td>
</tr>
<tr>
<td>Thought → PTSD</td>
<td>.30</td>
<td>.04</td>
<td>.01</td>
<td>3.914</td>
<td>***</td>
</tr>
<tr>
<td>Imagery → PTSD</td>
<td>.08</td>
<td>.04</td>
<td>.04</td>
<td>.987</td>
<td>.324</td>
</tr>
</tbody>
</table>

*** = p < .001

Table 10.4 reports the standardised and unstandardised beta weights, standard error and significance values for the model at Time One. It shows that anger unmediated by imagery or thought (i.e., in its direct association with PTSD) had the strongest relationship with PTSD. The indirect pathway between imagery and PTSD was not significant, indicating that the relationship between anger and PTSD was not mediated by imagery.

Table 10.5

*Squared Multiple Correlations for all Variables at Time 1, Time 2 and Time 1 Variables to Time 2 PTSD*

<table>
<thead>
<tr>
<th>Variables</th>
<th>Time 1</th>
<th>Time 2</th>
<th>Time 1:2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anger</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Thought</td>
<td>.24</td>
<td>.42</td>
<td>.24</td>
</tr>
<tr>
<td>Image</td>
<td>.25</td>
<td>.41</td>
<td>.26</td>
</tr>
<tr>
<td>PTSD</td>
<td>.48</td>
<td>.78</td>
<td>.33</td>
</tr>
</tbody>
</table>

The causal pathway between thought and PTSD was significant, albeit for police and not ADF-affiliated participants, and suggests that the relationship between anger and PTSD was partially mediated by negative thought. As shown in Table 10.5, the model explains 48% of the variance in Time One PTSD scores.
Table 10.6

Standardised Total Effects for Time 1, Time 2 and Time 1 to 2 Variables

<table>
<thead>
<tr>
<th>Pathways</th>
<th>Time 1 Standardised Total Effects</th>
<th>Time 2 Standardised Total Effects</th>
<th>Time 1 to Time 2 Standardised Total Effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anger → PTSD</td>
<td>.64</td>
<td>.81</td>
<td>.45</td>
</tr>
<tr>
<td>Anger → Thought</td>
<td>.49</td>
<td>.65</td>
<td>.49</td>
</tr>
<tr>
<td>Anger → Imagery</td>
<td>.50</td>
<td>.64</td>
<td>.51</td>
</tr>
<tr>
<td>Thought → PTSD</td>
<td>.30</td>
<td>.38</td>
<td>.38</td>
</tr>
<tr>
<td>Imagery → PTSD</td>
<td>.07</td>
<td>.25</td>
<td>.15</td>
</tr>
</tbody>
</table>

Figure 10.2  Model of anger’s direct and indirect (via imagery and thought) effect on PTSD at Time One

Legend:
V = Veteran
P = Police
W = Whole sample
Table 10.7

Relationships of Imagery and Thought to Alcohol Abuse and PTSD measures (Pearson’s r) for total population at time two

<table>
<thead>
<tr>
<th></th>
<th>DDFS</th>
<th>DDAS</th>
<th>DDVS</th>
<th>DDHS</th>
<th>NATS</th>
<th>HADS-a</th>
<th>HADS-d</th>
<th>DARS</th>
<th>Audit</th>
<th>PCL-Tot</th>
<th>PCL-Int</th>
<th>PCL-Av</th>
<th>PCL-Ar</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gen. Imagery Measure (GIM)</td>
<td>.74**</td>
<td>.92**</td>
<td>.79**</td>
<td>.74**</td>
<td>.54**</td>
<td>.46**</td>
<td>.56**</td>
<td>.57**</td>
<td>.05</td>
<td>.64**</td>
<td>.57**</td>
<td>.58**</td>
<td>.63**</td>
</tr>
<tr>
<td>Imagery Frequency (DDFS)</td>
<td>.6**</td>
<td>.42**</td>
<td>.36**</td>
<td>.38**</td>
<td>.38**</td>
<td>.52**</td>
<td>.42**</td>
<td>.06</td>
<td>.49**</td>
<td>.43**</td>
<td>.46**</td>
<td>.46**</td>
<td>.48**</td>
</tr>
<tr>
<td>Imagery Absorption (DDAS)</td>
<td>.66**</td>
<td>.60**</td>
<td>.44**</td>
<td>.44**</td>
<td>.54**</td>
<td>.51**</td>
<td>.00</td>
<td>.59**</td>
<td>.53**</td>
<td>.55**</td>
<td>.58**</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Imagery Vividness (DDVS)</td>
<td>.48**</td>
<td>.42**</td>
<td>.35**</td>
<td>.39**</td>
<td>.36**</td>
<td>.06</td>
<td>.51**</td>
<td>.49**</td>
<td>.45**</td>
<td>.44**</td>
<td>.47**</td>
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<td></td>
</tr>
<tr>
<td>Imagery Anger (DDHS)</td>
<td>.41**</td>
<td>.32**</td>
<td>.39**</td>
<td>.58**</td>
<td>.00</td>
<td>.49**</td>
<td>.45**</td>
<td>.44**</td>
<td>.47**</td>
<td>.44**</td>
<td>.47**</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Negative Thoughts (NATS)</td>
<td>.41**</td>
<td>.17</td>
<td>.49**</td>
<td>.11</td>
<td>.41**</td>
<td>.43**</td>
<td>.43**</td>
<td>.52**</td>
<td></td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>Anxiety (HADS-a)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>.69**</td>
<td>.61**</td>
<td>.09</td>
<td>.77**</td>
<td>.65**</td>
<td>.79**</td>
<td>.67**</td>
<td></td>
</tr>
<tr>
<td>Depression (HADS-d)</td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td>.65**</td>
<td>.13</td>
<td>.79**</td>
<td>.66**</td>
<td>.80**</td>
<td>.68**</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Anger (DARS)</td>
<td>.07</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>.83**</td>
<td>.74**</td>
<td>.78**</td>
<td>.81**</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Alcohol (Audit)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>.73**</td>
<td>.64**</td>
<td>.67**</td>
<td>.74**</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PTSD Overall (PCL-Tot)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>.14</td>
<td>.13</td>
<td>.15</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PTSD Intrusions (PCL-Int)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>.95**</td>
<td>.92**</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PTSD Avoidance (PCL-Av)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>.77**</td>
</tr>
</tbody>
</table>

*p < .05, **p < .01
10.4.2 Imagery, thought and PTSD: Time Two. Post-treatment, mean scale scores for all independent and dependent variables are reported in Table 10.1, while Appendix M reports Time Two mean scores on all variables for the study’s ADF-affiliated and police population samples. They show reductions in all scores compared to Time One overall and more so for police than ADF-affiliated participants. Consistent with these score decreases, and the absence of meaningful changes in standard deviations from Time One, there were fewer extreme scores at Time Two for both populations. These changes between Time One and Time Two scores are further commented upon in subsection 10.4.4.

Pearson product moment correlations measuring the association of imagery and thought predictor variables with the PTSD measures were positive and typically moderate-to-high in magnitude (Table 10.7). Negligible correlations between all variables and alcohol abuse (the Audit) were evident. This was similarly the case for age. Consistent with Time One analyses, both audit scores and age, on account of this, were subsequently eliminated from all further Time Two analyses. Of the imagery subscales included in the study, the weakest associations with PTSD measures were those for imagery frequency and anger. The strongest imagery-PTSD correlations were for absorption. Negative automatic thoughts obtained lesser correlations for PTSD Total, Intrusion and Avoidance scores, but not PTSD Arousal. For negative affects, imagery tended to have stronger associations with depression and anger. Thought was less correlated with anxiety, depression and anger compared to imagery. The differences between all such correlations were not significant and such differences existed as trends.

These results indicate that post-treatment imagery and thought were significantly and moderately-to-highly associated with PTSD overall. At the level of PTSD sub-cluster symptom scores, all imagery subscales tended to be most strongly associated with PTSD Arousal. They also indicate that thought had a greater association with PTSD overall and in all its symptom clusters, especially avoidance.

Analysis of Time Two data using SEM showed that anger had a strong direct causal relationship with PTSD (see Table 10.8 and Figure 10.3). The regression equations for ADF-affiliated and police participants (beta weights) differed significantly, indicating that the direct causal impact of anger on PTSD after treatment varied by group membership ($\beta = .48$ versus $\beta = .30$, $p = .01$) with police anger being
more impactful on PTSD. As for Time One, the direct impact of anger on PTSD was particularly noteworthy.

Again, as for time one, anger had a large and highly significant indirect causal impact on PTSD via imagery and thought as independent cognitive pathways. The impact of imagery and thought was increased from time one and was again significantly different for thought and imagery for the overall sample ($\beta = .44$ and $.62$, $p < .01$ respectively). Anger’s respective impact on these cognitive pathways differed substantially by group, with anger being more impactful for police compared to ADF-affiliated participants on thought ($\beta = .52$ versus and $\beta = .28$, $p = .01$) and particularly imagery ($\beta = .72$ versus $.40$, $p = .01$).

Both indirect paths maintained a causal pathway to PTSD, more so for thought than imagery. Thus, thought and imagery respectively determined 36 and 26 per cent of post PTSD score variance for the total sample. Importantly, there was a clear group distinction in the amount of PTSD variance explained by thought, with the thought of ADF-affiliated participants being impactful on PTSD ($\beta = .44$, $p < .01$) compared to police ($\beta = .29$, $p = .002$). Both police and ADF-affiliated participants PTSD scores were impacted upon by imagery ($\beta = .23$ and .30, $p = .04$), but were not significantly differentiated.

Table 10.8

<table>
<thead>
<tr>
<th>Observed variable</th>
<th>Standardised</th>
<th>Unstandardised</th>
<th>SE</th>
<th>C.R.</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anger → PTSD</td>
<td>.40</td>
<td>.14</td>
<td>.03</td>
<td>5.185</td>
<td>***</td>
</tr>
<tr>
<td>Anger → Imagery</td>
<td>.62</td>
<td>.32</td>
<td>.05</td>
<td>6.321</td>
<td>***</td>
</tr>
<tr>
<td>Anger → Thought</td>
<td>.44</td>
<td>1.02</td>
<td>.21</td>
<td>4.953</td>
<td>***</td>
</tr>
<tr>
<td>Imagery → Thought</td>
<td>.33</td>
<td>1.46</td>
<td>.43</td>
<td>3.400</td>
<td>***</td>
</tr>
<tr>
<td>Thought → PTSD</td>
<td>.36</td>
<td>.05</td>
<td>.01</td>
<td>4.887</td>
<td>***</td>
</tr>
<tr>
<td>Imagery → PTSD</td>
<td>.26</td>
<td>.17</td>
<td>.05</td>
<td>3.209</td>
<td>.001</td>
</tr>
</tbody>
</table>

***=p<.001
Imagery partially determined thought for both groups. The overall contribution was 33 per cent ($p < .01$) and there was little difference between police and ADF-affiliated participants ($\beta = .31$ versus $\beta = .32$), although the ADF-affiliated participant beta weight was non-significant. Together these direct and indirect anger paths explained 72 per cent of the post-treatment PTSD score variance. Thought and imagery explained equal amounts of this effect and imagery explained 14 per cent, with imagery’s effect being more prominent for police and thought more so for ADF-affiliated participants. Notably, as shown in Table 10.5, the model explains 78% of the variance in Time Two PTSD scores.

Table 10.8 reports the standardised and unstandardised beta weights, standard error and significance values for model at Time Two. It shows that anger unmediated by imagery or thought (i.e., in its direct association with PTSD) had the strongest relationship with PTSD. The indirect causal pathway between imagery and PTSD was significant for the whole sample, indicating that the relationship between anger and PTSD was mediated by imagery. The indirect causal pathway between thought and PTSD was similarly significant for the whole sample, suggesting that the relationship between anger and PTSD was partially mediated by negative thought for both ADF-affiliated and police participants. As shown in Table 10.5, the model explains 78% of the variance in Time Two PTSD scores.
10.4.3 Imagery, thought and PTSD: change from time one to time two.

Post-treatment, mean scale scores for all independent and dependent variables decreased relative to pre-treatment scores (Figure 10.4 and Table 10.1). Significant, but clinically low-to-moderate score changes applied to all variables bar depression. The greatest changes ($d = .25$ and above) were those for Total PTSD scores ($d = .27$) and its sub-factors of avoidance ($d = .32$) and arousal ($d = .26$), overall imagery scores ($d = .31$).
Appendix M reports means scores on all variables at Time Two for the study’s ADF-affiliated and police population samples. As for Time One, that data demonstrates that significant differences between ADF-affiliated and police participants existed across the entirety of variables, with a clear trend to ADF-affiliated participants scoring higher than police participants and non-Vietnam scoring highest ADF-affiliated participants.

Comparison of time one versus time two data using SEM showed that anger had a direct causal relationship with PTSD (See Table 10.9 and Figure 10.5). The strength of this predictive relationship was low (β = .18, p = .05) compared to both Time One (β = .45, p < .01) and Time Two (β = .40, p < .01), suggesting that a meaningful decline had occurred in the direct contribution of anger on PTSD. There was no difference in groups in this anger-PTSD contribution to and approximately the same for study participants with ADF-affiliated and police participants (β = .18, p =
Anger-in-PTSD: The Role of Visual Imagery

.05 versus $\beta = .18, p = .05$), suggesting group membership had no effect on direct anger to PTSD causal relationships.

There was also an indirect causal relationship of anger on PTSD. In line with predictions, this operated via independent imagery and thought pathways. The causal impact of anger on thought for the overall sample was low and approximately the same ($\beta = .17$ and $.14, p < .01$ respectively) for ADF-affiliated and police participants. In contrast, imagery was noticeably more affected by anger and there were clear differences between ADF-affiliated and Police participants ($\beta = .54, p < .01$ and $.33, p = .001$ respectively).

Both indirect paths persisted causally to PTSD for ADF-affiliated participants. This was more the case for thought ($\beta = .42, p = .001$) than imagery ($\beta = .35, p = .01$). Thought also determined PTSD for police ($\beta = .39, p = .002$), but not imagery ($\beta = .07, ns$). The strength of these associations was such that thought for the whole sample determined thirty nine per cent of PTSD score variance at Time two ($p < .01$). Imagery, in contrast had a low, non-significant impact on the change from Time One to Time Two PTSD scores ($\beta = .14$).

Imagery had a direct causal, but partial, impact on thought, with this impact being substantially greater in police ($\beta = .46, p < .01$) versus ADF-affiliated participants ($\beta = .28, p = .03$). Thought clearly mediated the impact of imagery on anger in police participants, given imagery’s impact did not continue through to PTSD.

Together anger directly and indirectly explained 33 per cent of the PTSD score change from time one to time two. Thought and imagery explained approximately equal amounts of this variance (7% and 6%). The amount of variance indirectly explained approached that directly explained (13% versus 19%) with more PTSD variance being in police by imagery and more PTSD variance being explained in ADF-affiliated participants by thought.

Table 10.9 reports the standardised and unstandardised beta weights, standard error and significance values for the model at Time One to Time Two. It and Figure 10.5 show that anger unmediated by imagery or thought (i.e., in its direct association with PTSD) had the strongest relationship with PTSD. The indirect pathway between imagery and PTSD was not significant, indicating that the relationship between anger and PTSD was not mediated by imagery. The pathway
between thought and PTSD was significant; suggesting the relationship between anger and PTSD was partially mediated by negative thought.

Table 10.9

<table>
<thead>
<tr>
<th>Observed variable</th>
<th>Standardised</th>
<th>Unstandardised</th>
<th>SE</th>
<th>C.R.</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anger → PTSD</td>
<td>.18</td>
<td>.07</td>
<td>.04</td>
<td>1.947</td>
<td>.05</td>
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<td>Anger → Imagery</td>
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<td>.24</td>
<td>.04</td>
<td>6.304</td>
<td>***</td>
</tr>
<tr>
<td>Anger → Thought</td>
<td>.28</td>
<td>.63</td>
<td>.16</td>
<td>4.010</td>
<td>***</td>
</tr>
<tr>
<td>Imagery → Thought</td>
<td>.40</td>
<td>1.85</td>
<td>.36</td>
<td>5.156</td>
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</tr>
<tr>
<td>Thought → PTSD</td>
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<td>.06</td>
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<td>***</td>
</tr>
<tr>
<td>Imagery → PTSD</td>
<td>.16</td>
<td>.12</td>
<td>.08</td>
<td>1.516</td>
<td>.13</td>
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</tbody>
</table>

***=p<.001

Table 10.10 reports the standardised indirect effects for all variables on PTSD at times one and two and from Time One to Time Two. As an overall summary of the results, it indicates that due to the non-significant pathway between imagery and PTSD at Time One, anger did not have an indirect effect through imagery on PTSD. This indirect pathway represented a lesser effect on PTSD compared to the direct anger pathway. At Time Two, anger had an indirect effect on PTSD through imagery and thought. Both pathways again had lesser effects on PTSD compared to the direct effect of anger on PTSD. The indirect effect of anger on PTSD through thought strengthened slightly in Time Two, compared to Time One.
Figure 10.5  Model of anger’s direct and indirect (via imagery and thought) effect on PTSD for Time One to Time Two Change
Table 10.10

*Standardised Indirect Effects for Time 1 Anger Through Variables on Time 1 PTSD and for Time 2 Anger Through Variables on Time 2 PTSD*

<table>
<thead>
<tr>
<th>Pathways</th>
<th>Time 1 Standardised Indirect Effects</th>
<th>Time 2 Standardised Indirect Effects</th>
<th>Time 1:2 Standardised Indirect Effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anger → Thought → PTSD</td>
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<td>.44 x .36 = .16</td>
<td>.28 x .37 = .10</td>
</tr>
<tr>
<td>Anger → Imagery → PTSD</td>
<td>-</td>
<td>.62 x .26 = .16</td>
<td>.49 x .16 = .14</td>
</tr>
<tr>
<td>Anger → Imagery → Thought → PTSD</td>
<td>.49 x .40 x .30 = .06</td>
<td>.62 x .33 x .36 = .13</td>
<td>.49 x .40 x .37 = .07</td>
</tr>
</tbody>
</table>

10.5 Conclusion

This study cross-sectionally and longitudinally explored the relationship of anger and imagery in PTSD in treatment-seeking populations of ADF-affiliated and police participants who had undergone programmatic treatment for PTSD. Via this approach, it tested an explicit model of anger in PTSD. In doing so, it sought to answer several questions. The first related to the extent to which anger was associated with PTSD. The second was concerned with the extent to which anger’s relationship with PTSD was impacted upon by imagery and thought. The third and final question was focused on whether there was inter-relationship between anger and imagery or facets of imagery and thought in anger in PTSD.

Findings pertaining to each question are now summarised, preparatory to a more detailed consideration of the meaning of such results and their theoretical and clinical implications in the concluding general discussion (Chapter Eleven). The strengths and weaknesses of the study’s design and possible future research directions will also be detailed in that chapter.

Time one data emphasised the central, direct relationship of anger to PTSD irrespective of population. Analyses also showed that there were indirect pathways from anger to PTSD that operated via imagery and thought. Importantly, these analyses demonstrated that this indirect element of anger’s relationship to PTSD differs on account of cognition and PTSD type. Thus, overall imagery tends to regress more on anger compared to thought and ADF-affiliated participants are more likely to have a stronger relationship with imagery in their anger compared to police participants when PTSD at intake. A differential relationship for anger and thought...
was not established. In turn, while a mediating effect of thought on imagery existed for both groups, this was particularly so for police participants, to the extent that thought for them determined PTSD. Overall at Time One, the model accounted for almost half of the variance in PTSD. This emphasises the importance of both thought and imagery to anger in PTSD when untreated.

Time two data, which for 75% per cent of the sample (65% of ADF-affiliated and 100% of police participants) represented contemporaneous treatment outcome data, supported and extended this picture. Initially, it showed that post-treatment, police participant anger had a significantly greater impact on PTSD. This was the case directly and indirectly via the predicted imagery and thought cognition pathways. For police there was a greater impact of anger on imagery and thought and this impact was particularly acute for imagery. It secondly showed that the difference between the direct and indirect impact of anger on PTSD was reduced, suggesting that imagery and thought as indirect causal pathways become more important determinants of PTSD post treatment. It finally also showed that group membership had an important impact, with ADF-affiliated participants demonstrating significantly higher thought-PTSD and imagery-PTSD regressions. Interestingly, as far as thought was concerned, this was the reverse of time one outcomes. Overall at Time Two, the model accounted for more than three quarters of the variance in PTSD scores. This highlights the importance of thought and imagery in the treatment of anger in PTSD.

Analysis of data relating to change from Time One and Time Two scores emphasised a direct, but compared to Time One and Time Two diminished direct relationship of anger to PTSD. SEM analyses demonstrated that there are indirect pathways from anger to PTSD that operate via imagery and thought and importantly demonstrated that this indirect element of anger’s relationship to PTSD differs on account of cognition and group membership. Anger tends to much more strongly determine imagery compared to thought in changes from time one to time two scores. ADF-affiliated participants also appear more inclined to experience anger-dependant imagery. The impact of anger-associated imagery on thought was significant for both groups, albeit noticeably greater for police. Thought in turn mediated imagery’s role, rendering the latter’s impact on PTSD insignificant for police. Thought and, less so, imagery remained important predictors of anger’s impact on PTSD for ADF-affiliated participants. Overall for change in scores from Time One to Time Two, the model accounted for one third of the variance in PTSD scores. Interestingly, the indirect
paths of anger to PTSD via imagery and thought accounted for a greater amount of PTSD variance compared to the direct route. This highlights the probability that altered cognitions are critical to the amelioration of PTSD.

Overall, this study confirmed the view, as expressed in Chapter Seven, that the best conceptualisation of the relationship of imagery to anger in PTSD is one in which dysregulated anger is understood as causally linked to dysregulated imagery. This understanding emphasises that imagery, but also thought, which is increased in the context of psychopathology, rather than the reverse. It fits well with the stress-diathesis explanation of PTSD, anger and anger in PTSD described across Chapters Two to Seven. The outcomes of this study are further discussed in the next, and final, chapter of this thesis, which presents the overall theoretical and clinical implications of this research program.
CHAPTER 11: GENERAL DISCUSSION

Nothing is so strong as gentleness; nothing is so gentle as real strength
Francis DeSales

This concluding chapter synthesises and contextualises the outcomes of this research program as established across the preceding three chapters. It does this by specific reference to the aims and hypotheses articulated in Chapter Seven. Thereafter, it discusses the key outcomes of this thesis for its theoretical, clinical and research implications and outlines its limitations.

11.1 Synthesising and Contextualising the Outcomes of This Research

11.1.1 Anger is strongly predictive of PTSD. The first of the two broad aims of this research program was to assess nature of the relationship of anger to PTSD. In doing so, this research sought to answer questions about the intensity of that relationship, whether it could be clarified by contrasting it with the relationship of PTSD to anxiety and depression, the impact of change in anger on PTSD and the directionality of any change relationship. Associated with these objectives, several hypotheses were tested.

The present findings provide support for the initial hypothesis (see Chapter Seven sub-section 7.4.1) that anger would be associated with PTSD. Studies one and three demonstrated unequivocally that anger has an important association with PTSD. The results of the first study showed that anxiety, anger and depression were equally associated with PTSD cross-sectionally. That study, however, confirmed that anger was the major affective predictor of change in PTSD. In it, when pre-treatment PTSD and negative affect were included in stepwise regression analyses as predictor variables of post-treatment PTSD, pre-treatment PTSD was the dominant predictor and anger the sole subsidiary predictor of post-treatment PTSD. Although anxiety and depression were more strongly associated with PTSD cross-sectionally compared to anger, neither played a predictive role in relation to post-treatment PTSD. A subsequent pre-post regression analysis run without PTSD as a predictor identified anger as a more powerful predictor of PTSD and its sub-clusters than the other negative affects. Anxiety entered the regression equations for PTSD intrusions and
avoidance, while depression did so for avoidance. In all cases, however, the contributions of anxiety and depression were less than that of anger.

In combination, the results of Study One and the SEM analysis of Study Three confirmed the predictive role of anger in different traumatised populations. Vietnam veteran participants, post-Vietnam veterans and current ADF-affiliated participants and current and former police participants each demonstrated evidence of a strong positive association of anger with PTSD. These findings confirmed the third hypothesis that anger would be a strong predictor of PTSD as measured by change in response to treatment.

Such outcomes align with previous research. The finding that anxiety predicted both intrusive and an arousal characteristic was consistent with literature which emphasises the connection of anxiety to intrusive phenomena in feared traumatic memories (see Foa et al., 1989). That anger is similarly predictive of intrusions and arousal, was consistent with research showing that PTSD’s arousal symptom cluster, because of its anger component, was the predominant predictor of PTSD severity overall, and on account of its substantial impact on PTSD intrusive phenomena (Schell et al., 2004; Shea et al., 2013; Weston, 2014). That depression predicted avoidance is in turn consistent with the DSM IV conception of PTSD and the factor analytic studies which underpin the four factor DSM 5 conceptualisation of the disorder, which have separated the DSM IV avoidance subcluster into emotional numbing and effortful avoidance (Friedman, 2011; & Friedman, Resick & Keane, 2014).

Some study outcomes, however, represent important qualifications of existing knowledge or new understandings. The finding in the first study that anger was, after pre-treatment PTSD severity, the second most important predictor of post-treatment PTSD severity is important and further supports previous research emphasising the relationship between anger and PTSD. It adds to the established understanding that anger has clear aetiological and treatment implications in PTSD, acting, as it does, as a key factor in PTSD’s development and a moderator of PTSD severity and change (Andrews et al., 2000; Forbes et al., 2008; Frueh et al., 1997; McHugh et al., 2012; Orth & Maercker, 2009). While Novaco and Chemtob (2002) reported that 40 per cent of PTSD score-variance was due to change in the single anger-related item of the PCL, the present research found somewhat weaker associations (anger predicted 25% of post treatment PTSD variance in the presence of
the pre-treatment PTSD score and 34% in its absence). Further, the SEM analysis of Study Three separately and strongly highlights the causal impact of anger on PTSD levels of symptoms before and after treatment and in score changes due to treatment. That anger alone, and neither anxiety nor depression, predicted PTSD change was unexpected and was a novel, important outcome.

11.1.2 Anger in PTSD varies according to occupational grouping and the nature of traumatic experience. Potentially traumatic events are a common experience and may take a near-infinite number of forms. This was detailed in Chapters Two and Four. Also as described in Chapter Two, discrete community groups are differentially exposed to PTEs in frequency and type, occupational group being a prime example. This research compared military and police personnel - two groups with distinct functional roles who are exposed to heavy and overlapping, yet substantially different types of traumata - and sought to test the effect of occupational grouping and the nature of trauma experienced (combat versus policing trauma) on anger in PTSD. It was also interested in the intrinsic question of how imagery connects to affect generally and sought to contrast its operation in traumatised and non-traumatised populations.

Consistent with predictions, anger scores were greater in the PTSD population samples compared to its community population sample. Notably, and again consistent with the prediction that anger would be correlated strongly with PTSD, especially where there was moral injury, within the PTSD population samples, scores were consistently higher for participants with military service. This higher score pattern among military-affiliated participants was also true of anxiety, depression and PTSD scores.

This first outcome is consistent with established research. It supports the historical finding that anger is more appropriately regarded as a “property” of PTSD and not the consequence of military combat (Lasko et al., 1994). To recall from Chapter four, anger is substantially related to PTSD among samples that have experienced all possible types of traumatic events (Orth & Wieland, 2006).

The second outcome is a novel finding enabled by the comparison of different PTSD populations. It shows that while anger is critically important to the PTSD of both ADF-personnel and police, its impact is greater in those with military trauma. This raises the possibility that although anger is an important element of PTSD per se, its intensity may vary in line with stressor type.
This is taken into consideration by McTeague and colleagues (2010), who reported that PTSD’s physiological reactivity varies according to trauma magnitude and whether discrete or multiple traumata are involved. They specifically propose that discrete traumata are associated with PTSD characterised by increased defensive reactivity to aversive imagery. Cumulative, higher magnitude traumata, are marked by PTSD characterised by higher anxious and depressive morbidity and a blunted affective response. The findings of this research qualify this perspective by suggesting that it is the meaning of traumatic events and not their dose which has primacy in the determination of traumatic response. To illustrate, while there is an overall absence of incidence data about police trauma exposure, compared to military personnel, police are considered to have among the highest rates of exposure to serious violence, rape, death and anti-social behaviour (Mayhew, 2001). Anecdote also suggests that the rate of death exposure for a thirty year career police officer may be in the order of 200 hundred cases (personal communication between the author and various senior Victorian police). Such exposure levels are unlikely to be typically exceeded by military personnel across the duration of their service. The comparative affect and PTSD scores of military and police participants in this research indicate that it is those with trauma of a military origin who have higher scores on all such indices, regardless of the time point at which they were measured and that the amount of exposure to serious traumatic events may be unlikely to explain the differences observed. From this it may be inferred that exposure dose per se cannot account for the higher levels of anger that occur in PTSD.

A qualification of the position of McTeague and associates that emphasises human event causation, the potential recklessness and dangerousness of such action(s) and the extent to which they transgress social norms (Digiuseppe & Tafrate, 2001; Hutcherson & Gross, 2011) provides a compelling explanation of anger in PTSD. This is arguably due to the presence of malevolent, morally- culpable behaviour in anger’s downstream occurrence, and its well-known connection to affects of repulsion (e.g., horror, disgust and outrage) and responsibility (e.g., guilt and shame) (see Lemerise & Dodge, 2008). The outcomes of this research pointing to the importance of trauma type are in accordance with that proposition. This is because, although police have among the highest rates of exposure to PTEs, that exposure is not always about death and very rarely involves the perpetration of killing. The data suggest that the impact of the volume and significance of policing traumata does not match the
impact of the moral dimensions involved in the killing central to war (Grossman, 1996). Warfare is the only environment where killing is legitimised as a part of routine behaviour, and although police exposure to trauma is not uncommonly about death, it rarely involves the perpetration of legal killing. In their comprehensive review of combat-related PTSD and issues of morality, Litz and associates (2009) offered a key insight into persisting PTSD, observing that available evidence demonstrates killing and wounding are strongly associated with PTSD and are better predictors of it than combat exposure per se. Contrary to sensationalised accounts of human nature, killing is the antithesis of human values, and purpose-specific, intense and prolonged training (e.g., in military organisations) or prolonged exposure to brutality in criminogenic environments is required to overcome the innate human disinclination to kill (Grossman, 1996). Despite the effectiveness of this training - and it is clear Western military forces are becoming more effective in raising the killing efficiency of their troops (Grossman, 1996) - those who kill militarily typically express irritable remorse and distress at their lethality and having to carry out lethal actions post-event, especially when they are no longer part of the military family.

11.1.3 Anger is linked to imagery and thought which subsequently mediate its relationship with PTSD. The second of the two broad aims of this research program was to explore the role of imagery in negative affect generally and within the context of PTSD. In doing so, this research sought to answer questions about the strength of relationship between imagery and anger, the comparative relationship between imagery and anxiety and depression, the role of visual imagery in determining anger-related PTSD patterns and the directionality of those relationships.

Consistent with experimental predictions, the findings of the studies involved demonstrate that ADF-affiliated participant participants scored highest on all imagery, thought and affect variables, with non-Vietnam ADF-affiliated participants in Study Three attaining the highest scores overall. Community population participants scored lowest and police attained intermediate scores on all such variables (for ease of comparison these scores are drawn together in Appendix L). This pattern of imagery, thought, affect and PTSD scores confirm the hypothesised impact of traumatic stress and traumatisation type on affect and the twin cognitive processes of imagery and thought (see Chapter seven, sub-section 7.4.2).
The findings of the third study demonstrate that, in line with the initial hypothesis, which predicted a positive relationship between imagery and anger in PTSD, imagery and thought influenced anger and PTSD via two separate but inter-related indirect pathways. The relationships between anger, imagery and PTSD were moderately high. Of these pathways, anger affected imagery more than thought, albeit differentiated by occupational grouping and the consequent nature of the trauma experienced by those groupings.

Pre-treatment military-affiliated participants were more likely than police to experience anger that predicted imagery and, to a lesser extent, thought. As imagery, thought, anger and PTSD scores decreased as a consequence of treatment, however, the reverse pattern applied. That is, police were more likely to experience anger that predicted imagery and thought compared to ADF-affiliated participants. This pattern held irrespective of mean post-treatment score levels, which were lower than their pre-treatment variable equivalents. Simultaneously, the whole sample difference between the strength of prediction of anger on imagery and thought declined from the pre-treatment level. These results suggest that as treatment effects occur, and anger, thought, imagery and PTSD scores decline - as they did for the entire participant sample in Study Three - the predictive effect of anger on imagery becomes greater than for thought.

The explanatory model of anger in PTSD tested in this research sought to address a number of unanswered questions, especially around the directionality of the relationships between anger and PTSD, and between anger and imagery and thought in PTSD. It also focused on the direct and, via imagery and thought, indirect contribution of anger to PTSD. As SEM defines the relationships between variables, it was therefore the ideal analytic device for assessing the predictive impact of anger, imagery and thought on PTSD, and determining the directionality of the relationships involved (MacCallum & Austin, 2000).

Arising from the relationships among anger, imagery and thought, Study Three’s SEM analysis suggests several possibilities with regard to PTSD. Pre-treatment data suggest that negative thought in police predicts PTSD. It further suggests that due to the mediation of imagery by thought (see sub-section 11.1.5), when PTSD is untreated, there is a mild-to-moderate, but statistically non-significant, relationship between imagery and PTSD in veterans but not police. Post-treatment, this picture alters. In the context of reduced scores for both independent and
dependent variables, thought had a slightly increased effect on PTSD. Imagery joins it as a predictor of PTSD for police. This pattern of imagery and thought predicting PTSD is repeated for score change data from pre-to-post treatment.

Post treatment, the mediating effect of thought on imagery was reduced, albeit largely due to the lesser regression of thought on imagery in police. Consequently, in police imagery did not maintain a direct predictive pathway to PTSD, but had a non-direct pathway determined by thought.

Similarly, when change scores from pre-to-post treatment were examined, this effect of imagery only remained for veterans. Again the role of imagery was reduced by thought. It became the primary pathway to PTSD for both groups, while imagery also had a direct, but lesser pathway for veterans only.

These outcomes are novel and constitute potentially important additions to the literature. The implications for theory and practice are described in sub-section 11.2.

11.1.4 Different facets of imagery are linked to different negative affects and in PTSD are influenced by occupational grouping and the nature of traumatic experience. The high correlations of imagery and negative thought with all negative affects in studies two and three, and the decrease in all such scores from pre-treatment to post-treatment in Study Three, emphasise the impact of these twin elements of cognition on affect levels. In line with predictions emphasising the specificity of affect-imagery characteristic relationships, the data in Study Two established different imagery-affect patterns in a non-clinical sample. It established that imagery vividness is associated with anxiety, imagery absorption with depression and angry imagery with angry affect.

These outcomes nuance earlier findings about imagery. Their implications for theory and practice are further detailed in sub-section 11.2.

11.1.5 Thought mediates the effect of imagery on negative affect and on anger in PTSD. The overall aim of this final element of this research project was to compare the role of thought in negative affect generally and within the context of PTSD. Three specific objectives were addressed. The first focused on the strength of the relationship between anger and thought in a general population. As part of this objective, that relationship was contrasted with those pertaining to the negative affects of anxiety and depression. The second objective contrasted the impact of imagery on anger with that of negative thought. As part of this objective, the inter-relationship of
such imagery and thought impacts was investigated. Finally, this research sought to examine these impacts generally and in the context of PTSD.

In a subsequent mediating relationship, anger-related imagery determined anger-related thought irrespective of intensity. There was a strong pre-treatment mediating effect of thought on imagery for both ADF-affiliated participants and police participants. As scores for all variables declined post-treatment, this becomes a smaller, partial effect, such that in the police sample there remained a direct route from imagery to PTSD accounting for 25 per cent of PTSD score variation. Post-treatment, the effect of thought on PTSD was significant for both groups, with a stronger effect in ADF-affiliated participants than police. Pre-treatment, thought strongly determined PTSD in police, but was not related to PTSD in the ADF-affiliated samples.

As noted in sub-section 11.1.4, the second study of this research demonstrated that facets of imagery were differentially associated with negative affects in a non-clinical population. It also showed that these imagery-affect relationships were mediated by thought.

11.1.6 Non cognitive factors must be accounted for in any consideration of imagery’s role in anger in PTSD. Although not a central focus of this research, as outlined in Chapter Six (see sections 6.1, 6.2 and 6.3) the operation of visual imagery is subject to the effect of various demographic and individual capacity-related factors. The data gathered in this research allows some observations to be made about the operation of two such factors.

The first pertains to the effect on imagery of gender. The outcomes of this research indicated female participants scored higher than men on imagery indices related to frequency, vividness and absorption, but less than men with respect to anger in imagery. Thus the women in this research imaged more frequently, had more vivid imagery and were more absorbed in it, but not angrily so. These differences were consistent across population samples. In addition, imagery levels were exacerbated in the presence of PTSD, and women with PTSD scored higher on all imagery variables compared to women in the community sample. Gender-based differences also applied to other variables measured and women outscored men on all outcome variables bar anger scores.

A second interesting finding pertains to the data relating to age. It showed a slight inverse relationship between imagery levels and age in the (university student)
population of Study Two. Thus younger people imaged more than older people. This also applied irrespective of gender and participant group. This is consistent with the existing literature which shows the capacity for imagery declines across age (Kosslyn, 1994; Giambra, 1999; 2000b). Interestingly, in Study Three, the opposite applied. Thus while the ages of the ADF-affiliated and police participants were substantially different (58.5 versus 48.4), imagery was higher in the former, older population.

It is possible to speculate as to why gender and age affect the manifestation and maintenance of imagery. With respect to gender, the data is consistent with research indicating women have superior imagery ability to men (Giambra, 1999; 2000b). It also reflects the well-established research findings that women have double the prevalence of PTSD compared to men (APA, 2013) irrespective of trauma type. Due caution is required in interpreting this data, however, given the low numbers of female participants in Study Three ($n = 13$). The results of Study Two (with 150 women and 47 men participants) are less equivocal however and emphasise the non-pathological tendency of women to image. Given the connection of imagery to PTSD, it is reasonable to infer that the increased proclivity for imagery in women may render them at greater risk for pathology in the presence of sufficient stress. This has implications for treatment of anger, anger in PTSD and PTSD itself in women.

With regard to the divergent age-related outcomes, there are at least five explanatory possibilities. Briefly, there is the prospect that the decline may be reflective of basic age-related organic neurological decline, in the neuronal loss referred to by Giambra (1999; 2000b). Second, it is possible that the decline in imagery with age does not occur at a linear rate, and may for example, degrade quicker in the third, fourth and fifth decades of life. Third, it is possible that with intellectual development and maturation, individuals simply become less reliant on imagery and increasingly engage with the more sophisticated lexical cognitive processes involved in thought. Fourth, the increase in imagery for the PTSD populations may be explained by the tendency for imagery to improve with practice. An example of this is Grigsby's observation (1987) that imagery fluency in Vietnam veterans with PTSD is greater on account of intrusion repetition. Finally, it is possible, as emphasised above, that the meaning-based core of imagery in traumatic stress prolongs and amplifies imagery function(s).

Regardless of the ultimate explanation, the relationship between age and imagery has implications for treatment of anger, anger in PTSD and PTSD itself. This
is because while the efficacy of imagery-based treatments like prolonged exposure for PTSD centres on the treatment recipient’s capacity to engage with the image, an age-related transition from imagery to the word-based information processing has implications for imagery engagement. The possibility that imagery-based treatments may be calling on a diminishing imagery capacity while PTSD amplifies imagery fluency needs to be considered in the treatment of anger in PTSD and PTSD itself.

11.2 Implications of this Research

The outcomes of this research have implications for the conceptualisation, research and treatment of anger in PTSD and, ultimately, the treatment of PTSD. These implications are both theoretical and clinical in nature.

11.2.1 Theoretical implications

11.2.1.1 Anger strongly impacts on PTSD. This research has implications for how anger in PTSD may be understood. Its initial study demonstrated that anger was significantly associated with PTSD, and predicted the efficacy of its treatment. In contrast, the common PTSD comorbidities of anxiety and depression, while strongly associated with PTSD cross-sectionally, had a smaller predictive effect on PTSD treatment outcome and none when pre-treatment PTSD scores were included as predicted.

Consistent with the existing literature, the first of these outcomes argues for conceptual and empirical recognition of the central role played by anger in PTSD, particularly in male ADF-affiliated participants and emergency service populations. Researchers have noted the prevalence of anger in the epidemiology of PTSD (Power & Fyvie, 2013). The reconceptualisation of PTSD as a trauma and stress disorder, rather than anxiety disorder, in DSM 5 is recognition that the syndrome’s character is not exclusively anxious in nature. This buttresses the emerging picture of PTSD as being not only profoundly anger influenced, but possibly characterised by dysfunctional anger in the majority of PTSD presentations (see Weston, 2014) and the ongoing conceptualisation of PTSD in terms of its overall dimensional structure (Armour et al., 2015).

The second outcome was expected on the basis that while anger has been marginalised as a predictor of PTSD, it has great relevance to the intensity and prolongation of PTSD. Anxiety and depression are significant comorbidities of PTSD.
This research emphasises the need for the predictive contribution of anger to be recognised alongside them.

11.2.1.2 Occupational grouping and the nature of traumatic experience in PTSD. This research discovered that the predictive impact of anger on PTSD varies according to occupational grouping and the nature of traumatic experience, with current serving military participants and ADF-affiliated participants with recent service displaying greater anger levels and lesser treatment gains than police. This is important and extends the initial finding of the central role of anger in PTSD, by indicating that, depending on the nature of the traumatisation, the impact of anger will vary. As noted above and in Chapter Four, where humans are involved in (or are witness to) actions involving the contravention of social norms and particularly where that behaviour is malfeasant or morally repugnant, or as McNally, (2006) terms it, malignant, there is likely to be an increased anger reaction. That angry reaction is, in turn, critical to the ongoing maintenance of PTSD. A model of PTSD which takes into account the role of anger and the effect of precipitating events on such anger will facilitate theoretical and research development. This research presented data consistent with this possibility.

11.2.1.3 Imagery and thought mediate anger’s relationship with PTSD. Overall, this research demonstrated that imagery is related to negative affect, particularly anger in PTSD. Its second study demonstrated connections between imagery, negative affect and anger in a general population and the third study showed that connection in different PTSD populations. As with anger, this relationship with imagery differed by occupational grouping and the nature of trauma experienced by those groups. Study two showed that different facets of imagery were associated with different negative affects in a community sample. Although not a specific focus of the SEM analysis undertaken in Study Three, preliminary regression analyses indicated that facets of imagery operated differently pre and post-treatment of PTSD and in the change in scores for imagery, affects and PTSD consequent to treatment.

Considerable research has established the general association of anxiety and imagery, and imagery has been implicated as an important aetiological and maintaining factor in anxiety disorders (Cuthbert et al., 2003). In contrast, research investigating the connection of imagery to anger has been lacking. This thesis qualifies and extends the limited research available. It confirms that imagery is important in anger per se and that imagery with angry content is specifically
associated with anger in non-clinical populations. More specifically, it confirms the research showing that imagery control (as measured by imagery frequency, absorption and frequency) is associated with anger in PTSD (Laor, Wolmer, Wiener, Sharon et al., 1999; Laor, Wolmer, Wiener, Weizman et al., 1999) and is a particular feature of PTSD affected populations.

The finding that the contribution anger makes to imagery in PTSD and to PTSD directly differs by population type is novel and important in understanding anger in PTSD. The explanation may, by inference, be related to the moral wound that can be associated with events encountered in combat and combat-related PTSD. Risk of moral wounding is unlikely to be present to the same extent in policing, where anger as a feature of PTSD appears to be less prominent. In that population, anger may more commonly be related to cumulative stress, criminal malfeasance, violence, danger, the vicissitudes of the judicial process and the common perception among police that the organisation fails to care for them (Mayhew, 2001).

Across both populations, dysregulated anger has the potential to cause disrupted and biased information processing. Key to this dysfunction, but hitherto overlooked, the capacity to visually image mental phenomena arguably contributes significantly to anger in PTSD. It is critical to re-emphasise that perception can be neutral, but mental imagery is not and the formation and retrieval of memory is a constructive process influenced by cognitive bias (Kosslyn, 2005a). Dysregulated intrusive memories (which are overwhelmingly reported by sufferers as being visual in nature) are a critical mediating influence on information processing. In their fragmentary and meaning-implicit guise they profoundly impact on affect expression and the ability to control it.

Here the views of Ahsen and Lang on imagery are highly relevant. To recall from Chapter Six, both emphasise the role of meaning in information processing by propositional networks based on imagery, albeit in slightly different ways. Lang in his Bioinformational Theory of Emotional Imagery (Lang, 1979; Lang et al., 1999) proposes imagery acts as part of a cognitive propositional network that intimately links physiological and behavioural patterns. The image acts as a template for overt responding. Modifying it or overt behaviour will result in a change in the other. Interestingly, Lang observed that even single words describing emotion-related events, via through their connection to imagery, prompt the same bodily responses as those cued by the events themselves.
Ahsen in his Triple Code Model of imagery (Ahsen, 1982; 1984) asserts an image has three fundamental elements. The first is that it must be centrally arousing and cause the individual to interact with it as if it were representative of the real world. Second, it must include a somatic response element. Third, it must contain a specific meaning, the content of which will vary individual-by-individual. His argument, unlike Lang’s idea that physiological responses emerge from thought based-propositional networks initiated by imagery, instead, proposes that an image can directly cause physiological responses.

Psychologically imagery has recently been recognised as having a greater impact than words on emotion (Arntz et al., 2005; Holmes & Mathews, 2010; Mathews, Ridgeway & Holmes, 2013). This is underpinned by emerging fMRI-related evidence of specialised and yet overlapping core networks of brain regions recruited during imagery and the processing of negative affect, anger included (Blair, 2102; Denson et al., 2009; McNorgan, 2012). It is also understood that imagery plays a critical important role in the regulation of emotional processes, and differences among individuals in their imagery ability have been hypothesised to relate to differences in ability to experience emotional ideation (Suler, 1985). Perceptual imagery by its nature is short-lived. Similarly, in the presence of low-to-moderate stressors, anger is a temporary affective state and Novaco (1993) has stressed the importance of understanding anger in its context. Under situations of abnormal stress, traumatic or otherwise, emotion and mental imagery tend to persist (see Fernandez, 2008 for a discussion of “tonic” anger).

As might be reasonably expected, as either the frequency or magnitude of imagery increases it negatively impacts upon thought. This is consistent with the observation that high prevalence/intensity visual information processing interferes with the capacity to reason (see Knauff & Johnson-Laird, 2002). This study implies, however, that, rather than competing for cognitive space and negatively impacting on the incidence and expression of the other, imagery and thought may be interactive, such that imagery partially determines thought in a mediated relationship in which thought ultimately predominates. This is entirely consistent with Kosslyn’s notion of a visual imagery buffer (see Kosslyn, 2005a) that allocates space to and regulates information processing speed. The precise nature of this relationship and whether there are thresholds at which this cognitive complementarity breaks down, remain
unanswered and were not intended to be resolved by this research. However, these are intriguing questions for future research.

Notwithstanding the preliminary status of this research’s findings around the role of imagery, thought, and their interplay in anger in PTSD, they have significant explanatory potential. Although not explicitly tested in this research, the relevance of imagery to rumination - a phenomenon long known to be a critical feature of treatment resistant PTSD - is a telling example.

Anger has latterly been identified as strongly associated with rumination (Potegal, 2010). Sukhodolsky and associates (2001) explain angry rumination by reference to three processes. In the first two, memories of past experiences lead to new anger episodes and the focusing of attention on anger-related experiences, thereby leading to increased intensity and duration of anger. In the third process, counterfactual statements (e.g., “I should done”) may relate to action tendencies toward regret, resolution and retaliation. Angry rumination of this kind can last for days, weeks and even months (Potegal, 2010).

Cognition as thought has long been considered an essential ingredient of rumination. On the basis of this research, it is arguable that conceptualisations of angry PTSD-related rumination would benefit from the incorporation of the role of voluntarily manifested and intrusive, involuntary imagery given that imagery is described as “realer” than words (Arntz et al., 2005; Holmes & Mathews, 2010; Mathews, Ridgeway & Holmes, 2013).

Rumination is known to be increased where there is autobiographical memory, especially involving others, and morally challenging issues. Imagery has a fundamental role in autobiographical memory (Greenberg & Knowlton, 2014; Schonfeld & Ehlers, 2006). In the presence of such content, memories are likely to become entrenched, revolving they do around issues of responsibility, “unfinished business” and regrettable action(s) which might have been avoided. Denson and associates (Denson, 2012; Siedlecka, Capper & Denson, 2015) emphasise the impact of the clarity of such rumination, noting that it feels close in time, and invokes neurobiological, affective, executive control and behavioural processes in its operation. Under such circumstance it becomes a dysfunctional mechanism for regulating emotion in PTSD (Ehring & Ehlers, 2014).
Drawing together the outcomes of this research, it is possible to generate a speculative understanding of how dysregulated anger can develop and the role of imagery in it. Such an understanding is schematically represented in Figure 11.1.

Placing this model in the context of theoretical explanations for the development of dysfunctional anger reviewed in Chapters Two, Three and Four, it is possible to articulate an imagery-informed existential model of dysfunctional anger. Initially, conditioning in early age or in prolonged and significant adulthood experiences, will increase the likelihood of the manifestation of negative affect. This is predicted by Bandura’s (1973) learning theory and in the military arena more specifically, Keane and colleagues’ (1985) conditioning theory description of the potential impact of military training. This negative affect is expressed physiologically and psychologically, with each amplifying the other. Such psychobiological phenomena become problematic when traumatic stressors and diatheses intersect (as first noted by Meehl, 1962). Under such circumstances, psychological and physiological dysregulation, as represented in the survivor mode theory proposed to account for anger in PTSD by Chemtob and others (e.g. Novaco & Chemtob, 2002) can occur. Negative emotions can also then become bonded to each other, as provided for by the primary emotion avoidance model described by Greenberg and Paivio (1997), and its application to anger in PTSD by Feeny and associates (2000). These emotional dysregulation and avoidance processes can become powerfully associated with physiological processes as provided for in Berkowitz’s (1990) neo-associationist model of anger as seen in its application to anger in PTSD by Taft, Vogt, Marshall, Panuzio and Niles (2007).
At each point in this sequence, imagery can play a potentially important role. To reiterate the assertions made in Chapter Four, this is because there will be an increased chance of affect dysregulation and avoidance and activation of an associationist network that emphasises the contribution of cognitive, affective and physiological factors where imagery is significant and dysregulated. Figure 11.1 is an
embellishment of the model documented in Chapter Seven and the SEM analysis diagrams of Chapter 10 and demonstrates this.

Despite the emphasis given to the need for nuanced research on anger in PTSD and its underlying mechanisms, theoretical models as observed in Chapter Four are scant, narrow in scope and inadequately researched. The overwhelming implication of this research is that a classification of anger in PTSD which places emphasis on imagery is capable of extending the reach of such models. The inclusion of imagery in explanatory models of anger in PTSD would also be consistent with the deeper-level, detailed, localised and practical theorising Dalgleish (2004) advocated in his comprehensive analysis of the requirements of PTSD research. It would simultaneously avoid the narrowness-of-focus and alienating complexity pitfalls he warns against.

11.2.2 Treatment Implications. Many individuals exposed to potentially traumatic events (PTEs) experience some posttraumatic symptomatology. For most, such symptoms are short-lived and sub-clinical in severity. Nevertheless, for a significant minority, they represent a persistent syndrome associated with significant distress, treatment resistance and substantial ongoing functional impairment. There is accumulating evidence that dysfunctional anger is often central to PTSD. The current research adds to that evidence.

Several of its outcomes have implications for improving the assessment and treatment of this chronic, angry form of PTSD. Its initial finding that presenting anger levels are a predictor of PTSD improvement emphasises the need for careful assessment of anger and a specific focus on it in PTSD treatment. Additional treatment for both anger and PTSD has been a repeated recommendation in recent clinical research (e.g., Elbogen et al., 2010; 2011; Worthen et al., 2014). This research supports those views.

The research’s second major finding, that anger in PTSD at least in part reflects the type of PTE experienced, demonstrates the need to recognise and understand the meaning of the event in the provision of treatment to the angry PTSD sufferer. That different event types lead to different outcomes in PTSD is well described in event typologies (e.g., Terr, 1991) and the literature recognising the differential impact of human involvement in event causation in PTSD. This research illustrated that this may also apply to anger in PTSD (as identified in the schematic representation in Table 4.3 of Chapter Four).
The third major finding of this research, that anger predicts PTSD through imagery and thought, adds to and yet qualifies existing research. That research indicates high arousal and visual intrusions in combination have a greater effect on PTSD than either alone (Schell et al., 2004; Shea et al., 2013). This research suggests that the inability to control imagery in the context of anger in PTSD is not only cardinal to the PTSD, but may be equally so to anger in PTSD. It lends support to calls for increased anger work in the presence of high symptom, enduring PTSD, but emphasises that work would benefit from being imagery-informed.

Dysfunctional anger, often to the relief of sufferers, can be successfully treated. This has been amply identified in meta-analyses showing successful treatments exist, improvement is real and lasts (see Digiuseppe & TafRATE, 2001). The efficacy of anger treatment has also been shown in populations with PTSD (see Chemtob et al., 1994; Novaco & Chemtob, 2002; Gonzalez, Novaco, Reger & Gahm, 2015) such that decreases in anger lead to decreases in PTSD (Novaco & Chemtob, 1998).

Effective treatment of dysfunctional anger in PTSD, like any other psychopathological condition, is predicated upon understanding its precipitating and maintaining factors and effective case formulation (Gonzalez et al., 2015). Across Chapters Two, Three and Four, this research identified high order risk factors for PTSD, anger and anger in PTSD, primary among them abnormal early age anger, exposure to dysfunctional family-of-origin anger, traumatic event type, emotion regulation capacity, cognitive content justifying anger, ongoing physical injury syndromes, especially pain and traumatic brain injury. A starting functional analysis and formulation of anger in PTSD would seek to document such factors. Beyond them, the outcomes of this research suggest the specific characteristics of imagery associated with the development and maintenance of negative and angry affect and the cognitive style of the individual need to be included in assessment and screening.

Consequently, this research suggests treatment of anger in PTSD will be improved by a focus on imagery. How this might proceed is a moot point, however, for there is a persisting lack of clarity around whether dysfunctional anger can be treated imaginally, or is best addressed via the adoption of a management approach that is imagery-informed. To illustrate, a repeated finding of best practice evidence reviews suggests that trauma focussed psychological intervention(s) via re-experiencing of memories associated with the trauma and associated feelings is the
first line treatment for PTSD (see ACPMH, 2013). Exposure based treatment has been one of the most important developments in the treatment of anxiety disorders (McNally, 2007), but requires the ability to regulate intense negative emotion. On this basis, Foa and colleagues (1995) have expressed caution about the (in)applicability of exposure to dysfunctional anger in PTSD on the basis that (a) fear imagery and thoughts that are effectively targeted by in vivo and imaginal exposure and (b) anger is unlikely to be extinguished in this manner and may even result in unintended deleterious outcomes.

There have been several attempts at using prolonged exposure in the treatment of problematic anger. These studies, using past memory activation, hierarchies of future problem scenarios and the application of barbs, have met with mixed results and have been unable to identifying the underlying psychological mechanisms at play. Consequently, alternative approaches have advocated the use of anger management, rather than anger engagement via self instruction training, reciprocal inhibition via relaxation training skills and cognitive restructuring (see (see DiGiuseppe, Cannella & Kelter, 2007)).

Rather than it being an issue of the suitability of imaginal exposure to the treatment of dysfunctional anger in PTSD per se, it is possible the answer to improvements in the treatment of dysfunctional anger in PTSD may lay in the subtlety of use and features of the imaginal approach employed. For example, it may be that anger work in PTSD is best applied as a necessary precursor to trauma-focussed exposure work (see Forbes et al., 2008). It could also be the case that titrating exposure work - for example, by reference to the pace and depth of processing or through a combination of anger focussed imaginal work, cognitive restructuring (Mueser et al., 2009) and anger-management skills based work, such as Self Instruction Training (Novaco, 1977) or the application of self soothing (Morland et al., 2012) - will increase capacity to engage with and tolerate imagery-focussed anger treatment. Alternately, it may be other models of imagery might be better suited when working with dysfunctional anger in PTSD. To illustrate, various researcher-practitioners (e.g., Arntz et al., 2007; Smucker & Moos, 2005) have endorsed the use of an imagery-based alternative to prolonged exposure Imagery Reprocessing and Rescripting Therapy (Smucker, 2012) - which involves imaginal reliving, the development of mastery imagery and the application of self-calming and nurturing imagery - as being efficacious in the treatment of difficult-to-shift anger in PTSD.
Regardless of the approach applied, as discussed in Chapter Six, it will be crucial that the individual’s cognitive style and imagery-thought balance is properly accounted for in any intervention applied. Gradually, safely and sensitively exploring the role of imagery in anger in PTSD via such means appears to have great promise in dealing with the noted barriers to treating anger and more particularly anger in PTSD. These include fear of anger expression (Forbes, et al., 2008), failure to effectively engage with the traumatic memory (Foa et al., 1995) and failure to remain engaged in treatment (Stephenson & Chemtob, 2000). They also include the use of non evidence-based and ineffectual treatment methods such as catharsis (Bushman, 2002; Potegal, 2010), which has little chance of success given its inability to either access the image in any depth or manage angry affect.

The fourth major finding of this research, that the causal contribution of anger to PTSD through imagery is mediated by thought, cautions against the adoption of a view of cognition emphasising the role of imagery alone. Clearly thought plays an important role in anger in PTSD. This research raises the possibility that, where symptoms persist in chronic angry PTSD, imagery and thought are likely to be inextricably linked, with the former driving the latter. As noted in Chapters Two, Three and Four, the involvement of guilt, shame, disgust, horror and repulsion is provided for in descriptions of dysfunctional anger and PTSD. Angry contemplation of moral dilemmas or social norm violations that occurred at the behest of national and community interest is likely to be strongly involved. Again, thought driven by imagery is unlikely to be far removed from this. Field leaders in research of perception have focussed on imagery to improve understanding of the neuroanatomy of imagery and much knowledge has been developed over the last 60 years via that focus. Better understanding the functional role of imagery in anger and anger in PTSD and applying this knowledge to downstream treatment practices, however, requires both dimensions of cognition to be considered.

11.2.3 Research Implications. This research produced promising outcomes in testing the key elements of an explanatory model of anger in PTSD that emphasises the contribution imagery makes to their relationship. It represents an initial examination of a prototypical model, however, and it is best understood as early work subject to limitations. Such findings, nevertheless, provide sufficient grounds for further researching the model. Continued research from this perspective will shed
light on anger in PTSD, and may also illuminate the role of dysfunctional anger in other trauma-related mental health conditions and dysfunctional anger per se.

Future research aimed at validating the role of imagery in anger in PTSD will require a strategic approach incorporating several stages of enquiry. First, the contribution of key imagery dimensions to anger in PTSD needs to be mapped. As observed by Forbes, McHugh and Chemtoc (2013), the phenomenology of anger in PTSD is poorly documented and a critical step will be to qualitatively establish the nature of anger in PTSD across trauma-types and PTSD populations and the role imagery plays in such anger. By doing so, it may become possible to identify anger in PTSD prototypes based around imagery. There is precedent for such the exploration of typologies in dysfunctional anger generally [e.g., Armed, Kingston & DiGuisepppe (2012) and DiGuisepppe & Tafrate (2006)] and dysfunctional anger in the presence of PTSD (see Chemtob et al., 1997; Finley, Baher, Pugh & Petersen, 2010). It is important to note that such typologies have been described as prototypes in need of further research in PTSD (Morland et al., 2012).

Following on from this, there is a need to subsequently undertake an imagery-informed quantitative mapping the experience of imagery in anger generally and in the context of PTSD. Consistent with the recommendations of reviews of imagery research and research innovations in the study of anger in PTSD (see Hackmann et al., 2004; Holmes and Mathews, 2005; 2010; Laor, Wolmer, Wiener, Sharon et al., 1999; Laor, Wolmer, Wiener, Weizman et al., 1999; Pearson et al., 2013; Singer & Antrobus, 1972), this thesis chose to focus on the role of the imagery process qualities of control, absorption, vividness and nowness. It showed that like previous research (e.g., Cahill et al., 2003; Foa & Rauch, 2004), such imagery dimensions are highly relevant to the impact of imagery in anger in PTSD. This research also examined the significance of imagery content in the form of angry imagery and its association with anxiety, depression and anger. While a measure for simultaneously testing all affective-imagery contents is in practice impossible to apply and very few measures which deliberately tap affective content exist, the challenge in future research will be targeting and contrasting different affective-imagery contents. To recall the observation made in Chapters Five, angry PTSD sufferers report being troubled by both the repetitiveness and content of out-of-control imaging. Anger, anxiety and depression as affects that figure prominently in PTSD are an important starting point. Given the emphasis placed on dysregulation across various theoretical
orientations, it will be important to measure affect and imagery by reference to thresholds of dysfunctionality.

It will also be important that research incorporates validated approaches suggested by emerging frameworks for imagery research. One such framework is that advocated by Pearson and colleagues (2013). To reiterate (see Chapter Eight methods section), they emphasise exploring the role of facets of imagery, such as the cognitive (e.g., imagery generation and manipulation), subjective (e.g., individual imagery experience) and clinical (e.g., imagery intrusions and bias).

This quantitative approach will expand its reach by examining other constructs identified in the present research as implicated in or illustrative of the role of imagery in anger in PTSD. At a minimum, it will be important that the role imagery may play in well-validated anger phenomena, such as anger expression, control suppression and perceived provocation, is examined. Thus, it will be possible to assess the extent to which anger influenced by imagery can be assessed via proven measures like the Novaco Anger Scale (Novaco, 1994) or STAXI-2 (Spielberger, 1999) or if it requires new or augmented metrics.

It is important that the measurement of PTSD in future research takes account of the frequency, magnitude and type of the traumatic experiences of research participants. As argued across this thesis, and as further detailed in subsection 11.3, the traumatic event remains a critical element of the PTSD nomenclature and trauma dose and type remain important predictors of response. Additionally, given the case made in this research for the role of imagery in angry rumination in PTSD, it is important that a specific measure of autobiographical memory be included in future research.

To build a more coherent picture of anger in PTSD and the operation of imagery within it, it is also important to demonstrate how imagery interacts with other cognitive processes involved in anger in PTSD, such as the anxiety sensitivity, negative attributional and looming cognitive styles identified by Elwood and colleagues (2009), and the assumptions about the world, others and self of Janoff-Bulman’s (1992) social cognition theory. As predicted and demonstrated by this research, imagery alone is unlikely to account for all presentations of anger in PTSD. At times, it can have little effect on anger in PTSD. Alternately it can have a large, singular effect on anger or, it can interact with thought to produce its effects. Consistent with the view that rumination can be voluntary or intrusive (Rubin &
Bernstan, 2009), it is important that any measure of imagery be capable of measuring both dimensions of memory.

While an imagery measure for simultaneously testing all affective-imagery contents is in practice impossible to apply, the challenge in future research will be targeting and contrasting different affective-imagery contents. It will also be important that research incorporates validated approaches suggested by established frameworks for imagery research; for example, the approach of Pearson, Deeprose, Wallace-Hadrill, Heyes & Holmes (2013), which emphasises exploring the role of facets of imagery, such as the cognitive (e.g., imagery generation and manipulation), subjective (e.g., individual imagery experience) and clinical (e.g., imagery intrusions and bias).

By these means, it may be possible to examine thresholds of imagery and thought associated with (dys)functionality. The importance of doing so is underscored by the well-established deleterious effect of ruminative thought in depressive disorder and also the observation that high prevalence/intensity visual information processing interferes with the capacity to reason (see Knauff & Johnson-Laird, 2002).

Finally, it is important that future investigation occurs on a longitudinal basis across different PTSD populations that vary demographically so that further data may be brought to bear on issues of gender, ethnicity, age and time from trauma. Each of these factors has been shown to affect the intensity and expression of anger and intrusive phenomena in PTSD (see Giambra, 1999; Orth & Wieland, 2006) and the generalisability of outcomes may be enhanced. Given there is evidence personality and dispositional factors are implicated in the manifestation of anger (Miller, 2003) and imagery (Singer & Antrobus, 1972), it is important investigation is also extended to these enduring characteristics.

11.3 Methodological Strengths and Weaknesses of this Research

This research has several methodological strengths. The use of a variety of data analytic strategies in a multi-study design permitted the comparison of anger and imagery phenomena across different populations, including clinical and non-clinical presentations. Via this staged approach to enquiry, it implemented several important design innovations. Initially, it cross-sectionally and longitudinally compared anger’s impact on PTSD to that of other negative affects. This comparison of anger, anxiety and depression across general and PTSD populations was novel and produced
important findings. Its inclusion of measures of imagery process (frequency, vividness and absorption) and content (anger) anticipated review recommendations arguing that researchers are best to measure beyond single dimensions when investigating imagery (see Pearson et al., 2013). It also provided a broader examination of the phenomena related to anger in PTSD - that is imagery, thought, negative affect, anger and PTSD - and thereby avoided the traditional tendency of cognition researchers to narrowly research target phenomena by reference to imagery or thought alone. As far as can be ascertained, the simultaneous inclusion of measures of negative thought, a range of affects and clinical dysfunction (alcohol misuse and PTSD) populations was unique and took recommendations about breadth of focus beyond such review recommendations. It also appeared to be the first research to simultaneously investigate such phenomena and their relationships in police and military-affiliated populations affected by PTSD. Research on the mental health in police is rare and this research’s comparison of police with PTSD and, the comparatively well-researched population of military personnel with PTSD enabled important comparisons to be made. Altogether, the innovations introduced by it provided a sound basis for further exploration of PTSD by reference to the variables included in this research.

This research inevitably also has methodological limitations. These in part derive from the inescapable restrictions of psychological research requiring the selection of finite populations of participants. In this research, university students and treatment-seeking PTSD sufferers who had completed a CBT-based group treatment program for PTSD were the chosen participant populations. Although the use of multiple groups in a variety of study designs was likely to reduce their population non-representativeness, the problem is hard to avoid in phenomenological and empirical research and in the interpretation of the consequent outcomes. Thus, the predominantly female undergraduate population sample of study two and the occupationally traumatised populations of studies one (Vietnam veteran) and study three (Vietnam veterans, contemporary veterans and police) are not easily generalised to other community populations exposed to trauma (e.g., mental health patients and disasters survivors).

There were also several data-collection related limitations. Because none of this research’s constituent studies were prospective in nature, it was not possible to establish either pre-measurement/treatment cognition (imagery and thought) and symptoms levels and thereby assess pre to post treatment change via a more
sophisticated multi-timepoint involving predictor and dependent measures. Additionally, while the sample size for the first study was large (with n of 353 and 256 at times one and two respectively), the large number of dropouts was undesirable desirable. While reiterating the difficulty of conducting prospective PTSD research, being able to do so in future research would be invaluable in further clarifying the causality of imagery to anger in PTSD.

There was also the inevitable limit due to the absence of any information collection regarding extraneous-to-study stress and treatment-related factors likely to have existed for some of the almost 800 participants during their involvement in the research. It is improbable some individuals did not experience stressors likely to impact on their well-being during the time of their involvement and this may have to some extent had a confounding effect on the results. Similarly, it was highly unlikely that many of the study’s participants did not receive additional treatment in the period between the first and second measurement points. There was also the non-randomised nature of this research, the cross-sectional nature of its second study and the absence of comparison groups in studies one and two. Finally, there was the use in studies one and two of this research on change data generated as a function of multi-faceted treatment program outcomes. Such limitations were unavoidable, inevitably create difficulty determining change causality and would be best addressed in future research.

There were further limitations relating to the measures utilised in this research. In particular, all measures were self-report in nature. While the utility of this form of enquiry in assessing visual imagery is well noted, more objective tests would have added to the credibility of the experimental procedure. In addition, imagery was assessed by reference to several process variables (frequency, absorption, imagery presence and vividness), but only one content variable (angry-imagery). In addition that measure used the term daydreaming as a proxy for imagery. This had the potential to confuse participants and future iterations of its use would do well to alter the wording of the measure to more directly refer to imagery processes. The use of the negative thought subscale of the Automatic Thoughts Questionnaire was also potentially problematic in that, while it was included as a measure of thought based cognition, preliminary analyses of Study Three data suggests it may have operated as a proxy measure for depression in the context of PTSD. Notwithstanding the aim of preventing the added participant burden due to the use of the entire measure, full scale
use or the use of a measure of cognitive style may have resulted in an improved determination of the impact of thought. Similarly, limits also derive from the use of the seven item DAR scale. Although it and its successor the DAR-5 have been shown to be robust screening tools for problematic anger (Forbes et al., 2004; 2014), they offer no information about important anger-related phenomena, such as the anger control, expression and suppression characteristics identified by Spielberger (1999). Consideration was given in the design of this research to the inclusion of a more comprehensive anger measure, such as the second edition of State-trait Anger Scale (STAXI-2; Spielberger, 1999) or the Novaco Anger Scale (NAS; Novaco, 1994). This was decided against on the twin grounds of the need to restrict the participant burden that would have accrued from the inclusion of such long measures and the judgement that a screening measure of anger, with the well-validated status of the DAR scale, was sufficient in this research for drawing conclusions regarding the relationship between anger and PTSD. Another complicating factor was the use of different measures of anxiety and depression in Study Two compared to studies one and three. This was to some extent addressed by percentile equilibration of sample performance on the measures, but ideally it would have been best to avoid. Finally, this research was limited by the absence of a measure capable of comparing the traumatic experiences of the different populations involved. The Combat Exposure Scale (CES; Keane et al., 1989) was used in relation to the ADF-affiliated participants of Study One and Study Three, but not the current serving military personnel of Study Three. In addition, it was not possible to apply either specific or comparative population measures of trauma exposure in the military and police populations, as the CES is clearly inappropriate for application to police. Currently there is no way of quantitatively or qualitatively comparing the nature and impact of traumatisation in these groups: a combat exposure scale exists to test this in the military, but no equivalent measure appears to exist for police. While a broad trauma experience measure does exist in Kubany’s (2000) *Traumatic Life Events Questionnaire* it was not perceived to have sufficient depth to effectively tap the importance of the specific trauma types experienced by the populations in this study.

11.4 Conclusion

This research employed an integrated, sequential series of studies to test a model of anger in PTSD predicated upon the role of imagery. It showed anger is a
crucial predictor of PTSD that has a strong association with imagery. Anger, through independent imagery and thought pathways and a mediated pathway via negative thought, was shown to be strongly related to PTSD. Although this applied to different populations, important between-population differences in both the intensity of imagery, thought, anger and PTSD and the predictive relationships among these phenomena were observed. Anger typically predicted imagery more than thought. That relationship, however, differed according to occupational grouping and the nature of traumatic experience involved. Those with PTSD of a military origin were more likely to experience anger associated with imagery, compared to those with PTSD of a policing origin. As imagery, thought, anger and PTSD scores decreased as a consequence of treatment, ADF-affiliated participants were equally likely to experience anger related to thought. Interestingly, the reverse applied to police participants and they were increasingly likely to experience increased imagery and reduced thought that regressed upon anger.

This research was rooted in two decades of clinical observation of anger in the PTSD populations involved in this research. Recognising the significance of its impact on their lives and those of their dependents, it endorsed the observations that anger in PTSD can only be understood by appreciating the psychological mechanisms which may underlie it (see Hellmuth et al., 2013; Orth & Weiland, 2006). It suggests that a model of anger in PTSD which affords imagery an appropriate place as one such mechanism will add to existing theory. Incorporation of the role of imagery along with the role of thought into theories of anger in PTSD would be entirely consistent with the multi-representational theorising advocated by Dalgleish (2004) and redolent of the recognition of Beck and Emery thirty years ago that “Undesirable visual images often stimulate verbal cognitions” (1985: page 222). This imagery-broadened view of the role of cognition in anger has the potential to offer new understandings of anger in PTSD and its treatment. Taking up A.T. Beck’s observation that effective treatment “depends greatly on moving beyond purely verbal exchanges to encouraging patients and therapist to resort to their auditory or visual imagery capacities” (Singer, 2006: page 107), recognising and addressing the role of imagery on anger in PTSD has the potential to assist sufferers unlearn unwanted posttraumatic reactions and develop new ways of responding to traumatisation.
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References


Appendix A:

*Formal DSM 5 diagnostic criteria for PTSD*

The person was exposed to the following event(s): death or threatened death, actual or threatened serious injury, or actual or threatened sexual violation, in one or more of the following ways:**

1. Experiencing the event(s) him/herself
2. Witnessing the event(s) as they occurred to others
3. Learning that the event(s) occurred to a close relative or close friend
4. Experiencing repeated or extreme exposure to aversive details of the event(s) (e.g., first responders collecting body parts; police officers repeatedly exposed to details of child abuse)

NOTE: *Witnessing or exposure to aversive details* does not include events that are witnessed only in electronic media, television, movies or pictures, unless this is part of a person’s vocational role. Exposure to aversive details of death applies only to unnatural death.

A. Intrusion symptoms that are associated with the traumatic event(s) (that began after the traumatic event(s)), as evidenced by 1 or more of the following:

1. Spontaneous or cued recurrent, involuntary and intrusive distressing memories of the traumatic event(s). Note: In children, repetitive play may occur in which themes or aspects of the traumatic event(s) are expressed.
2. Recurrent distressing dreams in which the content and/or affect of the dream is related to the event(s). Note: In children, there may be frightening dreams without recognisable content. ***
3. Dissociative reactions (e.g., flashbacks) in which the individual feels or acts as if the traumatic event(s) were recurring (Such reactions may occur on a continuum, with the most extreme expression being a complete loss of awareness of present surroundings.) Note: In children, trauma-specific re-enactment may occur in play.
4. Intense or prolonged psychological distress at exposure to internal or external cues that symbolise or resemble an aspect of the traumatic event(s)
5. Marked physiological reactions to reminders of the traumatic event(s)

B. Persistent avoidance of stimuli associated with the traumatic event(s) (that began after the traumatic event(s)), as evidenced by efforts to avoid 1 or more of the following:

1. Thoughts, feelings, or physical sensations that arouse recollections of the traumatic event(s)
2. Activities, places, physical reminders, or times (e.g., anniversary reactions) that arouse recollections of the traumatic event(s)
3. People, conversations, or interpersonal situations that arouse recollections of the traumatic event(s)

C. Negative alterations in cognitions and mood that are associated with the traumatic event(s) (that began or worsened after the traumatic event(s)), as evidenced by 3 or more of the following: Note: In children, as evidenced by 2 or more of the following:****
Anger-in-PTSD: The Role of Visual Imagery

(1) Inability to remember an important aspect of the traumatic event(s) (typically dissociative amnesia; not due to head injury, alcohol, or drugs).

(2) Persistent and exaggerated negative expectations about one’s self, others, or the world (e.g., “I am bad,” “no one can be trusted,” “I’ve lost my soul forever,” “my whole nervous system is permanently ruined,” "the world is completely dangerous").

(3) Persistent distorted blame of self or others about the cause or consequences of the traumatic event(s)

(4) Pervasive negative emotional state - for example: fear, horror, anger, guilt, or shame

(5) Markedly diminished interest or participation in significant activities.

(6) Feeling of detachment or estrangement from others.

(7) Persistent inability to experience positive emotions (e.g., unable to have loving feelings, psychic numbing)

D. Alterations in arousal and reactivity that are associated with the traumatic event(s) (that began or worsened after the traumatic event(s)), as evidenced by 3 or more of the following: Note: In children, as evidenced by 2 or more of the following:****

(1) Irritable, angry, or aggressive behaviour

(2) Reckless or self-destructive behaviour

(3) Hypervigilance

(4) Exaggerated startle response

(5) Problems with concentration

(6) Sleep disturbance - for example, difficulty falling or staying asleep, or restless sleep.

(APA, 2013)
Appendix B:

A sample of the Positive and Negative Qualities of Anger

<table>
<thead>
<tr>
<th>Positive Functions</th>
<th>Negative Functions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anger is an energiser. It provides vigour, mobilises the body’s resources for self-defence and provides stamina when a task becomes difficult. It enables individuals to deal with conflict by supplying the fuel for the fight.</td>
<td>Anger can be a barrier to clear thought and considered action. When angry, it is harder to think clearly and evaluate options. Individuals act on impulse without considering the consequences of their behaviour.</td>
</tr>
<tr>
<td>Anger can be a helpful way to express tension and communicate negative feelings to others. Occasionally things stay bottled up until people get angry. The constructive expression of anger is an important way to resolve conflict.</td>
<td>Anger sometimes allows individuals to become defensive unnecessarily. When an individuals feels hurt or embarrassed they may anger as a way to protect their pride. It is easier to be angry than to be anxious. Individuals sometimes become angry to prevent themselves from being hurt.</td>
</tr>
<tr>
<td>Anger, like other feelings, signals information about people and situations. It serves as a cue to notify people that there is something unjust, frustrating, threatening or annoying occurring. This signifies that it is time to begin coping with stress.</td>
<td>There is a connection between anger and aggression. Anger can instigate or lead to aggression. When individuals become emotionally upset, they sometimes try to discharge or release their feelings through their behaviour - they get angry and then try to take it out on something or someone.</td>
</tr>
<tr>
<td>The arousal of anger creates a sense of being in charge of a situation. It stimulates a feeling of control. When a situation escalates, anger enables people to take charge and assert their will or interest.</td>
<td>Being angry conveys an impression to others. At times individuals show their anger because they want others to see them in a certain way. Demonstrating anger becomes a way to build a particular kind of reputation.</td>
</tr>
<tr>
<td>Anger can be a response to wrongdoing or injustice. On the basis of a sense of rightness, morals or ethics, anger can motivate individuals and communities others into action.</td>
<td></td>
</tr>
</tbody>
</table>

Appendices Page 291
Appendix C:
Cardinal Texts and Commentaries about Anger’s Impacts

Anybody can become angry - that is easy, but to be angry with the right person and to the right degree and at the right time and for the right purpose, and in the right way - that is not within everybody's power and is not easy.

Aristotle

If a man meets with injustice, it is not required that he shall not be roused to meet it; but if he is angry after he has had time to think upon it, that is sinful. The flame is not wrong, but the coals are.

Beecher

Holding on to anger is like grasping a hot coal with the intent of throwing it at someone else; you are the one who gets burned.

Buddha

Anger is the most impotent passion that accompanies the mind of man; it effects nothing it goes about; and hurts the man who is possessed by it more than any other against whom it is directed.

Clarendon

The intoxication of anger, like that of the grape, shows us to others, but hides us from ourselves, and we injure our own cause, in the opinion of the world, when we too passionately and eagerly defend it.

Colton

Do not be quickly provoked in your spirit, for anger resides in the lap of fools.

Ecclesiastes 7:9

In your anger do not sin: Do not let the sun go down while you are still angry

Ephesians 4: 25-32

When you are offended at any man’s fault, turn to yourself and study your own failings. Then you will forget your anger.

Epictetus
Anger is never without a reason, but seldom with a good one.

Benjamin Franklin

People won't have time for you if you are always angry or complaining.

Stephen Hawking

A fit of anger is as fatal to dignity as a dose of arsenic is to life.

J. G. Holland

Anger is a short madness.

Horace

Anger blows out the lamp of the mind. In the examination of a great and important question, everyone should be serene, slow-pulsed, and calm.

R. G. Ingersoll

Everyone should be quick to listen, slow to speak and slow to become angry, for man’s anger does not bring about the righteous life that God desires.

James 1:19-20

But I tell you that anyone who is angry with his brother will be subject to judgment.

Matthew 5:22

There are two things a person should never be angry at, what they can help, and what they cannot.

Plato

A gentle answer turns away wrath, but a harsh word stirs up anger.

Proverbs 15:1

A hot-tempered man stirs up dissension, but a patient man calms a quarrel.

Proverbs 15:18
A fool gives full vent to his anger, but a wise man keeps himself under control.

Proverbs 29:11

Refrain from anger and turn from wrath; do not fret - it leads only to evil.

Psalms 37:8-9

He that will be angry for anything will be angry for nothing.

Sallust

Anger is like a ruin, which, in falling upon its victim, breaks itself to pieces.

Lucius Annaeus Seneca

Anger: an acid that can do more harm to the vessel in which it is stored than to anything on which it is poured.

Lucius Annaeus Seneca

Anger, if not restrained, is frequently more hurtful to us than the injury that provokes it.

Lucius Annaeus Seneca

If anger is not restrained, it is frequently more hurtful to us, than the injury that provokes it.

Lucius Annaeus Seneca

Men in rage strike those that wish them best.

Shakespeare

An angry man is again angry with himself when he returns to reason.

Publius Syrus
Part 1 What does my participation involve?

1.1 Introduction
You are invited to take part in this research project, which is called Improving Anger Treatment Outcomes for Traumatised Combat Veterans: A Trial of a Stand-Alone Psychological Group Treatment. You have been invited because based on conversations held with clinical staff where you expressed interest in participation or you responded to the advertisement placed within the Psychological Trauma Recovery Service.

This Participant Information Sheet/Consent Form tells you about the research project. It explains the processes involved with taking part. Knowing what is involved will help you decide if you want to take part in the research.

Please read this information carefully. Ask questions about anything that you don’t understand or want to know more about. Before deciding whether or not to take part, you might want to talk about it with a relative, friend or local health worker.

Participation in this research is voluntary. If you don’t wish to take part, you don’t have to.
If you decide you want to take part in the research project, you will be asked to sign the consent section. By signing it you are telling us that you:

- Understand what you have read
- Consent to take part in the research project
- Consent to be involved in the research described
- Consent to the use of your personal and health information as described.

You will be given a copy of this Participant Information and Consent Form to keep.

1.2 What is the purpose of this research?
This study has been designed to assess the effect of brief group treatment for problematic anger in Australian combat veterans with a trauma Related mental Health Disorder (such as PTSD (i.e., MDD, PTSD or another anxiety disorder)

It seeks to assist past and present Australian military personnel to better manage problematic post combat anger.

To this end, it aims to trial the effectiveness of a psychological group treatment package for problematic anger in current and ex-serving members of the Australian Defence Force.

The results of this research will be used by the researcher Tony McHugh, to obtain a PhD degree. A summary of the results will be made available to you at the completion of the project. This will be prepared using pooled data.

Should you wish to view your individual results, you can request so by writing to Tony McHugh, Principal Researcher, who will provide this data and discuss the relevance of the results obtained with you.

It is anticipated there will be a number of publications resulting from this project. These will be reported in appropriate professional journals; some of these may be picked up by the mass media and reported more widely.

This research has been funded by Weary Dunlop Research Foundation.

1.3 What does participation in this research involve?
Following assessment, participants will be included in one of three iterations of the treatment group. These groups will involve ten weekly two-hourly sessions of psychological anger-
related treatment. These sessions will be held at the Coral Balmoral Building of the Heidelberg Repatriation Hospital of Austin Health.

All treatment will be provided by trained and experienced psychology staff according to a manual provided to participants. The progress of clients will be monitored within and across all treatment sessions.

At assessment, the start and end of treatment and 12 months post the conclusion of treatment, a small battery of psychological tests measuring, anger, anxiety, depression, PTSD and other important issues will be completed to assess the impact of the treatment.

All data derived from this study, will be stored in a database, which is password protected and can only be accessed by the researchers involved. Any information obtained in connection with this research project which can identify participants will remain confidential and will only be used for the purpose of this research project. Data will only be disclosed with your permission, and not made available to any other individual or entity, except as required by law.

If you decide to take part in the research project, a prior interview will be arranged to fully describe the interview process, confirm your willingness to participate and determine if you are eligible to take part. Attending the interview will take approximately 15 minutes.

If the interview shows that you meet the requirements, then you will be able to start the research project. If the interview shows that you cannot be in the research project, the research coordinator will discuss other options with you.

This research project has been designed to make sure the researchers interpret the results in a fair and appropriate way and avoids study doctors or participants jumping to conclusions.

There are no costs associated with participating in this research project, nor will you be paid.

1.4 Other relevant information about the research project
The research project will comprise approximately 30 participants who will be divided evenly across three groups of veterans and military personnel. All groups will be identical in content and conduct.

1.5 Do I have to take part in this research project?
Participation in any research project is voluntary. If you do not wish to take part, you do not have to. If you decide to take part and later change your mind, you are free to withdraw from the project at any stage.

If you do decide to take part, you will be given this Participant Information and Consent Form to sign and you will be given a copy to keep.

Your decision whether to take part or not to take part, or to take part and then withdraw, will not affect your routine care, your relationship with professional staff or your relationship with the Psychological Trauma Recovery Service.

1.6 What are the possible benefits of taking part?
We cannot guarantee or promise that you will receive any benefits from this research; however, possible benefits may include assisting past and present Australian military personnel to better manage problematic post combat anger.

1.7 What are the possible risks and disadvantages of taking part?
You may feel that some of the questions we ask are stressful or upsetting. If you do not wish to answer a question, you may skip it and go to the next question, or you may stop immediately. If you become upset or distressed as a result of your participation in the research project, the research team will be able to arrange for counselling or other appropriate support. Any counselling or support will be provided by qualified staff who are not members of the research team. This counselling will be provided free of charge.

Whilst all care will be taken to maintain privacy and confidentiality, you may experience embarrassment if one of the group members were to repeat things said in a confidential group meeting.

1.8 What if I withdraw from this research project?
If you do consent to participate, you may withdraw at any time. If you decide to withdraw from the project, please notify a member of the research team before you withdraw. A member of the research team will inform you if there are any special requirements linked to withdrawing. If you do withdraw, you will be asked to complete and sign a ‘Withdrawal of Consent’ form; this will be provided to you by the research team. If you decide to leave the research project, the researchers will not collect additional personal information from you, although personal information already collected will be retained to ensure that the results of the research project can be measured properly and to comply with
law. You should be aware that data collected up to the time you withdraw will form part of the research project results. If you do not want your data to be included, you must tell the researchers when you withdraw from the research project.

1.9 Could this research project be stopped unexpectedly?
During the research project, new information about the risks and benefits of the project may become known to the researchers. If this occurs, you will immediately be told about this new information. This new information may mean you can no longer participate in this research. If this occurs, the person(s) supervising the research will cease your participation and you will be offered all available care to suit your needs and psychological wellbeing.

1.10 What happens when the research project ends?
It is anticipated that the results of this research project will be published and/or presented in a variety of forums, including scientific journals. Some of these may be picked up by the mass media and reported more widely. In any publication and/or presentation, information will be provided in such a way that you cannot be identified, except with your express permission. Information that can identify you will always remain confidential.
Part 2  How is the research project being conducted?

2.1  What will happen to information about me?

By signing the consent form you consent to the research team collecting and using personal information about you for the research project. Any information obtained in connection with this research project that can identify you will remain confidential.

All data derived from this study, will be stored in a database, which is password protected and can only be accessed by the researchers involved. Any information obtained in connection with this research project which can identify participants will remain confidential and will only be used for the purpose of this research project. Data will only be disclosed with your permission, and not made available to any other individual or entity, except as required by law.

Your information will only be used for the purpose of this research project and it will only be disclosed with your permission, except as required by law.

The personal information that the research team collect and use is questionnaires.

In accordance with relevant Australian and/or Victorian policy privacy and other relevant laws, you have the right to request access to the information about you that is collected and stored by the research team. You also have the right to request that any information with which you disagree be corrected. Please inform the research team member named at the end of this document if you would like to access your information.

Any information obtained for the purpose of this research project that can identify you will be treated as confidential and securely stored. It will be disclosed only with your permission, or as required by law.

2.2  Complaints and compensation

If you suffer any distress or psychological injury as a result of this research project, you should contact the research team as soon as possible. You will be assisted with arranging appropriate treatment and support with a member of the clinical team, independent of the research study.

In the event that you suffer an injury as a result of participating in this research project, hospital care and treatment will be provided by the public health service at no extra cost to you.
2.3. **Who is organising and funding the research?**

This research project is being conducted by Tony McHugh and is funded by the Weary Dunlop Medical Research Foundation.

No member of the research team will receive a personal financial benefit from your involvement in this research project (other than their ordinary wages).

2.4. **Who has reviewed the research project?**

All research in Australia involving humans is reviewed by an independent group of people called a Human Research Ethics Committee (HREC).

The ethical aspects of this research project have been approved by the HREC of Austin Health.

This project will be carried out according to the *National Statement on Ethical Conduct in Human Research (2007)*. This statement has been developed to protect the interests of people who agree to participate in human research studies.

2.5. **Further information and who to contact**

The person you may need to contact will depend on the nature of your query. If you want any further information concerning this project or if you have any problems which may be related to your involvement in the project, you can contact the researcher Tony McHugh, on (03) 9496 2184 or any of the following people:

<table>
<thead>
<tr>
<th>Name</th>
<th>Details removed for privacy reasons</th>
</tr>
</thead>
<tbody>
<tr>
<td>Position</td>
<td></td>
</tr>
<tr>
<td>Telephone</td>
<td></td>
</tr>
<tr>
<td>Email</td>
<td></td>
</tr>
</tbody>
</table>

For matters relating to research at the site at which you are participating, the details of the local site complaints person are:

<table>
<thead>
<tr>
<th>Name</th>
<th>Details removed for privacy reasons</th>
</tr>
</thead>
<tbody>
<tr>
<td>Position</td>
<td></td>
</tr>
</tbody>
</table>
If you have any complaints about any aspect of the project, the way it is being conducted or any questions about being a research participant in general, then you may contact:

**Reviewing HREC approving this research and HREC Executive Officer details**

<table>
<thead>
<tr>
<th>Reviewing HREC name</th>
<th>Austin Health Human Ethics Research Committee</th>
</tr>
</thead>
<tbody>
<tr>
<td>HREC Executive Officer</td>
<td>Manager, Austin Health Human Research Ethics Unit</td>
</tr>
<tr>
<td>Telephone</td>
<td>(03) 9496 5088</td>
</tr>
<tr>
<td>Email</td>
<td><a href="mailto:Sianna.panagiotopoulos@austin.org.au">Sianna.panagiotopoulos@austin.org.au</a></td>
</tr>
</tbody>
</table>

**Local HREC Office contact**

<table>
<thead>
<tr>
<th>Name</th>
<th>Austin Health Human Ethics Research Committee</th>
</tr>
</thead>
<tbody>
<tr>
<td>Position</td>
<td>Manager, Austin Health Human Research Ethics Unit</td>
</tr>
<tr>
<td>Telephone</td>
<td>(03) 9496 5088</td>
</tr>
<tr>
<td>Email</td>
<td><a href="mailto:Sianna.panagiotopoulos@austin.org.au">Sianna.panagiotopoulos@austin.org.au</a></td>
</tr>
</tbody>
</table>
Consent Form

Adult providing own consent

Title
Improving Anger Treatment Outcomes for Traumatised Combat Veterans: A Trial of a Stand-Alone Psychological Group Treatment.

Short Title
Treatment of Anger in Traumatised Veterans

Protocol Number
LNR/14/Austin Health/117

Project Sponsor
Weary Dunlop Foundation

Coordinating Principal Investigator/Principal Investigator
Tony McHugh

Associate Investigator(s)
Professors David Forbes, Glen Bates and Richard Kanaan

Location
Coral Balmoral Building, Heidelberg Repatriation Hospital

Declaration by Participant
I have read the Participant Information Sheet or someone has read it to me in a language that I understand.
I understand the purposes, procedures and risks of the research described in the project.
I have had an opportunity to ask questions and I am satisfied with the answers I have received.
I freely agree to participate in this research project as described and understand that I am free to withdraw at any time during the project without affecting my future care.
I understand that I will be given a signed copy of this document to keep.

Name of Participant (please print)
Signature Date

Declaration by Researcher†
I have given a verbal explanation of the research project, its procedures and risks and I believe that the participant has understood that explanation.

Name of Researcher† (please print)
Signature Date

† An appropriately qualified member of the research team must provide the explanation of, and information concerning, the research project.

Note: All parties signing the consent section must date their own signature.
Form for Withdrawal of Participation

Adult providing own consent

Title
Improving Anger Treatment Outcomes for Traumatised Combat Veterans: A Trial of a Stand-Alone Psychological Group Treatment.

Short Title
Treatment of Anger in Traumatised Veterans

Protocol Number
LNR/14/Austin Health/117

Project Sponsor
Weary Dunlop Foundation

Coordinating Principal Investigator/Principal Investigator
Tony McHugh

Associate Investigator(s)
Professors David Forbes, Glen Bates and Richard Kanaan

Location
Coral Balmoral Building, Heidelberg Repatriation Hospital

Declaration by Participant
I wish to withdraw from participation in the above research project and understand that such withdrawal will not affect my routine care, or my relationships with the researchers or the Psychological Trauma Recovery Service.

Name of Participant (please print)

Signature Date

In the event that the participant’s decision to withdraw is communicated verbally, the Senior Researcher must provide a description of the circumstances below.

Declaration by Researcher†
I have given a verbal explanation of the implications of withdrawal from the research project and I believe that the participant has understood that explanation.

Name of Researcher (please print)

Signature Date

† An appropriately qualified member of the research team must provide information concerning withdrawal from the research project.

Note: All parties signing the consent section must date their own signature.
### Appendix E:

**Summary of participant demography for all studies**

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Study One</th>
<th>Study Two</th>
<th>ADF-affiliated</th>
<th>Police</th>
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<tbody>
<tr>
<td><strong>Mean age (range)</strong></td>
<td>53.72 (43-75)</td>
<td>22.21 (17-67)</td>
<td>58.54 (26-83)</td>
<td>48.41 (33-67)</td>
</tr>
<tr>
<td><strong>Marital Status</strong></td>
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<td></td>
</tr>
<tr>
<td>Single</td>
<td>3.0%</td>
<td>84.8%</td>
<td>7.3%</td>
<td>4.1%</td>
</tr>
<tr>
<td>Divorced</td>
<td>15.7%</td>
<td>2.0%</td>
<td>5.5%</td>
<td>4.1%</td>
</tr>
<tr>
<td>Separated</td>
<td>15.7%</td>
<td>0.5%</td>
<td>3.7%</td>
<td>4.1%</td>
</tr>
<tr>
<td>Defacto</td>
<td>4.7%</td>
<td>8.1%</td>
<td>6.4%</td>
<td>11.4%</td>
</tr>
<tr>
<td>Married</td>
<td>74.2%</td>
<td>4.6%</td>
<td>77.1%</td>
<td>76.4%</td>
</tr>
<tr>
<td>Widowed</td>
<td>1.5%</td>
<td>0%</td>
<td>0%</td>
<td>0%</td>
</tr>
<tr>
<td><strong>Studying</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>94.4%</td>
<td></td>
<td></td>
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</tr>
<tr>
<td><strong>Working</strong></td>
<td></td>
<td>80.2%</td>
<td>1.8%</td>
<td>52%</td>
</tr>
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<td><strong>Military branch</strong></td>
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<td></td>
</tr>
<tr>
<td>Army</td>
<td>93.7%</td>
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</tr>
<tr>
<td>Navy</td>
<td>6.0%</td>
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<td></td>
<td>7.3%</td>
</tr>
<tr>
<td>Airforce</td>
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<td></td>
<td></td>
<td>6.4%</td>
</tr>
<tr>
<td><strong>National Service</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Vietnam</td>
<td>100%</td>
<td></td>
<td></td>
<td>67.0%</td>
</tr>
<tr>
<td>Rwanda</td>
<td></td>
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<td>0.9%</td>
</tr>
<tr>
<td>Somalia</td>
<td></td>
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<td>3.7%</td>
</tr>
<tr>
<td>East Timor</td>
<td></td>
<td></td>
<td></td>
<td>11.9%</td>
</tr>
<tr>
<td>Iraq</td>
<td></td>
<td></td>
<td></td>
<td>7.3%</td>
</tr>
<tr>
<td>Afghanistan</td>
<td></td>
<td></td>
<td></td>
<td>4.6%</td>
</tr>
<tr>
<td>Gulf War</td>
<td></td>
<td></td>
<td></td>
<td>1.8%</td>
</tr>
</tbody>
</table>

**Notes:**

- **Study One** participants respectively comprised 353 and 252 male Vietnam veterans at pre-treatment and post-treatment.
- **Study Two** participants comprised 50 male and 147 female undergraduate university psychology students.
- **Study Three** participants comprised 219 male and 13 female past and present ADF-affiliated (n=109) and police (n=123) participated (all women were police).
Appendix F:

Studies Used in Study 1

AUDIT QUESTIONNAIRE

Thank you for completing this test. Please circle the answer that best corresponds to you.

1. How often do you have a drink containing alcohol?
   - Never
   - Once a month or less
   - 2 to 4 times a month
   - 2 to 3 times a week
   - 4 or more times a week

Please remember a standard drink contains 10g of pure alcohol.

Each of these is a standard drink:
   - 1 Middy/Pot of Standard Beer
   - 1 Glass of Wine
   - 1 Glass of Sherry or Port
   - 1 Nip of Spirits

2. How many ‘standard’ drinks (see above) containing alcohol do you have on a typical day when you are drinking?
   - 1 or 2
   - 3 or 4
   - 5 or 6
   - 7 to 9
   - 10 or more

3. How often do you have six or more drinks on one occasion?
   - Never
   - Less than once a month
   - Monthly
   - Weekly
   - Daily or almost daily

4. How often during the last twelve months have you found that you were not able to stop drinking once you had started?
   - Never
   - Less than once a month
   - Monthly
   - Weekly
   - Daily or almost daily

5. How often during the last twelve months have you failed to do what was normally expected from you because of drinking?
   - Never
   - Less than once a month
   - Monthly
   - Weekly
   - Daily or almost daily

6. How often during the last twelve months have you needed a drink in the morning to get yourself going after a heavy drinking session?
   - Never
   - Less than once a month
   - Monthly
   - Weekly
   - Daily or almost daily

7. How often during the last twelve months have you had a feeling of guilt or remorse after drinking?
   - Never
   - Less than once a month
   - Monthly
   - Weekly
   - Daily or almost daily

8. How often during the last twelve months have you been unable to remember what happened the night before because you had been drinking?
   - Never
   - Less than once a month
   - Monthly
   - Weekly
   - Daily or almost daily

9. Have you or someone else been injured as a result of your drinking?
   - No
   - Yes, but not in the last twelve months
   - Yes, during the last twelve months

10. Has a relative, a friend, a doctor or other health professional been concerned about your drinking or suggested you cut down?
    - No
    - Yes, but not in the last twelve months
    - Yes, during the last twelve months

11. Do you think you presently have a problem with drinking?
    - No
    - Probably Not
    - Unsure
    - Possibly
    - Definitely

12. In the next twelve months, how difficult would you find it to cut down or stop drinking?
    - Very Easy
    - Fairly Easy
    - Neither Difficult Nor Easy
    - Fairly Difficult
    - Very Difficult
COMBAT EXPOSURE SCALE

*Please circle the number above the answer that best describes your experience*

1. Did you ever go on combat patrols or have other dangerous duty?
   - No
   - 1-3X
   - 4-12X
   - 13-50X
   - 51+times

2. Were you ever under enemy fire?
   - Never
   - <1 month
   - 1-3 months
   - 4-6 months
   - 7 months or more

3. Were you ever surrounded by the enemy?
   - No
   - 1-2X
   - 3-12X
   - 13-25X
   - 26+times

4. What percentage of the soldiers in your unit were killed (KIA), wounded or missing in action (MIA)?
   - None
   - 1-25%
   - 26-50%
   - 51-75%
   - 76% or more

5. How often did you fire rounds at the enemy?
   - Never
   - 1-2X
   - 3-12X
   - 13-50X
   - 51 or more

6. How often did you see someone hit by incoming or outgoing rounds?
   - Never
   - 1-2X
   - 3-12X
   - 13-50X
   - 51 or more

7. How often were you in danger of being injured or killed (i.e., being pinned down, overrun, ambushed, near miss, etc.)?
   - Never
   - 1-2X
   - 3-12X
   - 13-50X
   - 51 or more

## DIMENSION OF ANGER REACTIONS SCALE

*Do your best to judge as accurately as you can the degree to which the following statements describe your feelings and behaviour. That is, rate the degree to which each statement applies to you.*

1. I often find myself getting angry with people or situations.

<table>
<thead>
<tr>
<th></th>
<th>0</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
</tr>
</thead>
<tbody>
<tr>
<td>not at all</td>
<td>little</td>
<td>very little</td>
<td>some</td>
<td>not</td>
<td>moderately</td>
<td>fairly</td>
<td>much</td>
<td>very</td>
<td>exactly</td>
</tr>
</tbody>
</table>

2. When I do get angry, I get really mad.

<table>
<thead>
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</thead>
<tbody>
<tr>
<td>not at all</td>
<td>little</td>
<td>very little</td>
<td>some</td>
<td>not</td>
<td>moderately</td>
<td>fairly</td>
<td>much</td>
<td>very</td>
<td>exactly</td>
</tr>
</tbody>
</table>

3. When I get angry, I stay angry.

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<tbody>
<tr>
<td>not at all</td>
<td>little</td>
<td>very little</td>
<td>some</td>
<td>not</td>
<td>moderately</td>
<td>fairly</td>
<td>much</td>
<td>very</td>
<td>exactly</td>
</tr>
</tbody>
</table>

4. When I get angry with someone, I want to hit or clobber the person.

<table>
<thead>
<tr>
<th></th>
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<th>1</th>
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</tr>
</thead>
<tbody>
<tr>
<td>not at all</td>
<td>little</td>
<td>very little</td>
<td>some</td>
<td>not</td>
<td>moderately</td>
<td>fairly</td>
<td>much</td>
<td>very</td>
<td>exactly</td>
</tr>
</tbody>
</table>

5. My anger interferes with my ability to get my work done.

<table>
<thead>
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<th>2</th>
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</thead>
<tbody>
<tr>
<td>not at all</td>
<td>little</td>
<td>very little</td>
<td>some</td>
<td>not</td>
<td>moderately</td>
<td>fairly</td>
<td>much</td>
<td>very</td>
<td>exactly</td>
</tr>
</tbody>
</table>

6. My anger prevents me from getting along with people as well as I’d like to.

<table>
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<th>8</th>
</tr>
</thead>
<tbody>
<tr>
<td>not at all</td>
<td>little</td>
<td>very little</td>
<td>some</td>
<td>not</td>
<td>moderately</td>
<td>fairly</td>
<td>much</td>
<td>very</td>
<td>exactly</td>
</tr>
</tbody>
</table>

7. My anger has had a bad affect on my health.

<table>
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<tr>
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<tbody>
<tr>
<td>not at all</td>
<td>little</td>
<td>very little</td>
<td>some</td>
<td>not</td>
<td>moderately</td>
<td>fairly</td>
<td>much</td>
<td>very</td>
<td>exactly</td>
</tr>
</tbody>
</table>
**HOSPITAL ANXIETY AND DEPRESSION SCALE**

This questionnaire is designed to help identify how you feel. Read each item and then tick the appropriate box which comes closest to how you have been feeling in the past month. Do not take too long over your replies: your immediate reaction to each item will probably be more accurate than a long thought-out response.

1. I feel tense or 'wound up':
   - Most of the time
   - A lot of the time
   - From time to time, occasionally
   - Not at all

2. I still enjoy the things I used to enjoy:
   - Definitely as much
   - Not quite as much
   - Only a little
   - Hardly at all

3. I get a sort of frightened feeling as is something awful is about to happen:
   - Very definitely and quite badly
   - Yes, but not too badly
   - A little, but it doesn't worry me
   - Not at all

4. I can laugh and see the funny side of things:
   - As much as I always could
   - Not quite so much now
   - Definitely not so much now
   - Not at all

5. Worrying thoughts go through my mind:
   - A great deal of the time
   - A lot of the time
   - Not too often
   - Very little

6. I feel cheerful:
   - Never
   - Not often
   - Sometimes
   - Most of the time

7. I can sit at ease and feel relaxed:
   - Definitely
   - Usually
   - Not often
   - Not at all

8. I feel as if I am slowed down:
   - Nearly all the time
   - Very often
   - Sometimes
   - Not at all

9. I get a sort of frightened feeling like ‘butterflies’ in the stomach:
   - Not at all
   - Occasionally
   - Quite often
   - Very often

10. I have lost interest in my appearance:
    - Definitely
    - I don’t take as much care as I should
    - I may not take quite as much care
    - I take just as much care as ever

11. I feel restless as if I have to be on the move:
    - Very much indeed
    - Quite a lot
    - Not very much
    - Not at all

12. I look forward with enjoyment to things:
    - As much as I ever did
    - Rather less than I used to
    - Definitely less than I used to
    - Hardly at all

13. I get sudden feelings of panic:
    - Very often indeed
    - Quite often
    - Not very often
    - Not at all

14. I can still enjoy a book of radio or television program:
    - Often
    - Sometimes
    - Not often
    - Very seldom
PTSD Checklist (PCL M)

**Instructions:** Below is a list of problems and complaints that veterans sometimes have in response to stressful military experience experiences. Please read each one carefully, then circle one of the numbers to the right to indicate how much you have been bothered by that problem in the last month.

<table>
<thead>
<tr>
<th></th>
<th></th>
<th>Not at all</th>
<th>A little bit</th>
<th>Moderately</th>
<th>Quite a bit</th>
<th>Extremely</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Repeated, disturbing memories, thoughts, or images of a stressful military experience?</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td></td>
</tr>
<tr>
<td>2. Repeated, disturbing dreams of a stressful military experience?</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td></td>
</tr>
<tr>
<td>3. Suddenly acting or feeling as if a stressful military experience were happening again (as if you were reliving it)?</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td></td>
</tr>
<tr>
<td>4. Feeling very upset when something reminded you of a stressful military experience?</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td></td>
</tr>
<tr>
<td>5. Having physical reactions (e.g., heart pounding, trouble breathing, sweating) when something reminded you of a stressful military experience?</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td></td>
</tr>
<tr>
<td>6. Avoiding thinking about or talking about a stressful military experience or avoiding having feelings related to it?</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td></td>
</tr>
<tr>
<td>7. Avoiding activities or situations because they remind you of a stressful military experience?</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td></td>
</tr>
<tr>
<td>8. Trouble remembering important parts of a stressful military experience?</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td></td>
</tr>
<tr>
<td>9. Loss of interest in activities that you used to enjoy?</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td></td>
</tr>
<tr>
<td>10. Feeling distant or cut off from other people?</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td></td>
</tr>
<tr>
<td>11. Feeling emotionally numb or being unable to have loving feelings for those close to you?</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td></td>
</tr>
<tr>
<td>12. Feeling as if your future somehow will be cut short?</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td></td>
</tr>
<tr>
<td>13. Trouble falling or staying asleep?</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td></td>
</tr>
<tr>
<td>14. Feeling irritable or having angry outbursts?</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td></td>
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<tr>
<td>15. Having difficulty concentrating?</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td></td>
</tr>
<tr>
<td>16. Being “superalert” or watchful or on guard?</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td></td>
</tr>
<tr>
<td>17. Feeling jumpy or easily startled?</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
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<td></td>
</tr>
</tbody>
</table>
Appendix G:

*Scales Used in Study 2*

**AUTOMATIC THOUGHTS QUESTIONNAIRE**

Listed below are a variety of thoughts that pop into people’s heads. Please read each thought and indicate how frequently, if at all, the thought occurred to you over the last week. Please read each item carefully and circle the appropriate answers on the answer sheet in the following fashion.

(1 = “not at all”, 2 = “some times”, 3 = “moderately often”, 4 = “often” and 5 = “all the time”).

<table>
<thead>
<tr>
<th>Number</th>
<th>Thought</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
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</thead>
<tbody>
<tr>
<td>1</td>
<td>I feel like I’m up against the world.</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>2</td>
<td>I’m no good</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>3</td>
<td>Why can’t I ever succeed?</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>4</td>
<td>No one understands me</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>5</td>
<td>I’ve let people down</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
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<tr>
<td>6</td>
<td>I don’t think I can go on</td>
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<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>7</td>
<td>I wish I were a better person</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>8</td>
<td>I’m so weak</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>9</td>
<td>My life’s not going the way I want it to</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
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<td>10</td>
<td>I’m so disappointed in myself</td>
<td>1</td>
<td>2</td>
<td>3</td>
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<td>5</td>
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<tr>
<td>11</td>
<td>Nothing feels good anymore</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>12</td>
<td>I can’t stand this anymore</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>13</td>
<td>I can’t get started</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>14</td>
<td>What’s wrong with me?</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
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<tr>
<td>15</td>
<td>I wish I were somewhere else</td>
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<td>2</td>
<td>3</td>
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<td>16</td>
<td>I can’t get things together</td>
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<td>2</td>
<td>3</td>
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<td>5</td>
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<tr>
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<td>I hate myself</td>
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<td>3</td>
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<td>5</td>
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<tr>
<td>18</td>
<td>I’m worthless</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
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<tr>
<td>19</td>
<td>Wish I could just disappear</td>
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<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
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<td>What’s the matter with me?</td>
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<td>5</td>
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<tr>
<td>21</td>
<td>I’m a loner</td>
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<td>3</td>
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<td>My life is a mess</td>
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<td>I’m a failure</td>
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<td>I’ll never make it</td>
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<td>I feel so helpless</td>
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<td>26</td>
<td>Something has to change</td>
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<td>3</td>
<td>4</td>
<td>5</td>
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<td>27</td>
<td>There must be something wrong with me</td>
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<td>2</td>
<td>3</td>
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<tr>
<td>28</td>
<td>My future is bleak</td>
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<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
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<tr>
<td>29</td>
<td>It’s just not worth it</td>
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<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
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<tr>
<td>30</td>
<td>I can’t finish anything</td>
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<td>Rating Scale</td>
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<tr>
<td>1</td>
<td>I found myself getting upset by quite trivial things</td>
<td>0 1 2 3</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>I was aware of dryness of my mouth</td>
<td>0 1 2 3</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>I couldn't seem to experience any positive feeling at all</td>
<td>0 1 2 3</td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>4</td>
<td>I experienced breathing difficulty (e.g., excessively rapid breathing, breathlessness in the absence of physical exertion)</td>
<td>0 1 2 3</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>I just couldn't seem to get going</td>
<td>0 1 2 3</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>I tended to over-react to situations</td>
<td>0 1 2 3</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>I had a feeling of shakiness (e.g., legs going to give way)</td>
<td>0 1 2 3</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>I found it difficult to relax</td>
<td>0 1 2 3</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>9</td>
<td>I found myself in situations that made me so anxious I was most relieved when they ended</td>
<td>0 1 2 3</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>10</td>
<td>I felt that I had nothing to look forward to</td>
<td>0 1 2 3</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>11</td>
<td>I found myself getting upset rather easily</td>
<td>0 1 2 3</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>12</td>
<td>I felt that I was using a lot of nervous energy</td>
<td>0 1 2 3</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>13</td>
<td>I felt sad and depressed</td>
<td>0 1 2 3</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>14</td>
<td>I found myself getting impatient when I was delayed in any way (e.g., lifts, traffic lights, being kept waiting)</td>
<td>0 1 2 3</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>15</td>
<td>I had a feeling of faintness</td>
<td>0 1 2 3</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>16</td>
<td>I felt that I had lost interest in just about everything</td>
<td>0 1 2 3</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>17</td>
<td>I felt I wasn't worth much as a person</td>
<td>0 1 2 3</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>18</td>
<td>I felt that I was rather touchy</td>
<td>0 1 2 3</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>19</td>
<td>I perspired noticeably (e.g., hands sweaty) in the absence of high temperatures or physical exertion</td>
<td>0 1 2 3</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>20</td>
<td>I felt scared without any good reason</td>
<td>0 1 2 3</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>21</td>
<td>I felt that life wasn't worthwhile</td>
<td>0 1 2 3</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
**Reminder of rating scale:**

0 Did not apply to me at all  
1 Applied to me to some degree, or some of the time  
2 Applied to me to a considerable degree, or a good part of time  
3 Applied to me very much, or most of the time

<table>
<thead>
<tr>
<th></th>
<th>Description</th>
<th>Rating Scale</th>
</tr>
</thead>
<tbody>
<tr>
<td>22</td>
<td>I found it hard to wind down</td>
<td>0 1 2 3</td>
</tr>
<tr>
<td>23</td>
<td>I had difficulty in swallowing</td>
<td>0 1 2 3</td>
</tr>
<tr>
<td>24</td>
<td>I couldn't seem to get any enjoyment out of the things I did</td>
<td>0 1 2 3</td>
</tr>
<tr>
<td>25</td>
<td>I was aware of the action of my heart in the absence of physical exertion</td>
<td>0 1 2 3</td>
</tr>
<tr>
<td></td>
<td>(eg, sense of heart rate increase, heart missing a beat)</td>
<td></td>
</tr>
<tr>
<td>26</td>
<td>I felt down-hearted and blue</td>
<td>0 1 2 3</td>
</tr>
<tr>
<td>27</td>
<td>I found that I was very irritable</td>
<td>0 1 2 3</td>
</tr>
<tr>
<td>28</td>
<td>I felt I was close to panic</td>
<td>0 1 2 3</td>
</tr>
<tr>
<td>29</td>
<td>I found it hard to calm down after something upset me</td>
<td>0 1 2 3</td>
</tr>
<tr>
<td>30</td>
<td>I feared that I would be &quot;thrown&quot; by some trivial but unfamiliar task</td>
<td>0 1 2 3</td>
</tr>
<tr>
<td>31</td>
<td>I was unable to become enthusiastic about anything</td>
<td>0 1 2 3</td>
</tr>
<tr>
<td>32</td>
<td>I found it difficult to tolerate interruptions to what I was doing</td>
<td>0 1 2 3</td>
</tr>
<tr>
<td>33</td>
<td>I was in a state of nervous tension</td>
<td>0 1 2 3</td>
</tr>
<tr>
<td>34</td>
<td>I felt I was pretty worthless</td>
<td>0 1 2 3</td>
</tr>
<tr>
<td>35</td>
<td>I was intolerant of anything that kept me from getting on with what I was doing</td>
<td>0 1 2 3</td>
</tr>
<tr>
<td>36</td>
<td>I felt terrified</td>
<td>0 1 2 3</td>
</tr>
<tr>
<td>37</td>
<td>I could see nothing in the future to be hopeful about</td>
<td>0 1 2 3</td>
</tr>
<tr>
<td>38</td>
<td>I felt that life was meaningless</td>
<td>0 1 2 3</td>
</tr>
<tr>
<td>39</td>
<td>I found myself getting agitated</td>
<td>0 1 2 3</td>
</tr>
<tr>
<td>40</td>
<td>I was worried about situations in which I might panic and make a fool of my self</td>
<td>0 1 2 3</td>
</tr>
<tr>
<td>41</td>
<td>I experienced trembling (eg, in the hands)</td>
<td>0 1 2 3</td>
</tr>
<tr>
<td>42</td>
<td>I found it difficult to work up the initiative to do things</td>
<td>0 1 2 3</td>
</tr>
</tbody>
</table>
IMAGINAL PROCESSES INVENTORY (ABRIDGED)

**Introduction/Instructions**
This brief test is designed to assess the extent to which people experience visual imagery.

Throughout the test, this capacity is measured by the use of the term "daydream(s)". Although there is no agreed definition of this term, what it is meant to describe is mental activity involving thinking that is unrelated to something being undertaken. For example, thinking that may go on while you are getting ready for sleep or a long journey, rather than thinking specifically related to a work, study or recreational task. This imagery-based thinking can be negative or positive in nature.

Please answer the questions involved by reference to the instructions in the heading on each page. Try to answer all items as they apply to you now, not as they might have applied to you at some previous time in your life.

<table>
<thead>
<tr>
<th>For each question, circle the letter which is most true or appropriate for you</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
</tr>
<tr>
<td>1 I daydream</td>
</tr>
<tr>
<td>2 I daydream while working or studying</td>
</tr>
<tr>
<td>3 I recall or think over my daydreams</td>
</tr>
<tr>
<td>4 I lose myself in active daydreaming</td>
</tr>
<tr>
<td>5 Whenever I have time on my hands I daydream</td>
</tr>
<tr>
<td>6 When I am at a meeting, event or activity that is not very interesting, I daydream rather than pay attention</td>
</tr>
<tr>
<td>7 On a long bus, train or airplane trip I daydream</td>
</tr>
<tr>
<td>8 I would characterise myself as someone who daydreams</td>
</tr>
<tr>
<td>9 Instead of noticing people and events in the world around me, I spend approximately the following amount of my time lost in thought</td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td>---</td>
</tr>
<tr>
<td>10</td>
</tr>
<tr>
<td>11</td>
</tr>
<tr>
<td>12</td>
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<tr>
<td>13</td>
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<tr>
<td>14</td>
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<td>15</td>
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<td>16</td>
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<td>40</td>
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<tr>
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</tr>
<tr>
<td>41</td>
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<tr>
<td>42</td>
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<tr>
<td>47</td>
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<tr>
<td>48</td>
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<tr>
<td>49</td>
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<tr>
<td>50</td>
</tr>
<tr>
<td>51</td>
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<tr>
<td>52</td>
</tr>
<tr>
<td>53</td>
</tr>
<tr>
<td>54</td>
</tr>
<tr>
<td>55</td>
</tr>
<tr>
<td>56</td>
</tr>
</tbody>
</table>
Appendix H:

*Visual Imagery in Daydreams Scale*

For each question, circle the letter which is most true or appropriate for you

<table>
<thead>
<tr>
<th></th>
<th>Definitely not true for me</th>
<th>Usually not true for me</th>
<th>Usually true for me</th>
<th>True for me</th>
<th>Very true for me</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>A</td>
<td>B</td>
<td>C</td>
<td>D</td>
<td>E</td>
</tr>
<tr>
<td>2</td>
<td>A</td>
<td>B</td>
<td>C</td>
<td>D</td>
<td>E</td>
</tr>
<tr>
<td>3</td>
<td>A</td>
<td>B</td>
<td>C</td>
<td>D</td>
<td>E</td>
</tr>
<tr>
<td>4</td>
<td>A</td>
<td>B</td>
<td>C</td>
<td>D</td>
<td>E</td>
</tr>
<tr>
<td>5</td>
<td>A</td>
<td>B</td>
<td>C</td>
<td>D</td>
<td>E</td>
</tr>
<tr>
<td>6</td>
<td>A</td>
<td>B</td>
<td>C</td>
<td>D</td>
<td>E</td>
</tr>
<tr>
<td>7</td>
<td>A</td>
<td>B</td>
<td>C</td>
<td>D</td>
<td>E</td>
</tr>
<tr>
<td>8</td>
<td>A</td>
<td>B</td>
<td>C</td>
<td>D</td>
<td>E</td>
</tr>
<tr>
<td>9</td>
<td>A</td>
<td>B</td>
<td>C</td>
<td>D</td>
<td>E</td>
</tr>
<tr>
<td>10</td>
<td>A</td>
<td>B</td>
<td>C</td>
<td>D</td>
<td>E</td>
</tr>
<tr>
<td>11</td>
<td>A</td>
<td>B</td>
<td>C</td>
<td>D</td>
<td>E</td>
</tr>
<tr>
<td>12</td>
<td>A</td>
<td>B</td>
<td>C</td>
<td>D</td>
<td>E</td>
</tr>
</tbody>
</table>
Appendix I:

Swinburne University of Technology Approval of Study Informed Consent Form adopted

To: Dr S Knowles FLSS & Ms Katherine Chisholm

I refer to the ethical review of the above project protocol undertaken on behalf of Swinburne University's Human Research Ethics Committee (SUHREC) by a SUHREC Subcommittee (SHESC1). Your responses, as emailed on 7 August 2008, were put to a Subcommittee delegate for consideration.

I am pleased to advise that the project (as submitted to date) has been cleared to proceed in line with standard on-going ethics clearance conditions here outlined.

All human research activity undertaken under Swinburne auspices must conform to Swinburne and external regulatory standards, including the National Statement on Ethical Conduct in Human Research and with respect to secure data use, retention and disposal.

The named Swinburne Chief Investigator/Supervisor remains responsible for any personnel appointed to or associated with the project being made aware of ethics clearance conditions, including research and consent procedures or instruments approved. Any change in chief investigator/supervisor requires timely notification and SUHREC endorsement.

The above project has been approved as submitted for ethical review by or on behalf of SUHREC. Amendments to approved procedures or instruments ordinarily require prior ethical appraisal/clearance. SUHREC must be notified immediately or as soon as possible thereafter of: (a) any serious or unexpected adverse effects on participants and any redress measures; (b) proposed change in protocols; and (c) unforeseen events which might affect continued ethical acceptability of the project.

At a minimum, an annual report on the progress of the project is required as well as at the conclusion (or abandonment) of the project.

A duly authorised external or internal audit of the project may be undertaken at anytime.
Please contact me if you have any queries about on-going ethics clearance.
The SUHREC project number should be quoted in communication.
Best wishes for the project.
Yours sincerely
Keith Wilkins
Secretary
Human Ethics Committee
Swinburne University of Technology
PO Box 218
HAWTHORN VIC 3122
Tel+61 392 145 218
Fax+61 392 145 267
### PTSD Checklist - Civilian Version

**Instructions:**
Below is a list of problems and complaints that people sometimes have in response to stressful life experiences. Please read each one carefully, then circle one of the numbers to the right to indicate how much you have been bothered by that problem in the last month.

<table>
<thead>
<tr>
<th></th>
<th>Not at all</th>
<th>A little bit</th>
<th>Moderately</th>
<th>Quite a bit</th>
<th>Extremely</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Repeated, disturbing memories, thoughts, or images of a stressful experience from the past?</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>2. Repeated, disturbing dreams of a stressful experience from the past?</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>3. Suddenly acting or feeling as if a stressful experience from the past were happening again (as if you were reliving it)?</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>4. Feeling very upset when something reminded you of a stressful experience from the past?</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>5. Having physical reactions (e.g., heart pounding, trouble breathing, sweating) when something reminded you of a stressful experience from the past?</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>6. Avoiding thinking about or talking about a stressful experience from the past or avoiding having feelings related to it?</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>7. Avoiding activities or situations because they remind you of a stressful experience from the past?</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>8. Trouble remembering important parts of a stressful experience from the past?</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>9. Loss of interest in activities that you used to enjoy?</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>10. Feeling distant or cut off from other people?</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>11. Feeling emotionally numb or being unable to have loving feelings for those close to you?</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>12. Feeling as if your future somehow will be cut short?</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>13. Trouble falling or staying asleep?</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>14. Feeling irritable or having angry outbursts?</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>15. Having difficulty concentrating?</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>16. Being “superalert” or watchful or on guard?</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>17. Feeling jumpy or easily startled?</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
</tbody>
</table>
Appendix K:

Mean scores on all variables by gender for Studies Two and Three

<table>
<thead>
<tr>
<th>Variables</th>
<th>Study 2</th>
<th></th>
<th>Study 3 (Time 1)</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Men</td>
<td>Women</td>
<td>$f$</td>
<td>$p$</td>
</tr>
<tr>
<td>Imagery Frequency</td>
<td>33.7</td>
<td>37.4</td>
<td>4.52</td>
<td>.04</td>
</tr>
<tr>
<td>Imagery Absorption</td>
<td>47.4</td>
<td>52.8</td>
<td>4.70</td>
<td>.03</td>
</tr>
<tr>
<td>Imagery Vividness</td>
<td>17.8</td>
<td>18.8</td>
<td>.85</td>
<td>.36</td>
</tr>
<tr>
<td>Imagery Anger</td>
<td>20.9</td>
<td>17.2</td>
<td>7.52</td>
<td>.01</td>
</tr>
<tr>
<td>General Imagery</td>
<td>119.8</td>
<td>126.3</td>
<td>2.61</td>
<td>.05</td>
</tr>
<tr>
<td>Negative Thoughts</td>
<td>48.2</td>
<td>51.1</td>
<td>1.56</td>
<td>.21</td>
</tr>
<tr>
<td>Anxiety</td>
<td>9.8</td>
<td>10.5</td>
<td>2.19</td>
<td>.14</td>
</tr>
<tr>
<td>Depression</td>
<td>10.4</td>
<td>10.8</td>
<td>.54</td>
<td>.46</td>
</tr>
<tr>
<td>Anger</td>
<td>19.7</td>
<td>19.8</td>
<td>.00</td>
<td>.95</td>
</tr>
<tr>
<td>PTSD Overall</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PTSD Intrusions</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PTSD Avoidance</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PTSD Arousal</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Notes:
- Study Two participants comprised 47 men and 150 women and Study Three participants comprised 219 men and 13 women
- Anxiety was respectively assessed by the DASSA and the HADS-a in studies two and three
- Depression was respectively assessed by DASSD and the HADS-d in studies two and three
- Appendix N provides a percentile comparison of mean total scores on the DASS and HADS
## Appendix L:

*Mean scores on all variables for ADF-affiliated and Police Participants*

<table>
<thead>
<tr>
<th>Variables</th>
<th>Study 1</th>
<th>Study 2</th>
<th>Study 3</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Vietnam veterans</td>
<td>University students</td>
<td>ADF-affiliated &amp; Police personnel</td>
</tr>
<tr>
<td>General Imagery</td>
<td>T1</td>
<td>T2</td>
<td>T1</td>
</tr>
<tr>
<td>Imagery Frequency</td>
<td>36.5</td>
<td>40.0</td>
<td>36.7</td>
</tr>
<tr>
<td>Imagery Absorption</td>
<td>Not applicable</td>
<td>51.5</td>
<td>55.1</td>
</tr>
<tr>
<td>Imagery Vividness</td>
<td>18.6</td>
<td>24.0</td>
<td>22.2</td>
</tr>
<tr>
<td>Imagery Anger</td>
<td>18.1</td>
<td>27.7</td>
<td>25.3</td>
</tr>
<tr>
<td>Negative Thoughts</td>
<td>50.4</td>
<td>78.9</td>
<td>75.4</td>
</tr>
<tr>
<td>Anxiety</td>
<td>14.5</td>
<td>12.6</td>
<td>10.3</td>
</tr>
<tr>
<td>Depression</td>
<td>11.7</td>
<td>10.4</td>
<td>10.7</td>
</tr>
<tr>
<td>Anger</td>
<td>32.8</td>
<td>27.9</td>
<td>19.8</td>
</tr>
<tr>
<td>PTSD Overall</td>
<td>64.3</td>
<td>57.6</td>
<td>N/A</td>
</tr>
<tr>
<td>PTSD Intrusions</td>
<td>17.2</td>
<td>15.1</td>
<td>N/A</td>
</tr>
<tr>
<td>PTSD Avoidance</td>
<td>26.6</td>
<td>23.9</td>
<td>N/A</td>
</tr>
<tr>
<td>PTSD Arousal</td>
<td>20.5</td>
<td>18.6</td>
<td>N/A</td>
</tr>
</tbody>
</table>

**Notes:**
- Study Two data was collected at one timepoint only
- Study Two participants comprised 50 men and 147 women and Study Three participants comprised 219 men and 13 women
- Anxiety was respectively assessed by the DASSA and the HADS-a in studies two and three
- Depression was respectively assessed by DASSD and the HADS-d in studies two and three
- Appendix N provides a percentile comparison of mean total scores on the DASS and HADS
**Appendix M:**

_Overall mean scores on all variables at both times for police and ADF-Affiliated Participants_

<table>
<thead>
<tr>
<th>Variables</th>
<th>ADF-P T1 (a)</th>
<th>Police T1 (c)</th>
<th>Police T2 (d)</th>
<th>Order of magnitude</th>
<th>f</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Imagery Frequency</td>
<td>188.1</td>
<td>171.9</td>
<td>160.0</td>
<td>a&gt;b&gt;c&gt;d</td>
<td>1.89</td>
<td>.17</td>
</tr>
<tr>
<td>Imagery Absorption</td>
<td>40.9</td>
<td>39.2</td>
<td>35.9</td>
<td>a&gt;c&gt;b&gt;d</td>
<td>3.48</td>
<td>.06</td>
</tr>
<tr>
<td>Imagery Vividness</td>
<td>56.8</td>
<td>53.6</td>
<td>50.2</td>
<td>a&gt;b&gt;c&gt;d</td>
<td>22.55</td>
<td>&lt;.01</td>
</tr>
<tr>
<td>Imagery Anger</td>
<td>26.9</td>
<td>21.7</td>
<td>20.6</td>
<td>a&gt;b&gt;c&gt;d</td>
<td>11.87</td>
<td>&lt;.01</td>
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<tr>
<td>General Imager</td>
<td>30.2</td>
<td>25.5</td>
<td>24.1</td>
<td>a&gt;b&gt;c&gt;d</td>
<td>10.53</td>
<td>&lt;.01</td>
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<td>Negative Thoughts</td>
<td>82.7</td>
<td>75.5</td>
<td>68.6</td>
<td>a&gt;b&gt;c&gt;d</td>
<td>3.30</td>
<td>.07</td>
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<tr>
<td>Anxiety</td>
<td>12.5</td>
<td>10.4</td>
<td>10.6</td>
<td>a&gt;b&gt;d&gt;c</td>
<td>3.59</td>
<td>.06</td>
</tr>
<tr>
<td>Depression</td>
<td>30.2</td>
<td>24.5</td>
<td>22.0</td>
<td>a&gt;b&gt;c&gt;d</td>
<td>16.76</td>
<td>&lt;.01</td>
</tr>
<tr>
<td>Anger</td>
<td>10.5</td>
<td>9.5</td>
<td>8.8</td>
<td>b&gt;a&gt;c&gt;d</td>
<td>11.27</td>
<td>&lt;.01</td>
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<tr>
<td>PTSD Overall</td>
<td>58.8</td>
<td>53.6</td>
<td>48.3</td>
<td>a&gt;b&gt;c&gt;d</td>
<td>8.90</td>
<td>&lt;.01</td>
</tr>
<tr>
<td>PTSD Intrusions</td>
<td>15.9</td>
<td>14.8</td>
<td>13.8</td>
<td>a&gt;b&gt;c&gt;d</td>
<td>3.67</td>
<td>&lt;.06</td>
</tr>
<tr>
<td>PTSD Avoidance</td>
<td>24.2</td>
<td>21.7</td>
<td>18.9</td>
<td>a&gt;b&gt;c&gt;d</td>
<td>9.06</td>
<td>&lt;.01</td>
</tr>
<tr>
<td>PTSD Arousal</td>
<td>18.7</td>
<td>17.2</td>
<td>15.4</td>
<td>a&gt;b&gt;c&gt;d</td>
<td>7.46</td>
<td>&lt;.01</td>
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</tbody>
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_Note:_
- Study population comprised 109 male police, 13 female police and 110 ADF-affiliated participants.
Appendix N:

Percentile comparison of DASS Scores (Study 2) and HADs Scores (Study 3)

<table>
<thead>
<tr>
<th></th>
<th>Study 2</th>
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<th>Study 3</th>
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<td></td>
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<td>9.8</td>
<td>10.5</td>
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<td>DASS-d</td>
<td>10.4</td>
<td>10.8</td>
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<td>HADs-d</td>
<td>11.5</td>
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Notes:
The procedure for comparing the anxiety and depression of the DASS scores (Study Two) with scores on the HADS (Study Three) in terms of their percentile ranking was as described by Crawford and others (2009). Utilisation of the approach outlined there and conducted by reference to the statistical program “Mood Score – PRs.exe” (see homepages.abdn.ac.uk) revealed comparable scores for HADs-d (depression) and DASS-d (depression), but a meaningful difference in anxiety scores (DASS-a and HADS-a) between studies Two and Three.
Appendix O:

*Stepwise regression analysis results utilising imagery subscales as the independent variables and affects as the dependent variables in analysis*

<table>
<thead>
<tr>
<th>Dependent variables</th>
<th>Imagery variables in equation</th>
<th>$r^2$</th>
<th>$\Delta R^2$</th>
<th>$B$</th>
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<tr>
<td>Depression</td>
<td>Absorption</td>
<td>.05</td>
<td>-</td>
<td>.24</td>
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<td>Anger</td>
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<td>Frequency</td>
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Appendix P:

*Stepwise regression results utilising imagery subscales as the independent variables and PTSD and its sub-factors as the dependent variables in analysis*

<table>
<thead>
<tr>
<th>Dependent variables</th>
<th>Imagery variables in equation</th>
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<th>$\Delta r^2$</th>
<th>$B$</th>
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<tr>
<td>Absorption</td>
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<td>.13</td>
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<td>.23</td>
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<tr>
<td>Vividness</td>
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Author/s: McHugh, Anthony Francis

Title: I’m hurt, annoyed and see things: anger in PTSD and the role of visual imagery

Date: 2015

Persistent Link: http://hdl.handle.net/11343/57167

File Description: I’m hurt, annoyed and see things: anger in PTSD and the role of visual imagery